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【 CASE REPORT 】

Peculiar Unpleasant Dysgeusia as the Sole Initial Symptom of Guillain-Barré Syndrome

Takaaki Nakamura, Kenichi Tsukita, Asako Suzuki, Genya Watanabe, Ryuhei Harada, Emiko Kawasaki, Tetsuya Chiba, Ryo Sugaya and Yasushi Suzuki

Abstract:

Dysgeusia is rare in Guillain-Barré syndrome, particularly as the initial symptom. We herein report the case of a 59-year-old woman who presented with only dysgeusia as the initial symptom of Guillain-Barré syndrome, followed by gradually worsening muscle weakness and bilateral sensory disturbances in the extremities. Her dysgeusia was so unpleasant that she could not eat anything, so she received nasogastric tube feeding without dysphasia. We speculate that the dysgeusia in our patient was mainly caused by inflammation of the chorda tympani nerves. Guillain-Barré syndrome should be considered a possible cause of acute dysgeusia.

Key words: dysgeusia, taste disorder, Guillain-Barré syndrome, chorda tympani nerve

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Introduction

The clinical features of Guillain-Barré syndrome (GBS) are characterized by progressive symmetric muscle weakness. Cranial nerve impairment occurs frequently, and taste disorders appear in 1-2% of patients (1, 2) but are rare as the initial symptom.

We herein report a patient who presented with peculiar unpleasant dysgeusia as the initial symptom of GBS.

Case Report

A 59-year-old woman initially presented with dysgeusia (day 1). She described her dysgeusia as, “Any food or drink, even water, tastes unpleasant and induces nausea.” On day 3, her dysgeusia became so unbearable that she could not eat anything. Furthermore, she noticed mild bilateral dysesthesia of the fingers and mild bilateral muscle weakness in the lower extremities. On day 5, she began having difficulty walking and was admitted to our hospital.

She did not have any antecedent infectious illness episodes. Her medical history consisted of hypertension and chronic gastritis. She used nifedipine, vonoprazan, telmisartan, hydrochlorthiazide, and itopride daily. She did not have any history of changing medicines in the past few years. A neurological examination on admission revealed slight left facial palsy, muscle weakness in the extremities [manual muscle test (MMT): grade 3 in the lower extremities and grade 4 in the upper extremities], bilateral areflexia in the lower extremities, and dysesthesia in the upper distal extremities. She did not present with hyperacusis, a fever, rash, myalgia, or arthralgia.

A qualitative taste assessment using filter paper revealed a diminished gustatory sense for the four basic taste modalities (sweet, salty, bitter, and sour) in the anterior part of the tongue, whereas all taste modalities were preserved in the posterior part. Laboratory tests showed that complete blood count and blood chemistry results, including serum concentrations of zinc, vitamin B12, folic acid, blood urea nitrogen, creatinine, and C-reactive protein levels, were within normal ranges. A test of serum for anti-ganglioside antibodies on day 6 was mildly positive for anti-GM1 IgM, anti-GM1 IgG, anti-GD1a IgG, anti-GD1b IgG, anti-GD3 IgG, anti-GT1b IgG, and anti-GalNAc-GD1a IgG. Other serum autoantibodies, including anti-nuclear antibody, myeloperoxidase-antineutrophil cytoplasmic antibody, proteinase 3-antineutrophil cytoplasmic antibody, and anti-cyclic

Department of Neurology, National Hospital Organization Sendai Medical Center, Japan
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Correspondence to Dr. Takaaki Nakamura, takaaki@med.tohoku.ac.jp

Table 1. **Nerve Conduction Study Findings in Left Upper and Lower Extremities.**

CMAP	Distal latency (ms)	Velocity (m/S)	Amplitude (mV)	Duration and Phase
Median	cannot be assessed due to carpal tunnel syndrome			
Ulnar	**3.5**	58.6	**3.4**	normal
Tibial	**7.7**	44.8	**1.8**	prolonged, polyphasic
Peroneal	**7.2**	49.6	0.9	prolonged, polyphasic
SNAP	Peak latency (ms)	Velocity (m/S)	Amplitude (mV)	Duration and Phase
Medial	not evoked			
Ulnar	not evoked			
Sural	4.1	44.6	9.6	normal

Abnormal data by the standards of our hospital are shown in boldface.
CMAP: compound muscle action potential, SNAP: sensory nerve action potential

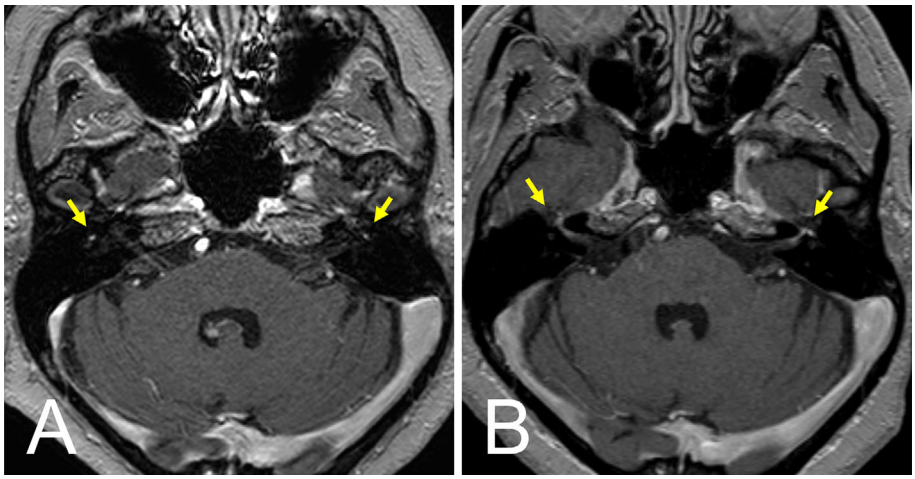

Figure. **MRI showed a high signal on contrast-enhanced T1-weighted imaging in the bilateral tympanic segment of the facial nerves (A, arrows) and geniculate ganglions (B, arrows).**

citrullinated antibody, were negative.

