Should postviral anosmia be further investigated?

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Key words: hyposmia; meningioma; postviral anosmia; rhinitis.

Distortions in olfactory function are common in the general population, hyposmia affecting 16–19% and anosmia 0.5–2%. Three of the most common causes for the loss of smell are: chronic rhinosinusitis and nasal polyposis (1), head trauma and upper respiratory tract infection (URTI) (2). Some authors (3) have also reported higher odour thresholds in allergic rhinitis patients than in the control groups. Recently, Guilemany et al. (4) have demonstrated that persistent allergic rhinitis induces a moderate loss of the sense of smell mainly in the moderate-to-severe disease. Nevertheless, there are more than 200 conditions that have been associated with changes in olfaction, which explain that there could be more than one cause for the loss of smell in an individual patient. Allergologists, otolaryngologists and general practitioners very often have to manage patients with smell impairment related or not to acute and chronic nose inflammatory diseases.

The loss of the sense of smell is a very prevalent symptom in the general population. Postviral anosmia is one of the three main causes. We report a case of an anosmic patient related with two different aetiologies: postviral and an olfactory grove meningioma.

We present the case of a 43-year-old woman who visited our Rhinology Unit & Smell Clinic complaining of persistent anosmia that suddenly appeared during the winter of 2005 after three episodes of viral URTI. Past medical history was positive for hypotyroidism, which was well controlled with oral medication, and gestational diabetes in 1996, which was well controlled after giving birth. There was a negative clinical history of sinonasal or neurological symptoms, previous head trauma or ear–nose–throat surgery.

Physical examination was normal, except for lower right turbinate hypertrophy diagnosed by nasal endoscopy. Due to a suspected diagnosis of postviral anosmia, a smell test using the Barcelona Smell Test 24 (5) and a paranasal sinus and brain CT scan were performed following the smell protocol used by our institution. Subjective olfactometry confirmed anosmia (0% in odour detection, memory and identification), with no taste problems. Skin Prick test was negative for the most common aeroallergens. The CT scan showed no pathology in the paranasal sinuses while some osteolitic changes were observed in the ethmoid bone. Further imaging investigation of these osteolitic changes with MRI demonstrated a giant olfactory grove meningioma (55×42 mm) (Fig. 1). Three weeks after the diagnosis, resection of the lesion was performed via a transfrontal approach. While waiting for the indicated surgery, the patient developed visual loss in her right eye that completely recovered after surgery. After her last follow-up in September 2008, her only remaining complaint was persistant anosmia.

There are several important issues, which can be learnt from this case report. Despite other aetiologies, three main causes should always be investigated: nasal inflammatory diseases (allergic rhinitis and chronic rhinosinusitis, with or without nasal polyps), postviral anosmia and head trauma. Detailed clinical history, subjective olfactometry and imaging (e.g. CT scan or/and MRI) should always be performed in patients with anosmia to discriminate different potential life threatening causes such as intracranial tumours. In conclusion, because hyposmia and anosmia are highly prevalent symptoms in the general population and there could be more than one cause for the loss of smell, this case study demonstrates that patients experiencing these symptoms require the physician's special attention and further investigation must be performed.

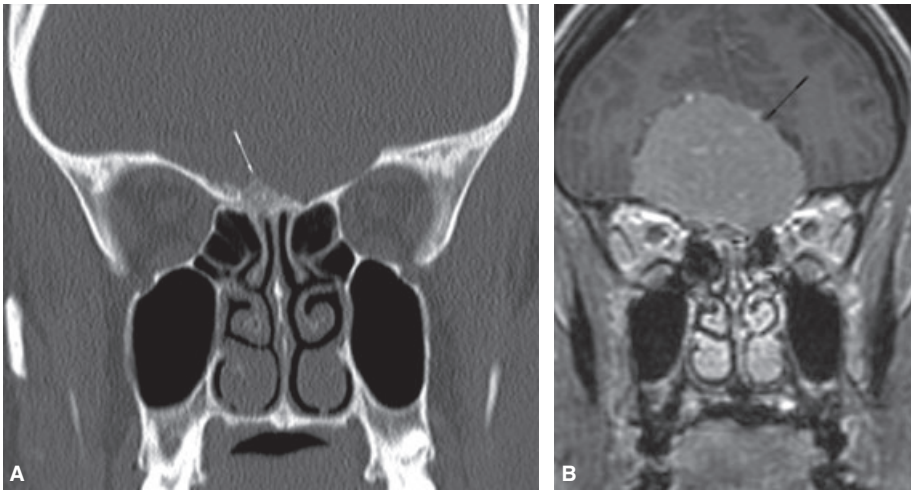

Figure 1. (A) Coronal CT image shows the focal hyperostosis in both cribiform plates (white arrow). (B) Contrast enhanced T1-weighted image shows a giant olfactory grove meningioma (black arrow). No pathology in nasal and paranasal regions was observed.

