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【 CASE REPORT 】

# Sensory Ataxic Guillain-Barré Syndrome with Dysgeusia after mRNA COVID-19 Vaccination

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**Abstract:**
Guillain-Barré syndrome (GBS) has occasionally occurred in people who have received coronavirus disease 2019 (COVID-19) vaccines. Dysgeusia is rare symptom of GBS. We herein report a rare case of sensory ataxic GBS with dysgeusia just after the second dose of the Pfizer-BioNTech COVID-19 vaccine. Although autoantibodies against glycolipids were not detected, immunotherapy with intravenous immunoglobulin and methylprednisolone pulse therapy effectively ameliorated the symptoms. Our report suggests that the COVID-19 vaccine may induce various clinical subtypes of GBS, including a rare variant with sensory ataxia and dysgeusia.

**Key words:** vaccination, COVID-19, Guillain-Barré syndrome, sensory ataxia, dysgeusia

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## Introduction

Guillain-Barré syndrome (GBS) has occasionally occurred in people who have received coronavirus disease 2019 (COVID-19) vaccines. In general, patients with GBS rarely show taste disturbance (approximately 1%) (1).

We herein report a rare case of sensory ataxic GBS with dysgeusia just after receipt of the second dose of the Pfizer-BioNTech COVID-19 vaccine.

## Case Report

A 70-year-old Japanese man received the second dose of the Pfizer-BioNTech COVID-19 vaccine 23 days after the first dose. The next day, he noticed bilateral distal leg paresthesia, gait disturbance, and constipation. On the fifth day, bilateral leg unsteadiness and finger paresthesia developed. On the sixth day, tongue paresthesia and dysgeusia (bitter aftertaste) appeared, although his sense of smell remained normal, and he still tasted sweetness, sourness, and saltiness. These symptoms gradually worsened. He was finally admitted to our hospital by wheelchair on the 14th day.

Neither antecedent infection nor inflammatory findings were observed. Except for dysgeusia and tongue paresthesia, a cranial nerve examination was normal, including findings for his sense of smell and facial muscle strength. He showed slight proximal weakness in the lower extremities (grade 4+/5 on the Medical Research Council scale), hand-grip strength of 17.6/20.9 kg, diminished tendon reflexes, distal paresthesia, hyperalgesia, and severely impaired position and vibration sense. We considered sensory ataxia as the main cause of his gait disturbance. In addition, he had mild signs of dysautonomia, i.e. constipation and nocturia.

Laboratory tests showed normal serum levels of minerals and vitamins, such as iron, calcium, magnesium, copper, zinc, vitamin $B_1$, vitamin $B_2$, vitamin $B_{12}$, and folic acid. There was no evidence of diabetes mellitus or collagen diseases, including Sjögren's syndrome. A cerebrospinal fluid (CSF) analysis revealed albuminocytological dissociation with a normal cell count (2 cells/μL) and elevated protein level (117 mg/dL), which was consistent with GBS. Serum

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**A**

**Motor Nerve Conduction Study**

| Site/Muscle | Latency (ms) | Amplitude (mV) | Duration (ms) | Velocity (m/s) | Stimulation (mA) |
|---|---|---|---|---|---|
| Median - APB | | | | | |
| Wrist | 5.00 | 5.74 | 7.75 | | 30 |
| Elbow | 10.50 | 3.94 | 9.40 | 38.2 | 30 |
| Ulnar - ADM | | | | | |
| Wrist | 2.90 | 11.20 | 8.10 | | 20 |
| Below-elbow | 7.30 | 8.09 | 8.40 | 44.3 | 40 |
| Above-elbow | 9.00 | 7.24 | 8.75 | 29.4 | 20 |
| Axilla | 11.20 | 5.98 | 9.10 | 43.2 | 80 |
| Erb's point | 15.40 | 4.29 | 10.70 | 65.5 | 100 |
| Tibial - AH | | | | | |
| Ankle | 3.40 | 4.34 | 7.65 | | 80 |
| Knee | 13.10 | 1.70 | 13.20 | 38.1 | 60 |
| Fibular - EDB | | | | | |
| Ankle | 5.20 | 3.39 | 5.75 | | 30 |
| Below-knee | 13.80 | 1.89 | 7.00 | 33.7 | 50 |
| Knee | 16.10 | 1.34 | 7.05 | 33.5 | 40 |

**Sensory Nerve Conduction Study**

| Nerve | Latency (ms) | Amplitude (μV) | Velocity (m/s) | Stimulation (mA) |
|---|---|---|---|---|
| Median | 3.06 | 3.40 | 40.2 | 20 |
| Ulnar | 2.38 | 3.10 | 45.8 | 20 |
| Sural | 3.30 | 0.80 | 45.5 | 20 |

