

dermatitis, presented an anaphylactic reaction few minutes after eating pumpkin seeds. The prick-to-prick test with pumpkin seeds was negative, but a positive oral provocation test confirmed the diagnosis, as a severe reaction with urticaria, angioedema and asthma was induced after 10 g of pumpkin seeds. By history, he tolerated fruits belonging to the Cucurbitaceae family.

Sunflower seeds allergy is rarely described in the literature, particularly in children (1–3). This allergy has been reported previously in adults owning birds fed sunflower seeds (1). Allergy to sunflower oil had also been described (5, 6), but most patients with sunflower seeds allergy tolerate sunflower oil containing only traces of seed proteins. No pumpkin seeds allergy in children was previously described in the literature (4). However, these two foods are frequently consumed and can be hidden in a lot of recipes. Allergic reactions observed in our three patients can be graded as severe; thus, it is important to consider allergy to snack seeds (sunflower and pumpkin seeds), particularly in patients with an unclear history of anaphylaxis even in the presence of negative skin tests. A complete allergy work-up, including an oral provocation test, should be envisaged for the diagnosis of suspected snack seeds allergy followed, if the diagnosis is confirmed, by a strict eviction. As more health foods are trendy, these case reports documenting severe allergic reactions to seeds may gain importance, particularly in children.

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Short-term prophylactic treatment of hereditary angioedema with icatibant

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Keywords: bradykinin receptor-2 antagonist; hereditary angioedema; icatibant; prophylaxis.

Hereditary type 1 angioedema (HAE) due to quantitative deficiency of C1-esterase inhibitor (C1-INH) is a bradykinin-dependent disease that can be treated with icatibant (Jerini, Berlin), a novel bradykinin B2-receptor antagonist (1).

Patients with HAE can suffer bouts of angioedema with surgical or invasive medical procedures. In such cases a short-prophylactic treatment has to be considered (2, 3). There is no approved medication for this indication in Spain. Until now, patients in this situation were treated with intravenously administered C1-INH concentrate derived from human plasma.

Icatibant was successful in the prevention of angioedema after thyroid biopsy in hereditary angioedema.

We present a case of HAE in which a fine-needle aspiration and biopsy (FNA) of the thyroid gland was performed after prophylactic treatment with icatibant.

A 46-year-old woman was diagnosed as having type I HAE after 40 years of suffering bouts of angioedema and repeated crises of abdominal pain and vomiting. On four occasions, laryngeal oedema was suspected in episodes of facial and neck oedema with dyspnoea and a sensation of a foreign body in the pharynx. Some years ago, she had an intrauterine contraceptive device put in place and immediately presented severe oedema of genitalia with urine retention. She has presented facial oedema four times after dental treatments.

In the last 2 years she has suffered more than 25 bouts of angioedema per year. Her complement levels were: C4 0.02 g/l (0.1–0.45), C1-INH < 12.5 mg/dl (15–34) with 2.82% functional activity (70–130%).

Long-term prophylactic treatment with tranexamic acid (2 g/8 h) was not effective and treatment with androgens (stanozolol or danazol) was only partially effective and was not tolerated due to severe myalgias. The patient was treated until January 2009, with intravenously administered C1-INH concentrate derived from human plasma in the event of moderate or severe bouts of oedema. From January 2009, her oedema crises were treated with subcutaneous infusion of 30 mg of Icatibant with good results, similar to those obtained with C1-INH concentrate.

An euthyroid goitre was found, and an FNA was performed 1 year ago. The patient did not warn staff that she suffered from HAE because she thought a needle puncture would not be dangerous. She suffered neck oedema with dyspnoea 45 min after the procedure. A second FNA was recently indicated and, to prevent recurrence of oedema, following informed consent and administrative authorization for compassionate use, a subcutaneous infusion of 30 mg of icatibant was administered 60 min before the procedure. An ultrasound-guided FNA was conducted on a thyroid nodule and she

was discharged 3 h later without any oedematous reaction.

Icatibant is a selective antagonist of the bradykinin B2 receptor. It is a synthetic peptide with five nonproteinogenic aminoacids that increases its half-life. Previous studies have demonstrated that Icatibant relieves angioedema crises in patients with HAE (4). Subcutaneous administration has been shown to be quick at relieving symptoms and just as effective as intravenous infusion. This is a clear advantage over C1-INH concentrate, which has to be administered intravenously. Icatibant safety profile is good, being its main side effect local erythema, pain and itching that spontaneously resolve.

Icatibant has not been previously used as a prophylactic treatment. Patients with HAE commonly present angioedema after surgical or invasive procedures. The latest consensus document on HAE alerts to the need for prophylactic treatment to avoid this problem that can sometimes produce life-threatening reactions (3).

Although further studies are still needed, our data showed a potential future role of Icatibant for short-term prophylactic treatment in HAE.

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Efficacy of omalizumab in delayed pressure urticaria: a case report

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Keywords: asthma; basic immunology; basophil; dendritic cells; dermatology; drug allergy; food allergy; pharmacology; urticaria.

Although the exact mode of action is presently unknown, the efficacy of treatment of chronic urticaria with anti-IgE (Omalizumab, Xolair, Novartis) has been reported recently (1). We present the successful treatment of a patient with severe delayed pressure

urticaria (DLP) with omalizumab 150 mg every second week.

The patient is a now 39-year-old female with a 10-year case history of severe debilitating DLP, elicited by even slight pressure preventing the patient from normal daily activities such as manual work, horse riding, walking in high heels, bicycling, or marital activities. Initially, the patient presented with leucocytosis of unknown origin, which was normalized after treatment with azithromycin 500 mg for 3 days and positive test for autoimmune urticaria (3) (HR-Test, Reflab, Copenhagen, Denmark). Partial relief, only, was obtained with fexofenadine 540 mg [triple dose (2)] and azathioprine 100 mg per day was added for a period of 10 months. On this treatment, partial relief was obtained initially but the effect vanished over time. Azathioprine was substituted with cyclosporine 200–350 mg per day (3–5 mg/kg) with initial marked effect which declined over the next 2 years and was stopped due to side effects (lanugo hair, renal impairment) hereafter.

April 22, 2009: the patient still on fexofenadine was given 150 mg omalizumab subcutaneous (serum IgE 32 kIU/l) and already 2 days after this first injection the patient was totally free from any symptoms of DLP. The injection was repeated every second week and the patient is still free from any symptoms on the same dose today (July 2009) without any side effects. Serum IgE has gradually risen to now 184 kIU/l.

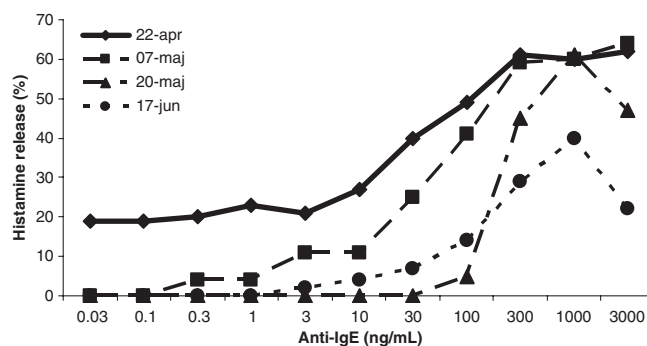


Figure 1 Changes over time in basophil releasability during treatment with omalizumab.