

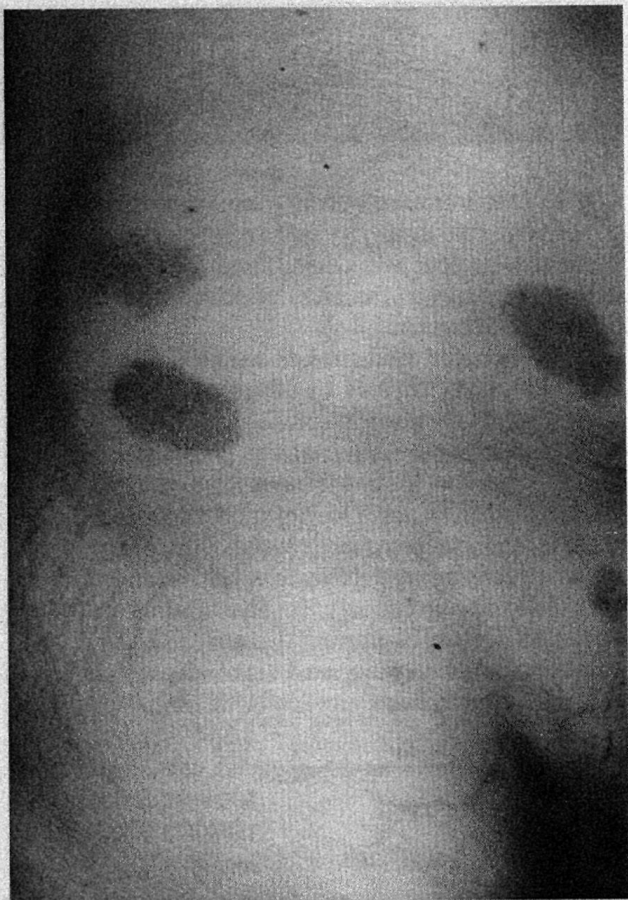


FIGURE 1 Eczematous plaques on abdominal wall.

matous and pruritic reactions. Intradermal tests with sodium heparin were negative at that moment. A third reading at 72 h showed very intensive eczematous reactions with papula and vesicles at places where undiluted tests with sodium enoxaparin, calcium nadroparin, and sodium heparin were performed. Sodium heparin presented the most intensive reaction and appeared positive even at 1/10 dilution (500 UI/ml).

Hypersensitivity reactions to heparin and derivatives are rare. Klein et al. (1) consider eczema-like plaques an abortive form of heparin necrosis. The lesions appear earlier and are usually accompanied by thromboembolic events in the case of heparin necrosis. In our patient, the lesions appeared 3 days after beginning treatment, and this could be considered too early for delayed-type hypersensitivity reaction except in the case of previous contact with the drug (sensitization period) as in previous surgery. The absence of necrosis confirmed histologically and the results of patch and intradermal tests (positive at 48 and 96 h, and 24 and 72 h, respectively) indicated a type IV hypersensitivity reaction. Recently, Méndez et al. reported a similar case of delayed hypersensitivity reaction to enoxaparin.

However, patch tests were negative, intradermal tests were not performed, and the subcutaneous challenge test was necessary to establish the diagnosis in that patient. The authors suggest that when the patch test is negative, subcutaneous challenge is the most reliable test modality (2). On the contrary, our data support the view that intradermal tests should be done previously in order to avoid a potentially higher risk of systemic reaction after challenge.

Finally, cross-reactivity among enoxaparin, nadroparin, and sodium heparin was demonstrated in this patient. Moreover, sodium heparin gave the strongest delayed intradermal response. The use of premedication such as corticosteroids and antihistamines could be appropriate in these cases.

Key words: adverse drug reaction; enoxaparin; heparin; nadroparin.

F. VALDÉS, C. VIDAL*, V. FERNÁNDEZ-REDONDO, C. PETEIRO, J. TORIBIO

*Departamento de Dermatología
Facultad de Medicina
C/. San Francisco s/n
15705 Santiago de Compostela
Spain
Tel. 34 81 563100 ext 12246
Fax: 34 81 547094
E-mail: mejaim@usc.es

Accepted for publication 12 February 1998

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ALLERGY 1998;53:625-626

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Pectin and cashew nut allergy: cross-reacting allergens?

AN anaphylactic reaction probably to cashew nut in chocolate candy is reported. The patient also experienced occupational rhinitis and urticaria from pectin dust. There seems to be allergenic similarity between pectin and cashew nut.

A 42-year-old woman with no history of atopy was working in a yogurt dairy. Extensive investigations because of long-lasting diarrhea made 6 years earlier revealed only abnormal positive skin prick test reaction to orange (wheal 9 mm). After the patient abstained from orange juice and citrus fruits, her diarrhea ceased. Recently, the patient had experienced a strong allergic reaction after

eating one piece of chocolate candy. Within 20 min, she developed shortness of breath, widespread urticaria, and angioedema. For low blood pressure, epinephrine and corticosteroid were administered, leading to complete recovery. The chocolate candy was of foreign origin and, according to the manufacturer, it contained cocoa, sugar, vanilla, wheat, egg, and "white Indian nut". We interpreted this to mean cashew nut.