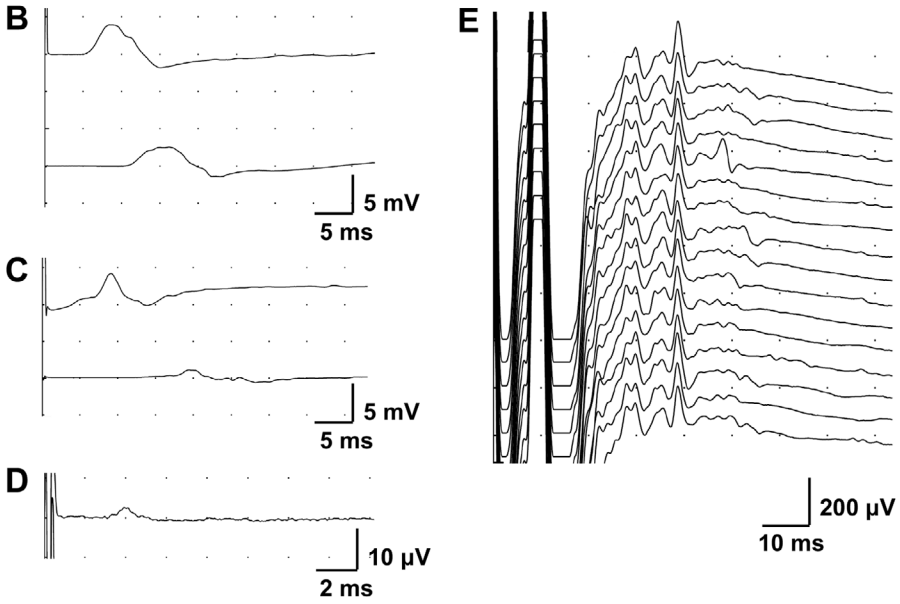


**Figure 1.** **Nerve conduction study findings. A: Nerve conduction studies diffusely showed an increased stimulation threshold, conduction delay, partial amplitude reduction, and increased temporal dispersion, which indicated sensorimotor demyelinating polyneuropathy. B: Median motor conduction study. C: Tibial motor conduction study. D: Median orthodromic sensory conduction study. E: Tibial F-wave study. Abundant A-waves made the accurate identification of the F-waves difficult. APB: abductor pollicis brevis muscle, ADM: abductor digiti minimi muscle, AH: abductor hallucis muscle, EDB: extensor digitorum brevis muscle**

immunoglobulin (Ig)M and IgG autoantibodies against glycolipids (GM2, GM1, GD1a, GalNAc-GD1a, GD1b, GT1a, GT1b, GQ1b, galactoceramide, GM1/GD1a complex, and GM1/GT1a complex) were negative. Serological evidence of recent infections by *Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae,* Cytomegalovirus, and Epstein-Barr virus were not obtained. Antibodies against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) spike proteins were positive in the serum (1,370 U/mL) and CSF (10.2 U/mL), whereas antibodies against SARS-CoV-2 nucleocapsid proteins were negative in the serum and CSF, indicating evidence of COVID-19 vaccination but not infection.

Nerve conduction studies performed on the 18th day (Fig. 1) suggested demyelinating neuropathy. Electrogustometry (EGM) is a clinical tool that has been well established to estimate the detection threshold of taste (2). EGM indicated an improvement in thresholds along with an improvement in dysgeusia (Fig. 2). The blink reflex was normal. Accordingly, he was given a diagnosis of GBS follow-

