Trigeminal Neuropathy Accompanied by a Pontine Lesion on MRI

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Abstract

A 63-year-old man presented with the loss of the sensations of pain and temperature sensation in the right facial region innervated by the trigeminal nerve (V1 to 3). He showed abnormal lesions in the pons and the trigeminal nerve on magnetic resonance imaging (MRI). He had recurrent herpes in the nasal cavity, and a history of left facial palsy. We herein present the unique MRI findings and suggest that herpes simplex infection may cause trigeminal neuropathy. This is the first reported case of dissociated trigeminal neuropathy with herpes simplex infection which was accompanied by a pontine lesion on MRI.

Key words: dissociated trigeminal neuropathy, herpes simplex virus, MRI, pontine lesion

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Introduction

The trigeminal nerve is the largest of the 12 cranial nerves. It carries the sensory input from the supratentorial dura mater, face, cornea, and the nasal, oral, and sinus mucosa (1). The trigeminal nerve has three main branches distal to the trigeminal ganglion: the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves, of which the latter also carries efferent motor signals to the muscles of mastication (1). In the brainstem, the trigeminal nerve has three sensory nuclei and a motor nucleus: a principle sensory nucleus, which mediates tactile sensation; a spinal trigeminal nucleus (STN), which mediates pain and temperature sensation; a mesencephalic nucleus, which contains the unipolar first-order ganglionic cells that receive proprioceptive input from the V3; and a motor nucleus, which lies medially to the principle sensory nucleus in the mid-pons (1). The complex anatomy of the trigeminal nerve makes it difficult to identify the foci that cause trigeminal neuropathies (TNOs). A TNO is distinct from trigeminal neuralgia (TNA), which is characterized by facial pain, but without sensory impairment or motor weakness. In a TNO, numbness or weakness usually appears due to the destruction of neurons. In TNA, the neurons are usually preserved, although their myelin sheaths may be destroyed (1). We herein present the first report of a patient with dissociated trigeminal sensory neuropathy, which was accompanied by a pontine lesion on MRI.

Case Report

A 63-year-old man noticed right palatine numbness after the appearance of herpes in his nasal cavity. It spread to his right chin, temple and forehead over several days. He was admitted to our hospital on the 14th day after the onset of symptoms. He had a history of left facial palsy and recurrent herpes in the oral mucosa and nasal cavity, but no history of hypertension. There was no remarkable family history. His blood pressure was 126/78 mmHg. His neurological examination revealed right facial dysesthesia, and the impairment of pain and temperature sensation in the V1-3 region. However, his facial tactile sensation was intact and symmetrical. He also reported right dysgeusia. The other cranial nerves were demonstrated normal function. An examination for motor function, including masseter function, was normal. A blood chemistry analysis was unremarkable, except for the elevation of the plasma glucose (121 mg/dL), triglyceride (310 mg/dL) and low-density lipoprotein (LDL) - cholesterol (156 mg/dL) levels. The patient was negative for serum antinuclear, anti - Sjögren’s-syndrome-related antigen A (SS-A) and anti - Sjögren’s-syndrome-related antigen B (SS-B) antibodies, as well as anti - prote-
inase 3 neutrophil cytoplasmic antibody (PR3 - ANCA), myeloperoxidase neutrophil cytoplasmic antibody (MPO-ANCA) and cyclic citrullinated peptide (CCP) antibodies. The protein and glucose levels and the cell counts in the patient’s cerebrospinal fluid were within the normal limits. We did not perform an electromyogram. A T2-weighted image on 1.5 T MRI showed a high signal region in the pons and the right trigeminal nerve, which was enhanced on the T1-weighted image by the administration gadolinium (Figure A-D), with no other remarkable findings. Spinal MRI showed a cervical and lumbar spinal canal stenosis. A chest X-ray and abdominal computed tomography showed no abnormal findings. On the 16th day after the onset of symptoms, an enzyme immunoassay (EIA) IgG titer for herpes simplex virus (HSV) in a serum sample was elevated to 44.4. A cerebrospinal fluid (CSF) sample showed a titer of 0.29. The patient’s serum titer subsequently reached 81 at 5 weeks after the onset of symptoms. The IgM titer for HSV was negative. The IgG titer for varicella zoster virus (VZV) was 19.4 (positive) in serum, and less than 0.2 (negative) in CSF. The IgM titer was negative for VZV. A polymerase chain reaction (PCR), which was performed to analyze the HSV genome in a serum sample, was negative. The patient was diagnosed with trigeminal neuropathy based on the presence of the herpes infection in his nasal cavity, and was treated with acyclovir for 7 days. His right facial hemidysesthesia improved by the end of the acyclovir therapy. We did not administer steroids. The patient’s dysgeusia disappeared within 4 weeks after the onset of symptoms. MRI was performed on the 42nd day after the onset of symptoms (Figure E-H). Although the abnormal enhancement remained, the patient’s right facial dysesthesia showed an almost complete recovery within 6 weeks after the onset of symptoms. The impairment of his sensation of pain and temperature recovered within 10 weeks.

