later, she experienced an itching sensation of her throat and a feeling of choking after going into a kitchen, unaware of the fact that *M. comosum* bulbs were being cooked in that room. On both occasions, prompt treatment with intramuscular injections of antihistamines and corticosteroids in an emergency unit relieved the symptoms. In the third episode, the patient had again urticaria and angioedema, which were controlled by treatment with parenteral corticosteroids. Initial symptoms reported by the patient were tingling on her tongue and mild throat tightness, occurring soon after dinner. After repeated questioning, she realized that her husband was eating 'lampascioni' during her meal, and she had inadvertently used the same spoon contaminated with the preserve oil in which the bulbs were plunged.

Muscari comosum raw and cooked bulb proteins were extracted as previously described (3). Protein contents were determined according to Bradford (6), and proved to be 2.13 and 0.62 mg/ml in extracts from raw and cooked bulbs, respectively. The sodium dodecylsulfate-polyacrylamide gel electrophoresis profile of raw and cooked bulb extracts showed similar bands ranging from 14 to 30 kDa, the most intense being the one at 29–30 kDa. Immunoblotting showed IgE reactivity in the patient's serum to a single band at approximately 29–30 kDa.

Skin prick tests with inhalant and food commercial extracts and diluted extracts of cooked and raw bulbs were performed, using saline solution and histamine (10 mg/ml) as negative and positive controls, respectively. Positive reactions to *Dermatophagoides pteronyssinus,* and extracts of raw *M. comosum* bulb (at both 1 : 100 and 1 : 1000 dilutions) and cooked bulb (1 : 100) were observed. Specific IgE to foods, including garlic, onion, and asparagus, was negative. Ten healthy volunteers were prick tested with extracts of cooked and raw bulb, both at dilution of 1 : 100, with negative results.

To our knowledge, this is the first description of an IgE-mediated reaction to *M. comosum* bulbs. In our patient symptoms developed even after inhalation of cooking vapors and ingestion of very low quantities resulting from a hidden source.

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Autoimmune urticaria at 3 years of age diagnosed by puncture skin test using autologous serum

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Key words: autoimmune urticaria; puncture skin test.

Functional autoantibodies against high affinity IgE receptor (FcεRI) or IgE, which can cause histamine release from mast cells and basophils, are found in a substantial subset of adults with chronic urticaria (1, 2). This may also be true for children with chronic urticaria (3); however, few reports are available, and its clinical features in children remain to be fully understood. An intradermal test using autologous serum is generally used to screen for functional autoantibodies (1–3). Here, we describe a 3-year-old boy with autoimmune urticaria diagnosed by puncture skin test using autologous serum.

The potential of puncture skin test using autologous serum to detect functional autoantibodies.

The patient had a 6-month history of daily short-lived urticaria at our outpatient allergy clinic. The eruption occurred on the skin of the face, trunk and extremities. In addition, compressing the skin invariably caused a wheal response. He had asthma with specific IgE against *Dermatophagoides farinae*, but no other allergies or angioedema. He had no family history of chronic urticaria or angioedema. The results of complete blood count, blood chemical variables, and tests for antinuclear and anti-DNA antibodies, and complement determinants (i.e. C3, C4, and CH50) were normal. No cause of urticaria could be identified. Ketotifen (60 μg/kg/day) markedly but incompletely suppressed widespread urticaria. No additional effects were seen with ebastine (0.25 mg/kg/day), prednisolone (1 mg/kg/day) or pranlukast (7 mg/kg/day). The patient thus received ketotifen (60 μg/kg/day) or ebastine (0.25 mg/kg/day) for over 2 years.