Three months later, the patient handled a new brand of pectin at work. Immediately she got a bout of sneezing, her conjunctivae became blood-shot and the eyelids swollen, and she developed urticaria on uncovered skin on her hands and forearms. The patient had not noticed any side-effects from the pectin she had handled before. This new brand (Genu Pectin type DF, Copenhagen Pectin A/S, Denmark) was extracted from citrus peel and consisted mainly of partial methyl esters of polygalacturonic acid.

The patient was extensively skin prick tested with inhalant and food allergens. Pollens (trees, grass, mugwort), mammals, dust mites, molds, grain plants, milk, egg, fish, and orange were from ALK (Hørsholm, Denmark). Other allergens were those of cocoa, fresh fruits, vegetables, spices, and nuts (cashew, pistachio, coconut, peanut, almond, pecan, walnut, hazelnut, and Brazil nut). The only positive (wheal of >3 mm) reactions were to cashew, pistachio, and pectin (Table 1). Five control persons were negative. The patient's wheal reaction to cashew increased locally to about 2 cm within 30 min, but no systemic symptoms appeared. The patient received 20 mg cetirizine and was monitored for 2 h.

No oral challenge with cashew nut was done later.

The blood basophil histamine release test (BHRT) was performed with orange, cashew, pistachio, and pectin as allergens (1). Positive results (>15% release) were detected with cashew, pistachio, and pectin (Table 1). The BHRT was negative for two control persons. Serum specific IgE was positive (>0.35 kU/l) for orange, cashew, and pistachio measured with the commercial CAP RAST kit (Pharmacia & Upjohn, Uppsala, Sweden) (Table 1). In addition, the patient's IgE to cashew, pistachio, pectin, the patient's own chocolate candy, and orange (peel, flesh, and juice concentrate) was evaluated by the immunospot method (2). Distinct IgE binding to cashew, pistachio, pectin, and chocolate candy was demonstrated

Table 1. Skin and laboratory test results

Allergen	Prick* (mm)	RAST (kU/l)	BHRT (%)	Immunospot (IgE binding)
Orange/juice concentrate	0	0.7	8	+
Cashew	11ps	1.4	66	++
Pistachio	7	1.0	57	+
Pectin	4	n.d.	88	++

*Positive control (10 mg/ml histamine) wheal 5 mm, prick test with 5 mg/ml pectin.

BHRT: basophil histamine release test; ps: pseudopodia; n.d.: not done.

(Table 1). IgE binding was also detected to orange juice concentrate, but not to orange flesh or peel. No or only weak binding to pectin was detected with the control sera (nonatopic and non-pollen-allergic sera). The patient's total IgE was 19 kU/l. The cross-reactivity of IgE antibodies to cashew and pectin was studied by immunospot and RAST inhibition (2). In the immunospot inhibition, pectin distinctly inhibited the binding of IgE to cashew. Cashew in turn inhibited the binding to pectin, but to a lesser intensity. In addition, pectin strongly inhibited the patient's cashew RAST (Rf 202, Pharmacia & Upjohn) (percentage of inhibition 100%, dilution of pectin 1:1500 w/v). No inhibition by pectin (1–2%) was demonstrated in the control latex RAST.

Pectins are high-molecular-weight carbohydrates found in fruit. They are used as a food additive as consistency-forming, gelatinizing, and stabilizing agents under the designation E440. Occupational sensitization with rhinitis or asthma from pectin has been described in the production of jam, marmalade, and candies (3–5). Our patient got rhinitis, conjunctivitis, and contact urticaria from pectin. Cashew nut belongs to the family Anacardiaceae. Other representatives of the family are pistachio, mango, and poison ivy. Common antigenic determinants have been found in pistachio and cashew nuts (6). As far as we know, this is the first report on antigenic similarity between cashew nut and pectin. Interestingly, in an earlier report, one patient with pectin-induced asthma developed urticaria and shortness of breath from cashew nuts (4).

In the immunospot or RAST-inhibition studies, the binding of IgE to cashew was completely blocked by pectin, whereas the binding to pectin was less evidently inhibited by cashew. The inhibition was specific, since no inhibition of latex RAST was demonstrated by pectin. These results indicate that pectin contains all the same allergen epitopes as cashew, whereas cashew does not exhibit all the same epitopes as pectin relevant to our patient. Further studies are necessary to characterize cross-reacting allergens in cashew nut and pectin in greater detail.

*An atopic woman
occupationally
sensitized to pectin
developed anaphylaxis
after eating cashew nuts.*

Key words: cashew nut; cross-allergy; pectin.

L. RÄSÄNEN, S. MÄKINEN-KILJUNEN, R. J. HARVIMA*

*Department of Dermatology

Kuopio University Hospital

PO Box 1777

FIN-70211 Kuopio

Finland

Fax: 358-17-172530

Accepted for publication 12 February 1998

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ALLERGY 1998;53:626-628

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