**Discussion**

We herein present the case of a patient with dissociated sensory trigeminal neuropathy in whom MRI revealed abnormalities of the right ventral side of the pons and the right trigeminal nerve.

The sensory root of the trigeminal nerve passes backwards below the superior petrosal sinus and tentorium cerebelli, and after entering the pons, divides into upper and lower roots (2). The upper root (the main sensory tract) ends partly in a nucleus situated in the pons, while the lower root (the spinal trigeminal tract) descends through the pons and medulla oblongata, and ends in the upper part of the substantia gelatinosa of Rolando. The main sensory tract mediates tactile sensation, while the spinal tract mainly mediates pain and temperature sensation (2). Thus, this patient showed sensory dissociation. The spinal trigeminal nucleus and tract receive primary afferent fibers from the trigeminal, facial, glossofaryngeal, and vagal nerves (3). Thus, our patient not only had facial sensory impairment, but also dysgeusia.

Sensory trigeminal neuropathy is well known as a com-
mon complication of autoimmune connective tissue and inflammatory diseases, such as mixed connective tissue disease, progressive systemic sclerosis and multiple sclerosis (1, 4, 5). However, infection is not considered to be a common mechanism of trigeminal neuropathy (TNO) (1). Connective tissue diseases based on the results of the blood analysis and multiple sclerosis was excluded based on the MRI findings. Consequently, the patient was diagnosed with a herpes infection. Although herpes simplex virus has been implicated in many cases of presumed idiopathic Bell’s palsy, in which patients typically exhibit acute peripheral facial nerve palsy, ipsilateral TNO may be seen in up to 25% of patients with Bell’s palsy (6). There are only two reported cases of herpes infection-related pure trigeminal sensory neuropathy in which an intramedullary lesion was observed on MRI (3, 7, 8) (Table): one had extensive sensory disturbances involving the three divisions of the trigeminal nerve; the other had dysgeusia, and was similar to our case. Only our case had dissociated sensory impairment - in other words, they all had disturbances of the sensations of touch, pain and temperature, but our case is the first report of dissociated trigeminal sensory neuropathy, which was apparently caused by HSV and in which an abnormal pontine lesion was observed on MRI. Interestingly, our patient, who showed dissociated sensory impairment, had enhanced lesions not only in the pons, but also in the trigeminal nerve tract. It is unclear why the patient’s sensory impairment was dissociated; however, we speculate that only the lower root was damaged at the end of trigeminal nerve tract, because the enhancement of the lesion on MRI was not uniform (Figure B, D). We could not determine whether our patient’s herpes infection was caused by HSV type 1 from serum or CSF examinations, however, the effectiveness of acyclovir is consistent with this as a possible cause.

We presented the case of a patient with dissociated trigeminal sensory neuropathy and unique MRI findings concerning the foci responsible for the impairment.

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

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