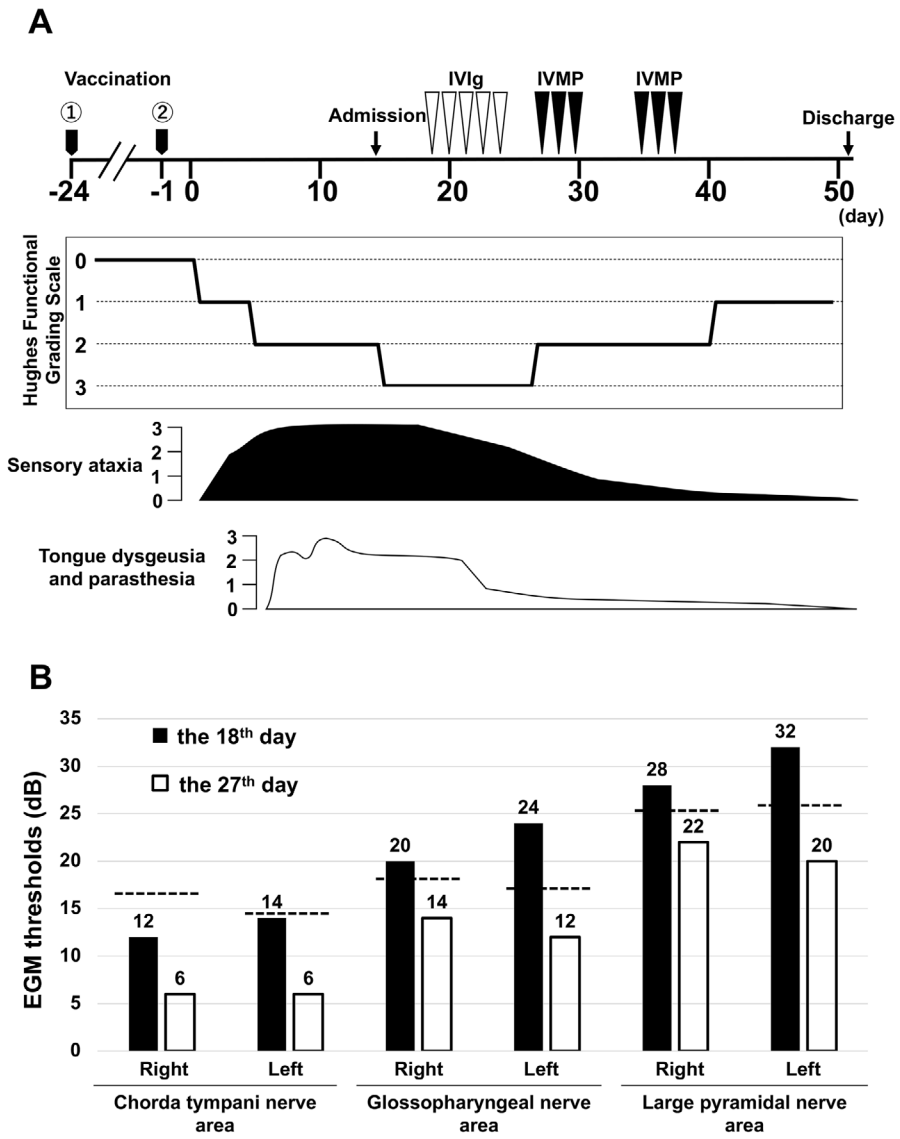


**Figure 2.** **Clinical features. A: Clinical course and treatments. IVIg: intravenous immunoglobulin therapy (400 mg/kg/day), IVMP: methylprednisolone pulse therapy (1,000 mg/day). Sensory dysfunction was evaluated as follows: 0=normal, 1=mild, 2=moderate, 3=severe. B: Electrogustometry (EGM) thresholds. Black column, pre-treatment data from the 18th day; white column, post-treatment data from the 27th day. Dotted lines indicate thresholds in each area (9).**

ing COVID-19 vaccination and treated with intravenous immunoglobulin (IVIg, 400 mg/kg/day for 5 consecutive days) on the 20th day. Dysgeusia with tongue paresthesia rapidly decreased, which was confirmed by follow-up EGM on the 27th day (Fig. 2). However, sensory ataxia was not fully improved. Because we postulated that immune-mediated inflammation rather than molecular mimicry was the more likely mechanism underlying these symptoms, we added treatment with methylprednisolone pulse therapy (IVMP, 1,000 mg/day for 3 consecutive days), which enabled him to walk without assistance.

## Discussion

This is the first case report of sensory ataxic GBS with dysgeusia following COVID-19 vaccination. To date, 36 cases of GBS after COVID-19 vaccination have been reported. None of the patients had taste impairment, although the facial nerve was often affected. Sensory ataxic GBS is often associated with autoantibodies against GQ1b, GT1a, or GD1b; it is characterized by demyelination of large sensory fibers (3). This type of neuropathy has also been reported in COVID-19-associated GBS (4). Taste disorder is classified into ageusia, hypogeusia, hypergeusia, dysgeusia, and phantogeusia. Although the precise mechanism underlying dysgeusia is unclear, elevated taste thresholds are observed in ageusia, hypogeusia, and dysgeusia (5), indicating that taste disorders constitute a spectrum of impairment in the facial and glossopharyngeal nerves. In fact, taste disturbance in GBS is usually accompanied by facial palsy (6). However, our patient did not have facial weakness or an abnormal blink reflex, suggesting that the nerve involvement was re-

stricted to the chorda tympani, a branch of the facial nerve containing small, thinly myelinated taste fibers. Taste disturbance is a well-known symptom of COVID-19, presumably due to direct tissue invasion by SARS-CoV-2, which is distinct from the mechanism in our patient (7). Patients with GBS after COVID-19-vaccination have been seronegative for antibodies observed in conventional GBS, probably because the SARS-CoV-2 spike proteins produced by the COVID-19 vaccine are not substantially similar to any known human antigens (8). In our patient, no serum autoantibodies against glycolipids were detected, and IVMP was more effective than IVIG, suggesting the probability of immune-mediated inflammation mechanism rather than molecular mimicry. Recent reports have suggested that all GBS subtypes can be expected in COVID-19-associated GBS. Similarly, this case expands the clinical phenotypes of GBS after COVID-19 vaccination, which can present with sensory ataxia and dysgeusia, symptoms found in conventional GBS.

The benefit of vaccination is undoubtedly significant to prevent COVID-19. However, we also should pay attention to neurological complications associated with COVID-19 vaccination, although our report did not clearly demonstrate causality between COVID-19 vaccination and GBS.

## Conclusion

This case indicates that COVID-19 vaccination-associated GBS can present with various symptoms of conventional GBS, including rare variants with sensory ataxia and dysgeusia.

Informed consent was obtained from the patient.

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

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