

whole of the trunk. Despite antihistamine treatment the pruritus remained severe and unbearable, requiring definitive transfer to haemodialysis two months later. The skin reactions resolved rapidly.

The histology was non-specific: parakeratotic epidermis, with slight serosity in the interspinal spaces, fibrosis of the superficial dermis, numerous capillary or arteriolar vessels with slight perivascular infiltrate of lymphocytes, histiocytes or plasmocytes.

Prick tests with the common aero-allergens, latex, formol and ethylene oxide were negative. Patch tests with ICDRG (International Contact Dermatitis Research Group) allergens, chlorhexidine, iodine alcohol, strong iodine solution, povidone iodine, ethylene oxide and latex were also negative. Skin test with icodextrin (prick test, intradermal tests from 10^{-3} to pure, patch test) were negative. Icodextrin-induced leukocyte histamine release was negative.

Relationship to icodextrin was retained on chronological criteria: symptoms started 10 days after contact with icodextrin and resolved completely one week after discontinuation. The clinical picture is similar to reactions reported in the literature (3,4,6). Ten days corresponds to onset times previously reported (i.e., from 8 to 14 days) (2–4,6). The delay is shorter in the event of re-challenge (3). Clinical signs generally resolve between 5 and 7 days after discontinuation of icodextrin (3,4,6). One of the particularities in our observation is that the rash started at the catheter exit site. The catheter cannot be responsible for inducing this allergic reaction because when the glucose-based dialysate was administered through the same catheter the reaction resolved. The clinical picture of these reactions is not compatible with dextran-induced anaphylactic reactions, as has been suggested. The mechanism of this reaction probably differs from that described by Del Rosso et al. with fever and abdominal pain occurring a few hours after using icodextrin (7).

The starting point of this rash (peritoneal catheter exit site), the negative skin test results (when read immediately or later), suggest an immune process that begins in the peritoneum. This hypothesis is compatible with the observation of Heering et al. who demonstrated that icodextrin could induce pseuperitonitis and lymphocyte stimulation (9). The

maculopapular skin rash could be a distant manifestation of the peritoneal immune process.

*Gisèle Kanny

Service de Médecine Interne
Immunologie Clinique et Allergologie
Center Hospitalier Universitaire
Hôpital central
29, avenue de Lattre de Tassigny
54035 Nancy Cedex
France

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Cow's milk allergy – a matter of fat

U. Hoppu, M. Kalliomäki, E. Isolauri

Key words: breastfeeding, cow's milk allergy, diet, fatty acid.

• ELIMINATION of cow's milk from the breastfeeding mother's diet is an approach commonly used in attempts to prevent allergic disease in infants. Results, especially long-term, have often been conflicting and disappointing (1). One explanation may be that previous studies have concentrated qualitatively, not quantitatively, on allergenic proteins in the diet, and not on other dietary factors with immunomodulatory properties such as dietary fatty acids. Specific polyunsaturated fatty acids have been shown to be

associated with **Breastfeeding mothers intake of fat may influence sensitization** on the other of

allergic inflammation (2). We therefore now determine maternal intakes of cow's milk protein and fat during breastfeeding and evaluate their effects on the infant's sensitization to cow's milk and the incidence of cow's milk allergy (CMA).

Altogether 114 infants with a family history of allergic disease, who had been breastfed for at least three months, were followed up during their first year of life. The mothers compiled a four day record of food intake when the infants were 3 months old. Nutrient intakes were calculated using Nutrica software (Research Center of the Social Insurance Institution, Turku, Finland). The infants were clinically examined at 3, 6 and 12 months of age and skin prick tests (SPT) were performed at 12 months of age as previously described (3). In the case of symptoms suggestive of food allergy, double-blind, placebo-controlled challenges followed by open consumption were carried out. Sensitization to cow's milk was defined as a positive SPT reaction to milk, and cow's milk allergy (CMA) as positive to cow's milk challenge. Results are given as means with 95% confidence interval (CI) in parentheses.

Positive skin prick test reactivity to milk was detected in 7/114 (6%) infants. A total of 15/114 (13%) infants had challenge-proven cow's milk allergy. The mean age at diagnosis of CMA was 7.2 (5.9-8.5) months. The mean duration of breastfeeding was exclusively 3.3 (3.0-3.6) and overall 7.7 (7.1-8.3) months and was comparable in infants with and without positive SPT or challenge.

