

Correspondence

Common millet anaphylaxis: a case of a bird-keeper sensitized to millet via inhalation, who developed anaphylaxis after oral ingestion

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We report a case of anaphylaxis induced by common millet (*Panicum miliaceum*) in a 37-year-old Japanese woman with no previous history of anaphylaxis. She had a history of allergic rhinitis and had kept a budgerigar from 1984 to 1994; the bird was not confined to a birdcage but was allowed to roam free within the patient's room. In 1994, the bird escaped and the patient disposed of the cage.

Ten years later, in December 2004, the patient ate rice with common millet, scallops, carrots and orange juice. She had not eaten common millet previously. One hour later, she felt uncomfortable and then developed vertigo, shivering, chills, dyspnoea, dysphonia, nausea, vomiting, abdominal pain, general itching, weals and diarrhoea.

On arrival at the emergency unit of our hospital, the patient's blood pressure was 100/50 mmHg (usual blood pressure 130/80 mmHg). She had generalized erythema and oedema covering the whole body. She was treated with intravenous corticosteroid and fluid therapy, to which she responded within 1 h and her symptoms resolved completely after 6 hours. She was given a prescription for a corticosteroid (betamethasone) and antihistamine (DL-chlorpheniramine maleate).

Three weeks later, in early January 2004, the patient attended for skin-prick testing (SPT) against common millet, rice, scallop, carrot and orange juice. The SPT showed a weal of 30 × 50 mm and an area of erythema 50 × 220 mm in size, a strongly positive reaction to common millet. Radioallergosorbent testing (RAST) for specific IgE antibody to common millet was 11.5 IU/mL (normal < 0.34). Other positive specific IgE antibodies included: 1.16 IU/mL for budgerigar feathers, 0.38 IU/mL for budgerigar faeces, 1.16 IU/mL for budgerigar protein and 5.71 IU/mL for Japanese millet. Radioimmunosorbent testing (CAP, Phadia, Uppsala, Sweden) for total IgE antibody was 114 IU/mL (normal ≤ 250). The test was negative for rice, Italian millet, orange, carrot, buckwheat, soybean, apple, oatmeal, blue mussel, various pollens [Japanese cedar, hogweed, rye, wheat, reed, sweet vernal grass, cock's foot grass (*Dactylis glomerata*), Johnson grass (*Sorghum halapense*)] and house dust.

In January 2007, RAST for specific IgE antibodies was repeated. Positive specific IgE antibodies included: 1.80 IU/mL for common millet, 0.40 IU/mL for budgerigar feathers, 0.51 IU/mL for budgerigar faeces and 0.77 IU/mL for Japanese millet. Total IgE antibody was 54 IU/mL.

A diagnosis of common millet-induced anaphylaxis was made based on the positive SPT and RAST and the systemic clinical symptoms.^{1–3} We prescribed an epinephrine self-injection kit and suggested that the patient should avoid millet. After the anaphylactic episode, the patient ate rice daily, but did not have any reaction to it, and she has had no further food allergic reactions.

Common millet is mostly used as birdseed in Japan. The birdseed that the patient fed her budgerigar contained common millet and Japanese millet. However, with the increasing interest in natural foods, products not previously considered as food are being consumed by the Japanese population.

Anaphylaxis to millet was first reported in 1981 by Parker *et al.*¹ Since then, other cases have been reported sporadically, showing that anaphylaxis to millet is still extremely rare.^{1–3} Bohle *et al.* reported that patients who develop an allergic reaction to millet show millet-specific IgE, and that 63% of bird keepers who are atopic possess millet-specific IgE.⁴ They assumed that these patients used the millet as birdseed and that sensitization to millet occurred via inhalation. However, in their study, patients with allergic reaction to millet did not show any specific IgE to bird antigens, whereas in our case, specific IgE antibodies to two types of millet (common and Japanese) and to bird antigens were found. We believe that the patient had been exposed to millet as a bird keeper and developed anaphylaxis following ingestion of millet. Stuck *et al.* reported a similar case of sensitization to millet via inhalation and subsequent anaphylaxis after oral ingestion.³ Thus, the current case provides support for Bohle's hypothesis.⁴

Given the patient's history of bird-keeping, her allergic rhinitis was most likely due to hypersensitivity to millet-containing birdseed. Previous studies have shown that sensitization to inhalation allergens is the primary event that leads to the induction of specific IgE antibodies, which are then capable of cross-reacting with homologous food allergens.^{5,6} Asero *et al.* studied the association between

inhalation allergy duration and food allergy; they found that the incidence of food allergy increases over time from 1 to >20 years of inhalation allergy.⁶ We believe that our case was first sensitized to the bird and to the birdseed containing millet, then, after a lapse of several years, millet ingestion induced a severe food allergic reaction. The patient might not have been exposed to a sufficiently large quantity of allergen via inhalation to develop a severe allergic reaction, and it was not until she orally ingested a sufficient quantity of the homologous food allergen that the allergic reaction developed. Over the past 2 years, there has been a decrease in several of the patient's positive specific IgE antibodies for bird and birdseed, and it is likely that the discontinuation of exposure to particular allergens via inhalation might lead to a decrease in specific IgE antibodies against them.

Our literature search showed that this is the first such case reported from Japan, and the first dermatological case report of this condition. When dealing with a case of millet allergic reaction, clinicians should ask patients about any pets. The increased use of millet as birdseed and as human food due to the heightened interest in natural diets may increase the incidence of millet-related allergic reactions.

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