

Three months later, he underwent orthopedic surgery to rectify the orthopedic ailment and within 11 months he had a patch test.

The test used 30% of commercialized cefadroxil powder in white petrolatum and water. Pure white petrolatum and water served as a placebo. The test spot was taped-strapped 10 times prior to occluding the test materials for 48 h. The same test was performed on five healthy volunteers who had not had any prior exposure to cefadroxil and one healthy nonallergic volunteer with known prior exposure to cefadroxil. Test results on the patient showed strong positive reaction on hours 48, 72 and 96, either for cefadroxil in white petrolatum and in water. Both of the placebos were negative. The results on all of the six controls were negative.

DRESS syndrome is an idiosyncratic severe adverse drug reaction that begins acutely in the first two months after initiation of the offending drug. The diagnostic criteria encompasses: (1) cutaneous eruption; (2) absolute eosinophilia ($\geq 1500/\mu\text{l}$) with or without atypical lymphocytes; and (3) systemic involvement (lymphadenopathy ≥ 2 cm, AST $\geq 2\times$ normal, interstitial nephritis, interstitial pneumonitis, or carditis). Diagnosis is confirmed if three criteria are present (1, 2). Most commonly drugs are antiepileptic and sulfonamides. Less common offending drugs are allopurinol, gold salt, sorbinil, minocycline, zalcitabine, calcium channel blockers, ranitidine, thalidomide, or mexilatine (1, 2).

Nonimmediate type hypersensitivity reaction or a defect in ability to detoxify the toxic metabolic product is considered to be the pathogenesis of DRESS syndrome (1–3). Patch test is the safest skin test to prove nonimmediate type drug hypersensitivity, which should be performed between 6 weeks and 6 months after complete convalescence from the drug reaction (4). To avoid the relapse of severe drug reaction, such as in DRESS syndrome case, 1–10% of commercialized form of the drug, either in white petrolatum or in water is recommended (4, 5). Tape-stripping eight to 12 times can be performed to increase percutaneous drug penetration and avoid false negative (6).

The patient underwent the test 14 months after complete convalescence; to avoid a false-negative result, 30% of commercialized cefadroxil was used, and the spot was tape-strapped 10 times. Six months later, no relapse was observed. As far as we know, this is the first case of DRESS syndrome from cefadroxil confirmed by positive patch test.

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Allergy to *Muscari comosum* bulb

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Key words: bulbs; IgE-mediated hypersensitivity; *Leopoldia comosa*; *Muscari comosum*; urticaria angioedema syndrome.

Muscari comosum (*Leopoldia comosa*), commonly known as grape hyacinth, is a herbaceous perennial plant, growing from a bulb and spring-blooming, which is native to the Mediterranean area. The bulbs of *M. comosum*

Allergic reactions caused by ingestion of *Muscari comosum* bulbs or inhalation of their cooking vapors.

can be eaten cooked, usually after being boiled for a long time to reduce their bitter taste. The food use of these bulbs, originated from the ancient Greeks, is common in Turkey, Greece and Southern Italy and very popular in our region, where they are named 'lampascioni'. The genus *Muscari*, formerly included among *Liliaceae*, is included by other recent classification systems in *Hyacinthaceae* family (order: *Asparagales*) (1, 2). According to these systems, other families belong to monocotyledonous plant order *Asparagales*, such as *Alliaceae* (including onion and garlic) and *Asparagaceae* (e.g. the asparagus family), which were previously considered members of *Liliaceae*. Immunoglobulin E-mediated reactions after inhalation or ingestion of plant species from *Asparagales* have been documented (3–5). We present a patient who developed symptoms related to allergy to *M. comosum* bulbs on three different occasions. Only one of these episodes was provoked by the actual ingestion of bulbs.

The patient was a 32-year-old woman, with a history of perennial allergic rhinitis and atopic hand eczema. She reported that the first episode occurred 7 years earlier and presented with urticaria and angioedema, after eating *M. comosum* bulbs. As then, she cautiously avoided the ingestion of this food. Two years

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 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

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