Puncture skin test was performed 2 days after medication was stopped. Autologous serum and 0.9% saline were separately placed on the volar forearm, and each site was slightly lifted with a 25 G injection needle. Fifteen minutes later, mean wheal diameter was measured; the results were 10 mm with autologous serum and 2 mm with 0.9% saline. After pretreatment with ketotifen (30 μg/kg), the response was abolished, but the dermatographism on the forearm did not change. Basophil histamine release test was performed using an HRT Shionogi

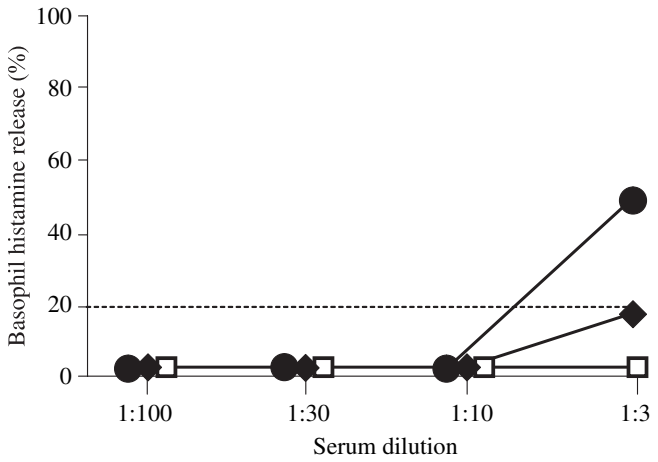


Figure 1. Basophil histamine release using inactivated sera from the patient (closed circles). Control sera, showing negative autologous serum skin test, were obtained from a 9-year-old girl with chronic urticaria (closed diamonds), and an allergic basophil donor (open squares). Dotted line indicates the cut-off point (20%) for this commercial assay kit (HRT Shionogi kit).

kit (Shionogi, Osaka, Japan) (4). Basophils were obtained from an allergic volunteer. Heated sera (56°C for 15 min) from the patient and the basophil donor released histamine at 54% and 0% total histamine content, respectively (Fig. 1). To distinguish between the roles of autoantibodies against IgE and FcεRI, human myeloma IgE (50 μg/ml; Yamasa Co., Choshi, Japan) was supplied to the patient's serum as an inhibitor of anti-IgE. However, it had no effect. This study was performed after obtaining informed consent from the patient's parents and control subjects.

We detected histamine-releasing autoantibodies in patient serum on autologous serum skin test and basophil histamine release test. This is the first report showing the potential of puncture skin test to detect functional autoantibodies. In addition, skin test after antihistamine treatment was useful to confirm the involvement of mast cell-derived histamine. Anaphylatoxins and autoantibodies to IgE are unlikely to be histamine-releasing factors, as serum heat-inactivation and supplied human myeloma IgE did not inhibit histamine release from basophils. Autoantibodies to FcεRI were thus suggested to be a histamine-releasing factor, and these are found most frequently in adults with autoimmune urticaria (1, 5). Antihistamine therapy did not achieve complete remission, as is also seen in adult patients with autoimmune urticaria (5, 6).

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Salbutamol, terbutaline and pirbuterol allergy in an asthmatic patient

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Key words: adverse effect; asthma; drug allergy; drug induced; IgE; short-acting β2-agonist.

We report a case of allergy to short-acting β2-agonists producing acute bronchoconstriction.

'Paradoxical' bronchoconstriction following inhalation of β2-agonists has been documented (1–3) and was ascribed to propellant, preservatives, osmolality and/or pH of the inhalant (4, 5). To our knowledge, bronchoconstriction to β2-agonists has not been described. We present a case with severe bronchoconstriction following inhalation of short-acting β2-agonists for which an immediate IgE-dependent mechanism is suspected.

A 41-year-old farmer with a 40-pack-year smoking history was admitted to our ICU for a near-fatal asthma. The asthma had started 3 years earlier. He had been treated with methylprednisolone 16 mg/day and formoterol. He had stopped salbutamol/albuterol as reliever medication a few months earlier, because of loss of perceived effect. Two weeks before admission, he developed an asthma exacerbation. He increased the number of daily formoterol puffs. On the morning