Fisher Syndrome with Taste Impairment

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Abstract

A 60-year-old woman was admitted to our hospital with a two day history of truncal ataxia and diplopia. Three days after admission, complete paresis of eye movements, left ptosis, taste impairment and absence of deep tendon reflexes appeared. The patient displayed normal facial movements; however, she reported decreased sensations of sweet and salty tastes. Anti-GQ1b antibodies were detected in the serum, and Fisher syndrome was therefore diagnosed. Intravenous immunoglobulin was administered starting five days after admission, with limitations of eye movements, areflexia and taste impairment showing improvements by 12 days after onset. Taste disturbance is rare in patients with Fisher syndrome. In this case, we hypothesize that autoantibodies may have targeted antigens in the chorda tympani, glossopharyngeal nerve or taste buds.

Key words: Fisher syndrome, taste impairment

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Introduction

Taste impairment shows a frequency of 0.6-2% in patients with Guillain-Barre syndrome (GBS) (1, 2) and it appears to be even more rare in patients with Fisher syndrome (3). We herein report a case of Fisher syndrome associated with taste impairment and discuss the possible mechanisms.

Case Report

A 60-year-old woman was admitted to our hospital with an unsteady gait. She had developed an upper respiratory infection 11 days earlier. She had a history of hypertension and ventricular tachycardia, and had undergone pacemaker implantation. On admission, her temperature was 36.5°C, her heart rate was 80 beats/min, her respiratory rate was 24 breaths/min and her blood pressure was 150/90 mmHg. Other than the presence of the implanted cardiac pacemaker, cardiac, lung, and abdominal examinations yielded normal results. The patient’s pupils measured 3.5 mm in diameter, with prompt reactivity to light and a normal near response. Bilateral horizontal gaze nystagmus was evident, and the patient’s extraocular movements showed slight limitations of abduction in the right eye. Her deep tendon reflexes were symmetrically brisk without Babinski sign. The patient’s motor power was normal; however, her gait was ataxic. No sensory disturbances were evident. Three days after admission, the patient’s eye movements became totally fixed, left ptosis appeared and the deep tendon reflexes became almost non-existent. Interestingly, the patient claimed to be unable to differentiate between sweet and salty tastes. The mucosa of the tongue appeared normal.

An examination of the cerebrospinal fluid (CSF) revealed the following results: 1 cell/μL, 67 mg/dL of glucose, and 46 mg/dL of protein. Brain computed tomography revealed no abnormalities. Immunoglobulin (Ig)G anti-GQ1b antibodies were detected in the patient’s serum, and Fisher syndrome was diagnosed.

Electrophysiological testing of the blink reflex did not show any abnormalities. Due to the reported changes in gestation, quantitative analyses of taste were performed. The filter-paper disk method was used to evaluate sweet, salty, sour and bitter tastes. Only salty taste showed reduced sensitivity in the anterior two-thirds and posterior one-third of the tongue. Intravenous immunoglobulin (16 g/day, five days) was administered starting five days after admission. The impairment of sweet taste improved within seven days after onset, while the impairment of salty taste improved within twelve days after onset. The patient’s clinical symptoms, other than truncal ataxia and diplopia, also improved, and she was discharged eighteen days after admission.
Fisher syndrome accompanied by taste impairment is rare, with only a single case (3) having been reported in the literature. Facial palsy is a major complication of GBS, and impairment of taste represents one symptom of facial nerve dysfunction. Taste disturbance in GBS may be attributable to involvement of the chorda tympani, trigeminal nerve, or both (2, 4).

In GBS, large and heavily myelinated fibers become demyelinated. The fibers subserving taste are supposed to be small and thinly myelinated. The facial nerve predominantly comprises large myelinated fibers, while the chorda tympani is made up almost exclusively of small fibers (5). This is presumably why taste impairment in GBS is so rare.

The actual incidence of the taste impairment in GBS may well be underestimated. Nishijima et al. (6) evaluated taste thresholds in 10 cases of GBS using electrogustometry and, found an impaired taste sensation in six cases. Only two of the 6 patients with taste impairment had complained of taste impairment, thus indicating that this symptom may be more frequent than reported in patients with GBS. Electrogustometry is useful for making a precise evaluation of taste; however, it is frequently unavailable in ordinary neurological clinics.

Uchibori et al. (3) reported a case of Fisher syndrome accompanied by taste loss without facial palsy, similar to the present case. They suggested that the presence of specific antibodies, including anti-GQ1b antibodies, may affect peripheral nerves associated with taste and taste buds in Fisher syndrome patients showing taste impairment.

In the present case, an impairment of sweet and salty tastes was found as a dissociated taste impairment without facial motor palsy. Taste buds contain various sorts of taste receptors and are distributed over the surface of the tongue. Taste receptors differ in quality, with sweet taste requiring G protein-coupled receptors and salty taste using ion channel receptors. The physiological structure of taste may be associated with a dissociated taste impairment in the present case. The impairment of taste recovered fully within a short duration. This is because taste receptors turn over in as little as 10 days (7-9). Taste impairment in this case may have resulted from unidentified antibodies or an unknown function of anti-GQ1b antibodies in the taste buds themselves that resulted in a short duration and distribution of taste impairment without facial palsy.

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References