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Case Report

A case of milk allergy that presented anaphylaxis after cutaneous contact with allergen

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Abstract

Milk allergy in a 1-year and 8-month-old boy is reported. At 1 year and 1 month of age, the patient presented with anaphylaxis, including erythema, which was initially localized to the contact site of the anterior chest, and wheezing accompanied by dyspnea, 5 min after contact with milk allergen through his atopic skin. These symptoms continued for 50 min. Seventy minutes after the disappearance of the initial erythema, the patient developed subsequent erythematous lesions distributed throughout the neck and head area that persisted for as long as 24 h. On another occasion, he also exhibited a pale face and generalized erythema immediately after an accidental oral ingestion of milk at the age of 1 year and 8 months. He had been unsettled for several hours when an intravenous steroid was administered. His serum IgE was 590 IU/mL and the radioallergosorbent test (RAST) scores against milk, α-lactoalbumin, β-lactoglobulin, casein and cheese were 5, 2, 3, 5 and 5, respectively. This is a rare case of a patient with milk allergy who fell into anaphylaxis following both cutaneous contact with and oral ingestion of the offending milk protein. Care should be taken with patients with food allergies because cutaneous contact with the offending food may cause adverse reactions, including anaphylaxis.

Key words: anaphylaxis, atopic dermatitis, cutaneous contact, milk allergy.

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Introduction

Food allergy has been defined as a state of immunologic reaction resulting from the ingestion of a food or a food additive.[1] Clinical manifestations of food allergy involve multiple organs, such as skin, the gastrointestinal tract, the respiratory tract and the central nervous system. Of the clinical manifestations of food allergy, anaphylaxis is the most severe reaction that may sometimes prove fatal. However, it is rare that food adsorbed through the skin causes systemic allergic reactions. Herein, we report on an intriguing case of milk allergy that presented anaphylaxis immediately after cutaneous contact with milk protein.

Clinical summary

A 1-year and 1-month-old boy was admitted to our hospital because of asthmatic bronchitis and atopic dermatitis. He had eliminated cow's milk from his diet because milk ingestion had induced eczematous lesions during his infancy. In addition, he avoided some foods on the basis of results of the radioallergoimmunosorbent test (RAST). On admission, erythematous patches with lichenification were noted on his face, especially on his cheeks. His neck, axillary, antecubital and inguinal areas were scattered with lichenification associated with small spots of crusting. During his hospital stay, another patient nearby spilled approximately 10 mL cow's milk over his anterior chest and the localized area of his clothing was dampened by the spilt milk. The patient then presented with several spots of erythema localized on the anterior chest and wheezing accompanied by dyspnea 5 min after this accidental contact with milk through his atopic skin. The erythematous lesions were approximately 1–1.5 cm in diameter with no exudation or erosion, which gradually reduced in size and then disappeared in 50 min. His

Table 1 Serum radioallergosorbent test score

Allergen	RAST score	Allergen	RAST score
House dust	2	Melon	1
Egg white	5	Banana	2
Egg yolk	4	Peach	1
Wheat	4	Orange	1
Soy been	2	Strawberry	1
Rice	2	Grapefruits	2
Milk	5	Apple	2
α-Lactoalbumin	2	Tuna	2
β-Lactoglobulin	3	Salmon	2
Casein	5	Mackerel	3
Cheese	5	Horse mackerel	3
Pumpkin	2	Sardine	3
Spinach	2	Beef	1
Tomato	2	Chicken	1
Carrot	1	Pork	1
Pea	1	Gelatin	0
Barley	2	Latex	2
Potato	1	SEA	1
Onion	1	SEB	2

RAST, radioallergosorbent test; SEA, SEB, Staphylococcal enterotoxin A and B, respectively.

Table 2 Skin prick test

Allergen	Results	Allergen	Results
House dust	+	Eggplant	+
Chicken	++	Bamboo shoot	+
Pork	++	Corn	+
Bonito	++	Green soybeans	–
Dried bonito	++	Buckwheat	–
Tuna	++	Rice	–
Salmon	++	Wheat	–
Mackerel	++	Potato	–
Horse mackerel	++	Tomato	–
Flatfish	++	Spinach	–
Yellowtail	++	Taro	–
Cod	++	Shrimp	–
Sardine	++	Octopus	–
Mackerel pike	++	Crab	–
Cuttlefish	++		

–, no change; +, erythema alone; ++, wheal 4 mm in diameter or larger with erythema.

