Dysgeusia Limited to Sweet Taste in Myasthenia Gravis

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Abstract

Total dysgeusia, an inability to interpret all of the basic tastes, often occurs with zinc deficiency. Partial dysgeusia (dissociation dysgeusia) is a rare inability to interpret a limited number of these basic tastes. We present the case of a patient with myasthenia gravis who became unable to discern sweet taste, but other basic tastes were unaffected. Such dysgeusia can be explained by obstruction of selective taste receptors in taste cells. We considered that this symptom was induced by an autoimmune mechanism related to myasthenia gravis.

Key words: dysgeusia, taste receptor, taste-modifying protein, myasthenia gravis, thymoma, autoimmune disorder

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Introduction

The brain recognizes different tastes by interpreting the 5 basic tastes (sweet, salty, sour, bitter and umami) mediated via a series of chemical reactions in the taste cells of taste buds (1). Total dysgeusia, an inability to interpret all the basic tastes, often occurs with zinc deficiency. Partial dysgeusia (dissociation dysgeusia) is a rare inability to interpret a limited number of these basic tastes (2). We present the case of a patient with myasthenia gravis who became unable to discern sweet taste, but the other basic tastes were unaffected. Such dysgeusia can be explained by obstruction of selective taste receptors in taste cells. We considered that this symptom was induced by an autoimmune mechanism related to myasthenia gravis.

Case Report

A 38-year-old man suddenly noticed peculiar dysgeusia in November 1999. He described sweet-tasting foods as having no taste, like water, but was able to discern salty, sour, umami and bitter tastes. He visited a few local hospitals, but the cause of dysgeusia remained unclear. Serum zinc levels were normal, and administration of zinc sulfate failed to improve dysgeusia. Dysgeusia persisted for 8 years. Bilateral blepharoptosis appeared in December 2006. Blepharoptosis was less severe in the morning and became aggravated in the evening. Diplopia developed in January 2007, and the patient was admitted to our hospital for therapy of both dysgeusia and blepharoptosis in February 2007.

On admission, body temperature was 36.4°C, heart rate was 68 beats/min, and blood pressure was 130/86 mmHg. Physical and neurological findings were uninformative except for bilateral blepharoptosis. Blood cell counts were all within normal limits. Serum sodium, potassium, creatinine, urea nitrogen and liver enzyme levels were normal, as was blood glucose. Serum zinc level was 95 μg/dl (normal, 65-110 μg/dl). Anti-acetylcholine receptor antibody (anti-AchR Ab) level was elevated to 43.0 nmol/l (normal, <0.2 nmol/l). Anti-nuclear antibodies, and antibodies to Sjögren’s syndrome A and B were negative. Serum thyrotropin, free triiodothyronine, and free thyroxine levels were normal. Anti-thyroid peroxidase, and anti-thyroglobulin were negative. Blepharoptosis immediately resolved with administration of edrophonium chloride. Chest radiography yielded normal results, but axial computed tomography showed an anterior mediastinal mass lesion suggesting thymoma. The tumor (7.0×5.0 cm) was surgically resected and pathological findings confirmed a diagnosis of lymphocyte-predominant benign thymoma. Blepharoptosis improved following surgery, but dysgeusia persisted. Serum anti-Ach Ab levels remained high within 6 months after operation (range, 31.0-61.0 nmol/l).
Discussion

Several different forms of dysgeusia have been described. The most common cause of dysgeusia is taste cell damage by zinc deficiency (2), in which total dysgeusia occurs. We have previously described another form of dysgeusia in which all food tasted sweet in a case of lung cancer associated with syndrome of inappropriate antidiuretic hormone secretion (SIADH). We speculated that the pathophysiology in that case involved secretion of a taste-modifying substance from the lung cancer (3). Differing from these other forms of dysgeusia, dysgeusia in the present case was limited to sweet taste only, and salty, sour, umami and bitter tastes were unaffected (dissociation dysgeusia). Sweet was not able to be tasted over the entire lingual surface, oral cavity and pharynx. Dysgeusia appeared suddenly and persisted for 8 years. Serum zinc level was normal, and SIADH was not present.

The patient in the present case later developed bilateral blepharoptosis, and myasthenia gravis with thymoma was diagnosed. We consider that dysgeusia in this case was related to the pathophysiology of myasthenia gravis. Taste disorder rarely occurs in myasthenia gravis, but 6 similar cases have been reported (4-8). Thymoma is associated in all of the cases. In 2 cases from these reports, dysgeusia was limited to sweet taste, as in the present case (4, 6). In one case, the sensation was lost for sweet and diminished for other taste modalities (8). Taste disorder in all these cases improved with therapy for myasthenia gravis, although dysgeusia was not improved after thymectomy in our case.

The symptom in the present case can be explained by taste receptor disorder. Sweet stimuli, such as sugar or artificial sweeteners, have recently been shown to bind to a G protein-coupled receptor (1). An unknown antibody to sweetness receptors may be produced by thymoma, binding to sweetness receptors. Recently, the taste cells were found to be one of the peripheral targets for leptin, a hormone primarily produced in adipose cells. Leptin appears to act as a modulator of sweet taste within the signal transduction system of sweetness receptors (9). Therefore, the alternative explanation for dysgeusia in our case is that thymoma produces antibodies binding to leptin receptors on the sweet taste cells. Dysgeusia in our case may not have been improved by thymectomy for following reason. Considering that serum anti-Ach Ab levels remained high even after thymectomy, unknown antibodies to sweetness receptors also seemed to remain.

Dissociation dysgeusia limited to sweet taste has also been reported in Guillain-Barré syndrome (10). Both myasthenia gravis and Guillain-Barré syndrome are autoimmune diseases involving the production of autoantibodies. In conclusion, myasthenia gravis may cause dysgeusia, particularly dissociation dysgeusia limited to sweet taste.

References