The mean energy intake of the mothers was 8.0 (7.7-8.3), MJ/day. Maternal intake of cow's milk protein ranged from 1.9 to 63.1 g/day, mean 26.2 (24.0-28.3) g/day and, adjusted for energy intake, 3.3 (3.0-3.5) g/MJ/day. The mean proportion of energy derived from fat was 36.6 (35.6-37.6) E% and from saturated fatty acids 15.8 (15.2-16.3), E%. Maternal intake of cow's milk protein was not associated with SPT reaction to milk or CMA in the infant (Table 1). In contrast, high intakes of total fat and saturated fatty acids were associated with sensitization to milk.

Our result indicates that sensitization to food allergens may not depend directly on the absolute amount of allergenic proteins in the diet, but rather on a complexity of dietary determinants. The Western diet typically contains an abundance of fat, especially saturated fatty acids, and the prevalence of allergic disease is highest in

Western societies. It would appear that breastfeeding mothers should perhaps reduce their fat intake rather than eliminate cow's milk from their diet to prevent allergy in the infant.

Ulla Hoppu, Department of Paediatrics
University of Turku
20520 Turku
Finland
Tel. + 358 2333 6063
Fax: + 358 2313 1460
E-mail: ulla.hoppu@utu.fi

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Table 1. Maternal dietary intake during breastfeeding associated with cow's milk allergy and sensitization to milk in the infant

Maternal intake	CMA +	CMA -	p-value *	SPT milk +	SPT milk -	p-value *
Milk protein (g/MJ)						
Low (<3.3) n = 57	9 (16%)	48 (84%)	0.58	3 (5%)	54 (95%)	0.72
High (= 3.3) n = 57	6 (11%)	51 (89%)		4 (7%)	53 (93%)	
Total fat (E%)						
Low (<36.6) n = 59	6 (10%)	53 (90%)	0.41	0 (0%)	59 (100%)	0.005
High (= 36.6) n = 55	9 (16%)	46 (84%)		7 (13%)	48 (87%)	
Saturated fatty acids (E%)						
Low (<15.8) n = 56	4 (7%)	52 (93%)	0.09	0 (0%)	56 (100%)	0.01
High (= 15.8) n = 58	11 (19%)	47 (81%)		7 (12%)	51 (88%)	

*. Categories defined on basis of mean values, Fisher's exact test
CMA+, child with cow's milk allergy; CMA -, child without cow's milk allergy
SPT milk +, positive skin prick test reaction to milk; SPT milk -, no skin prick test reaction to milk

Allergenic similarities of 2S albumins

R. Asero*, G. Mistrello, D. Roncarolo, S. Amato

Key words: 2S albumins; anaphylaxis; cross-reactivity; mustard; sunflower seed.

● A 54-YEAR-OLD man with a history of more than 10 years of pellitory allergy experienced an anaphylactic episode 15 min after the ingestion of a piece of bread 'baked with five cereals' purchased at a supermarket. Skin prick tests (SPT) with commercial extracts of cereals (wheat, rye, barley, rice, oats) and soybean (Dome-Hollister/Stier 1:10 w/v), and with both the crust and crumb of the offending bread roll were negative. In contrast, some fragments included in the crust of the roll induced a strong skin reaction (wheal diameter 10 mm); these turned out to be sunflower seeds (not declared among the ingredients of the roll). The patient did not recall any sunflower seed intake before, but had bred parrots and reported that their food contained sunflower seeds. In view of possible cross-reactivity between the major allergen of sunflower seed, a 2S albumin (1,2), and similar proteins in other vegetable foods (3-7), SPT were performed with extracts of peanut, walnut, Brazil nut (DHS, 1:10 w/v), sesame (Stallergenes 1:3 w/v), and with a commercial mustard (Louit Freres brand; Dijon type). The patient reacted to the mustard only.

Sunflower seeds (raw and roasted) and mustard seeds were homogenized and extracted (5%) in 0.1 M phosphate-buffered saline (PBS; pH 7.4), and shaken overnight at 4°C. Extracts showed final protein concentrations of 3.4, 2.3, and 0.9 mg/ml, respectively. Immunoblot analysis of sunflower seed extract was carried out by conventional methods. To investigate the cross-reactivity between allergens of sunflower seed and mustard, 0.3 ml of the patient's serum was absorbed with 0.8 ml of mustard extract for 3 h at 25°C before immunoblotting.

Sunflower seed-induced anaphylaxis with cross-reactivity to mustard.