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Food allergy to chicken meat with IgE reactivity to muscle α-parvalbumin

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Key words: allergen; alpha-parvalbumin; chicken meat; chicken parvalbumin; food allergy.

IgE-mediated allergic food reactions provoked by consumption of chicken meat have been described in patients with egg allergy and bird feather hypersensitivity (1). Chicken serum albumin (Gal d 5), also known as α-livetin, has been shown to be the cross-reactive allergen (2). A few cases have been published concerning chicken meat allergy without sensitization to egg proteins. In these studies, the exact nature of the responsible allergens has not been defined (3, 4).

This is the first report of chicken meat allergy with α-parvalbumin identified as food allergen.

We report the case of a 54-year-old patient who experienced severe allergic reactions to poultry products. Swelling of tongue and oral mucosa, vomiting and hypotension occurred a few minutes after ingestion of chicken meat, turkey meat or chicken broth. The patient reported mild oral reactions when eating tuna and salmon. The patient tolerated chicken eggs.

Skin prick tests (SPT) performed with a series of commercial aeroallergens (ALK, Varennes en Argonne, France) were positive for tree and grass pollen. Skin prick tests with food allergens were positive with chicken meat, turkey meat and different fishes (tuna, salmon, cod and carp).

The total IgE level was 80 kU/l (Phadia ImmunoCAP System, Uppsala, Sweden). Specific IgE were positive for chicken meat (12 kU/l), turkey meat (6 kU/l), pork meat (1 kU/l), cod (3 kU/l), tuna (3 kU/l) and salmon (2 kU/l), but negative for egg yolk and egg white (< 0.35 kU/l).

Protein extracts were prepared from leg muscle of chicken and turkey. In IgE immunoblot analysis, a distinct 14 kDa protein band was detected in poultry extracts with patient serum (Fig. 1A). In addition, several addi-

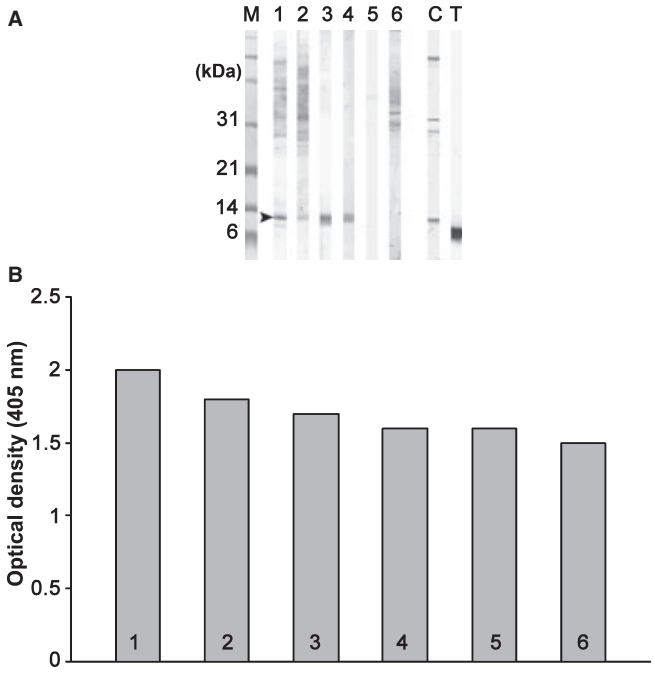

Figure 1. IgE analysis of chicken allergens. A. Allergens are detected at 14 kDa and > 30 kDa by the patient's IgE antibodies in chicken (1) and turkey (2) leg meat. IgE bind specifically to purified native (3) and recombinant (4) chicken α-parvalbumin, but not to tuna (5) β-parvalbumin. IgE reactivity to chicken leg extract is inhibited by preincubation with 20× excess of recombinant chicken parvalbumin (6). (C) Chicken parvalbumin is detected in total extracts using a commercial antibody (Abcam, Cambridge, UK). (T) Identity of purified tuna parvalbumin is confirmed by an anti-β-parvalbumin antibody (Swant, Bellinzona, Switzerland). B. IgE quantification by *enzyme-linked immunosorbent assay*. Similar IgE titers are obtained for native chicken (1), recombinant chicken (2) as well as for frog (3), cattle (4), horse (5) and pig (6) recombinant α-parvalbumins.