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Food allergy to egg and soy lecithins

M. Palm*, D.A. Moneret-Vautrin, G. Kanny, S. Denery-Papini, S. Frémont

Key words: double-blind challenges; food allergy; lecithins; RAST inhibition.

● EGG and soy lecithins are commonly used in the food industry as emulsifiers (E322). The possibility of residual proteins leads to suspicion of their role in allergic manifestations in subjects allergic to egg or soy. Two cases are

reported in which the double-blind, placebo-controlled oral challenge

Two patients with DBPCOC positive to 50–100 mg of allergen.

(DBPCOC), the “gold standard” of diagnosis, led to confirmation that lecithin had caused lesions of atopic dermatitis (AD) in patients suffering from egg and soy allergies.

E.G., a 15-month-old girl, presented with AD, progressive from the age of 2½ months, as she was fed a milk formula. There was a family history of atopy (paternal pollinosis). The SCORAD was estimated at 26/103.

Prick tests to five inhalants were negative. Positive prick tests were obtained for egg white (10 mm) (codeine 9%=3 mm) and egg lecithin (3 mm) (negative in 61 patients allergic to egg), and RAST to egg was class 2 (Coopérative Pharmaceutique Française, Paris, France).

The labial challenge test (1) for egg white was positive, leading to exacerbation of eczema a few hours later, with an increase in the DBPCOC index from 26 at the start to 38/

103. The DBPCOC to egg lecithin was positive at 50 mg, with the appearance of an erythematous rash on the neck and shoulders 1 h after ingestion.

The second patient, G.S., a 4-year-old boy, with nonatopic parents, presented with asthma and AD. AD occurred at the age of 6 weeks and wheezing at 3 months. At around 1 year, the mother reported an episode of conjunctivitis complicated by angioedema in the eyelids immediately after ingestion of a small piece of peanut. A worsening of the asthmatic symptoms was noted after the age of 2 years. Strict avoidance of peanuts did not bring about satisfactory improvement.

Prick tests were positive for *Dermatophagoides pteronyssinus*, birch and grass pollens, and peanut, with crossed sensitization to soy, but the prick test with soy lecithin was negative (Table 1), and RAST was 33.2 kU/l. The DBPCOC for peanut was positive at a total dose of 15 mg. The DBPCOC for peanut oil (6-ml dose) was also positive, causing an asthma attack.

RAST for soy was 11 kU/l. The inhibition by soy lecithin rose to 62%. The DBPCOC at 100 mg of lecithin was positive, with the appearance of an erythematous rash on the jaw 1 h after ingestion.

Protein assay of soy lecithin by the Kjeldahl method revealed a level of 3.5%, and of 11.3% in the egg lecithin.

Lecithins are included in the excipients of certain drugs and/or lipidic emulsions used for parenteral feeding. Adverse reactions have been described, due to the inhalation of a bronchodilator with soy lecithins in the excipient, or to intravenous infusions (2, 3). In the food industry, asthma from soy lecithin inhalation has been reported (6). The level of soy lecithin in bread can be 0.015% (6). Many milk formulas including hydrolysates contain soy lecithin. Porras et al.'s study (6) confirmed the presence of soy proteins in oil, margarine, and lecithin, and the allergenicity of these proteins was demonstrated in Awazuhara et al.'s study (7). RAST inhibition of 62% for soy, obtained by lecithin solution, confirmed the allergenic nature of the residual proteins (3.5%). To our knowledge, we report the first case of allergy to egg lecithin.

The presence of lecithins in formulas, as well as in cookies and other foods, may explain why avoidance diets for soy and eggs that do not exclude lecithins yield only partial results. Therefore, we recommend that labeling be more precise.

*Department of Clinical Immunology and Allergy
Hôpital Central
Av. de Lattre de Tassigny
54035 Nancy
France

Table 1. Study of cross-sensitivity of legumes by prick tests to native and cooked foods

Legumes	Raw (mm)	Cooked (10 min, 100°C) (mm)
(9% codeine: 3 mm)		
Peanut	23	n.d.
Soy	6	10
Soy lecithin	0	n.d.
Chickpeas	5	3
Lentils	3	0
Beans	1	0
Green peas	2	0
Lima beans	0	0
Lupin flour	0	0
n.d.=not done.		

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Anaphylactic reaction to castor bean seeds

R. Navarro-Rouimi, D. Charpin*

Key words: anaphylaxis; CAP-RAST; castor bean.

● CASTOR bean (*Ricinus communis*) seeds contain castor bean oil, which is nonallergenic and nontoxic; a toxic albumin called ricine; and a globulin which causes allergic reactions.

This seed has a highly sensitizing effect upon occupational workers and residents living close to castor-oil-processing factories

(1). Case reports and epidemiologic observations have discussed asthma in relation to inhalation of castor bean dust. There are a few case reports of severe symptoms and even death occurring after castor bean seeds were chewed by children and even by adults who attempted to use the whole bean, instead of the oil, as a purgative. Ingested ricin may prove fatal in a dose of 0.03 gm. In contrast, in 1943, Spies & Coulson demonstrated that the castor bean allergen is a nontoxic, heat-stable polysaccharide-protein fraction representing 1.8% of defatted castor bean meal (2). Allergic reactions to the seeds are very uncommon. In the literature, we found one report (3) describing two cases in which the ingestion of a single bean produced edema of the mouth, pharynx, and glottis, followed within 5 min by generalized urticaria with giant wheals, which was readily relieved by epinephrine.

After moving to a new home in June 1991, Mrs Cor, born in 1955, developed severe asthma. Skin tests demonstrated allergy to house-dust mite, cat dander, and cypress pollen. A few weeks later, asthma attacks became intermittent and spirometric measurements went back to normal values.

In early July 1998, Mrs Cor, while playing with her son, chewed a castor bean seed. Within a few minutes, she developed giant urticaria lesions and became drowsy. An emergency unit was called. Her blood pressure was 6 mmHg. She was dyspneic and had Quincke's edema. After receiving epinephrine, intravenous steroids, antihistamines, and intravenous fluids, she quickly recovered. One month later, blood analysis demonstrated CAP-RAST (Pharmacia-Upjohn) to castor bean of 80-100 IU/l; i.e., class 5.

This paper deals with an anaphylactic reaction to castor bean seed. In addition to a suggestive history, RAST, which is a very reliable diagnostic tool (4), was strongly positive.

Retrospectively, the occurrence and rapid improvement of asthma noticed in 1991 was

probably due to castor bean-pollen allergy. When the patient moved into her new house, the many castor bean plants growing there were immediately removed. The castor bean plant pollinates from May to October in southern France. Its pollen is considered to be a low sensitizer. There is cross-reactivity between castor bean pollen and seed (5).

*Department of Chest Diseases and Allergy
 Hôpital Nord
 13915 Marseille Cedex 20
 France

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Unconventional medicine: a risk of undertreatment of allergic patients

G. Senna, G. Passalacqua, M. Crivellaro, P. Bonadonna, F. Gani, R. Dorizzi, A. Dama, G.W. Canonica*, C. Lombardi

Key words: allergy; risk factors; unconventional medicine; undertreatment.

● ALLERGIC respiratory diseases (asthma and rhinitis) represent a problem with high and