respiratory symptoms were attenuated by the inhalation of a β_2-adrenoceptor agonist. Approximately 2 h after cutaneous contact with the milk, widely distributed erythema accompanied by itching appeared on the skin of his neck and head and persisted for almost 24 h; this outbreak required treatment with steroid ointment. These symptoms were considered to be anaphylactic reactions caused by cutaneous contact with milk protein. At 1 year and 8 months of age, the patient's face became pale shortly after an accidental ingestion of milk and he was then medicated with DL-chlorphenylamine maleate (Polaramine®; Schering-Plough, Osaka, Japan) and was transferred to our hospital by ambulance. On arrival, he was irritable and had generalized erythema, which was determined to be an anaphylactic reaction, although he presented no obvious signs of hypotension. The patient's stomach was pumped to avoid further absorption of the milk and then intravenous steroid was administered for a period of 4 days to prevent a late-phase reaction and delayed-type hypersensitivity. His condition improved with time and he was discharged from hospital after 5 days.

At the time of his first anaphylactic reaction caused by cutaneous contact with milk, the patient showed hypereosinophillia (1780/μL) with elevated serum IgE levels (590 IU/mL). Milk allergen-specific IgE, as determined by capsulated hydrophilic carrier polymer (CAP)-RAST were 5 for whole milk protein, 2 for α-lactoalbumin, 3 for β-lactoglobulin, 5 for casein and 5 for cheese (Table 1), which suggested that he had already been sensitized with milk protein and this may be an explanation for his anaphylaxis. This patient was sensitized with multiple allergens other than milk protein, as indicated by the positive RAST scores (Table 1) and positive skin prick tests (Table 2).

After successful treatment for the second episode of anaphylaxis, the patient has been in relatively good health, although he has needed to eliminate certain foods from his diet, as demonstrated by the multiple offending foods shown in Tables 1,2.

Discussion

The immediate skin symptoms and respiratory signs caused by cutaneous contact with offending allergen in this patient are similar to those described in patients with contact urticaria.[2] Severe cases of this disease may include systemic anaphylaxis, which is defined as contact urticaria syndrome.[2,3] Allergic symptoms in remote areas, such as erythema in the neck and head, and respiratory involvement, such as wheezing and dyspnea, as well as localized erythema indicate that mechanisms similar to contact urticaria may operate in this case. This patient, however, presented erythema instead of papula, typical

of urticaria, suggesting that his disease is similar but not the same as contact urticaria (syndrome).

The local erythema expressed in the anterior chest as a first symptom should correspond to the immediate-phase reaction of type I allergy. Subsequent redness that appeared from 2 h after the challenge and persisted for 24 h may be the late-phase reaction in immediate allergy. In the case of contact urticaria, active atopic skin showing inflammation has been suggested to be more sensitive to allergen challenge than intact skin.[2] In addition to the condition of the local skin, the site of the skin may affect the sensitivity: the upper back, extensor side of the upper arm and the elbow fossa are sites of high sensitivity.[4] In this patient, the site of allergen invasion was the anterior chest and the skin at this site had eczematous lesions with exudate, which may have allowed the penetration of the offending milk protein and heightened the responsiveness to the allergen. The diagnosis of contact urticaria is based on the results of a patch test and scratch test, which may sometimes cause hazardous anaphylaxis. The patient in this report experienced anaphylaxis through both skin contact with and oral ingestion of the allergen, which should preclude diagnostic skin tests for milk.

This patient showed a high titer of serum IgE antibody against whole milk protein and its components, which may explain the anaphylaxis seen even after cutaneous contact with the allergen. In fact, the second episode of anaphylaxis induced by oral ingestion of milk at 1 year and 8 months of age underscores this patient's highly hypersensitive state against milk protein. The sensitization to milk may have been caused by the oral intake of milk protein either by the child himself or through the transplacental pathway during his intrauterine life (before birth).

The mechanisms whereby cutaneous contact with allergen evokes an allergic reaction in remote organs has not been definitely resolved. The first possibility comes from the immediate entrance of the offending proteins into the systemic circulation, spreading into the resident cells in the various organs. The second idea stems from the migration of inflammatory cells resident in the site of local challenge into remote areas using homing receptors or chemokine receptors. The last hypothesis is the accumulation of the resident cells into the local skin site by chemokines and then their return to their organs of origin to express their inflammatory function.

Thus, anaphylaxis is rarely induced by skin contact with an allergen in patients with atopic dermatitis. However, the medical management of patients with severe food allergy should include the care of the patient following cutaneous contact with allergen.

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