A cerebrospinal fluid study on day 6 showed a normal cell count (<1/μL) and elevated protein level (130 mg/dL). A nerve conduction study revealed mixed axonal and demyelinating neuropathy, especially in the motor nerves of the lower extremities (Table 1). Magnetic resonance imaging (MRI) on day 6 showed hyperintensities in the bilateral geniculate ganglions and the tympanic segment of facial nerves on contrast-enhanced T1-weighted imaging (Figure). Chest and abdominal contrast-enhanced computed tomography showed no remarkable findings.

She received intravenous immunoglobulin (20 g/day, 5 days, 2 cycles for 2 months) from day 6. Her dysgeusia responded well to the treatment and disappeared on day 30. Her muscle weakness had rapidly worsened by day 7 and reached clinical nadir on day 9 (MMT: grade 2 in the lower extremities and grade 3 in the upper extremities). Shortly after the second intravenous immunoglobulin infusion (day 36-40), her symptoms began to recover. She was again able to walk independently on day 120, although mild muscle weakness remained in the lower extremities (MMT: grade 4).

Discussion

Taste disorders are clinically classified based on symptoms as follows: hypogeusia (diminished sense of taste), ageusia (complete loss of taste), hypergeusia (enhanced gustatory sensitivity), phantogeusia (spontaneous abnormal taste without external stimulus), and dysgeusia (distortion of the sense of taste) (3); however, this classification has not been clearly established, and these terms are sometimes used interchangeably.

Dysgeusia is a symptom commonly caused by certain conditions, such as zinc deficiency or exposure to certain drugs, but is rare in GBS. To our knowledge, seven cases of GBS in which taste disorders were the initial symptom have been reported (1, 4-7; Table 2). Among them, only one presented with dysgeusia as the initial symptom, accompanied by facial sense disturbances (4). This is the first case in which a patient presented with dysgeusia alone as the initial symptom.

In these reported cases, some taste impairments were accompanied by facial weakness or facial sense disturbance, while others were not. In our patient, the area of dysgeusia corresponded to the distribution of the chorda tympani

Table 2. Clinical Features of Patients with Taste Disorder as the Initial Symptom of GBS.

Reference	Age/ gender	Clinical features					
		Taste impairment		Area of taste impairment	Facial weakness	Facial sensory disturbance	Treatment responsiveness of taste
		Dysgeusia	Decrease or loss of taste				
4	30/M	+	n.m.	n.m.	+	+	partial
6	64/M	-	+	anterior part	-	+	good
1	45/M	-	+	n.m.	+	+	good
	24/F	n.m.	+	n.m.	+	-	n.m.
	50/F	n.m.	+	n.m.	+	+	n.m.
5	23/F	-	+	n.m.	-	-	good
7	47/F	n.m.	+	anterior part	-	-	good
This case	67/F	+	+	anterior part	+	-	good

M: male, F: female, n.m.: not mentioned

nerves, without facial weakness. Therefore, we suspect that the dysgeusia in our patient was mainly caused by the impairment of the chorda tympani nerves. Considering that mild facial palsy appeared a few days after the onset of the taste disorder, we speculated that the inflammation started from the chorda tympani nerves and spread to the proximal or distal parts of the facial nerves. The enhancement of these nerves on MRI might have reflected inflammation, although the labyrinthine segment or more distal parts of the facial nerve are known to be occasionally enhanced in healthy subjects (8).

Dysgeusia in our patient was impressively unpleasant. Taste disorders are generally considered to cause a low quality of life. However, we were unable to find any previous reports showing dysgeusia originating from a neurological disease that was so severe it prevented the patient from eating in order to avoid nausea. Although why our patient presented with such strongly unpleasant dysgeusia is unclear, we have speculated about possible mechanisms. One involves impairment of the chorda tympani nerves partially interfering with nerve conduction, such that the sweet and salty taste bud stimuli suffered greater interference than the sour and bitter ones, which likely produced an unpleasant taste (9). Alternatively, inflammatory or immune-associated molecules, such as cytokines, might have affected the taste buds and induced dysgeusia (10). Given that the sense of taste is modified by many complex factors, such as olfactory perception, drugs, and psychological effects, any or all of these factors might have influenced her symptoms.

Regardless of the mechanism underlying the dysgeusia, this phenomenon may hamper the diagnosis of GBS or other taste disorders. To avoid overlooking such atypical GBS cases, it is important to pay attention to the appearance of other neurological symptoms and to check whether the onset is acute or not.

GBS can cause strongly unpleasant dysgeusia as the initial symptom and should be considered as a differential diagnosis of acute-onset taste disorder.

The authors state that they have no Conflict of Interest (COI).

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