

and timely surgical intervention are fundamental in averting catastrophic vascular events.

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# Pure motor trigeminal neuropathy in a woman with tegmental pontine infarction



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## ABSTRACT

A 50-year-old woman presented with drowsiness, severe dysarthria, left facial palsy, and left hemiparesis. Diffusion-weighted MRI showed cerebral infarction in the left pontine tegmentum and right cerebellum and angiography revealed basilar artery occlusion. Altered consciousness and hemiparesis fully resolved following successful recanalisation of the basilar artery by emergent intra-arterial thrombolysis. However, about 8 months later, obvious wasting of the left temporal and buccal area without sensory signs was detected. Electrophysiological evaluation revealed a chronic denervation process in the left masseter and temporalis muscles. We suggest that a small infarction in the trigeminal nucleus of the pontine tegmentum can cause an isolated trigeminal motor neuropathy with masticatory muscle atrophy.

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## 1. Introduction

Isolated trigeminal motor neuropathy is an unusual disease that is characterized by trigeminal motor paralysis without trigeminal sensory disturbances.<sup>1,2</sup> Only a small number of cases of secondary trigeminal neuralgia or isolated sensory trigeminal neuropathy due to an ischemic lesion of the pons have been reported.<sup>3–6</sup> Trigeminal motor neuropathy with muscle atrophy due to cerebral infarction is very rare.<sup>7</sup> We describe a patient with unilaterally isolated trigeminal motor paralysis with muscle atrophy, without sensory involvement or other cranial neuropathies.

## 2. Case report

A 50-year-old woman was admitted to our hospital with altered consciousness and left hemiparesis. Examination revealed drowsiness with a left hemiparesis (National Institutes of Health Stroke Scale score = 11).

Diffusion-weighted MRI revealed multiple high signal intensity lesions in the left pontine tegmentum, right cerebellum and right

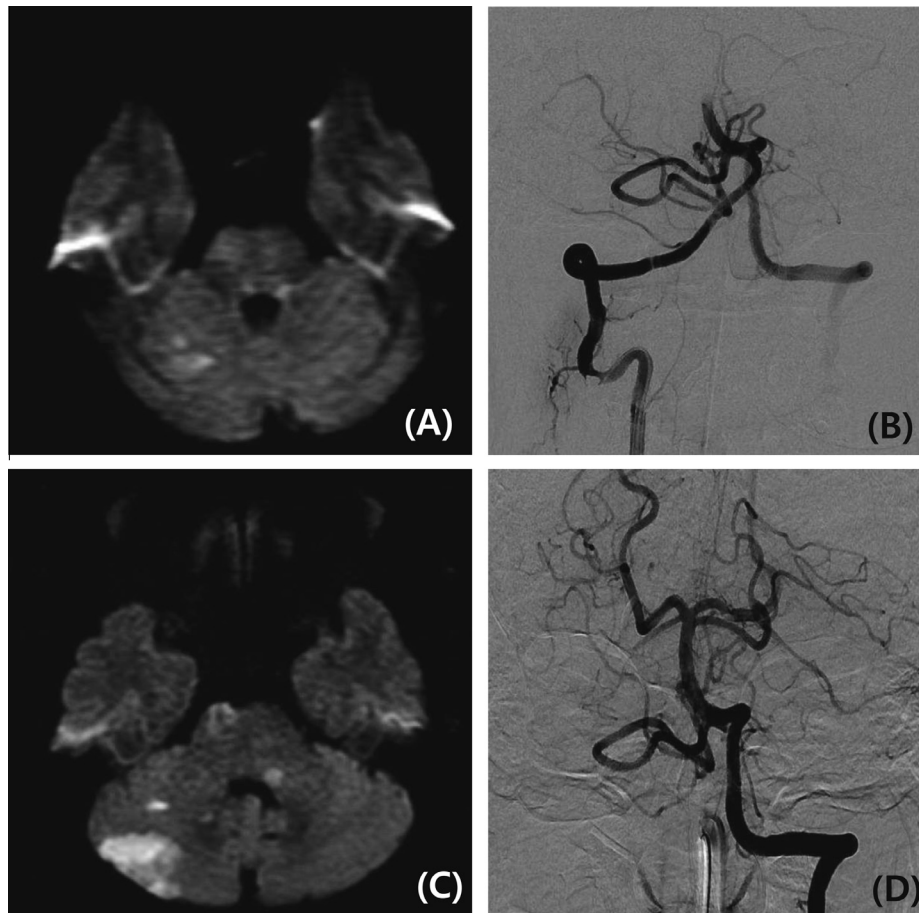
pons. Transfemoral cerebral angiography diagnosed a stump occlusion at the midportion of the basilar artery (Fig. 1A, B). The patient was taken for intra-arterial reperfusion therapy 9 hours after the onset of neurological symptoms. Consequently, the occluded basilar artery was successfully recanalized. A follow-up diffusion weighted MRI confirmed a focal infarction on the left pontine tegmentum (Fig. 1C, D). The day after treatment, the patient achieved a remarkable recovery with full strength in all extremities and intact language and cognition, except for mild dysarthria and chewing difficulties due to weakness of the left masseter muscle. An anticoagulant was administered after atrial fibrillation was diagnosed on electrocardiography.

Within 8 months of this episode, the patient noticed a slow, progressive, painless facial asymmetry without sensory disturbances. Neurological examination revealed jaw deviation on the left during mouth opening. Also, prominent hollowing below and above the left zygoma due to atrophy of the masseter and temporalis muscles (Fig. 2A, arrows) was detected. The patient did not complain of facial pain and numbness or change of taste sense. The corneal reflex was normal. No other abnormal findings, including of the cranial nerves, were detected.

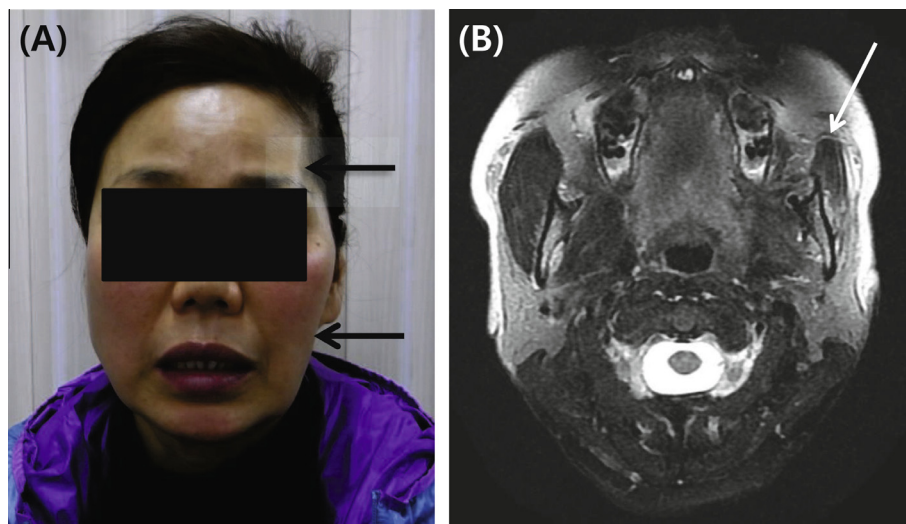
Atrophy in the left temporalis muscle was detected on T2-weighted MRI of the face (Fig. 2B). Needle electromyography of the left masseter and temporalis muscle showed sparse fibrilla-

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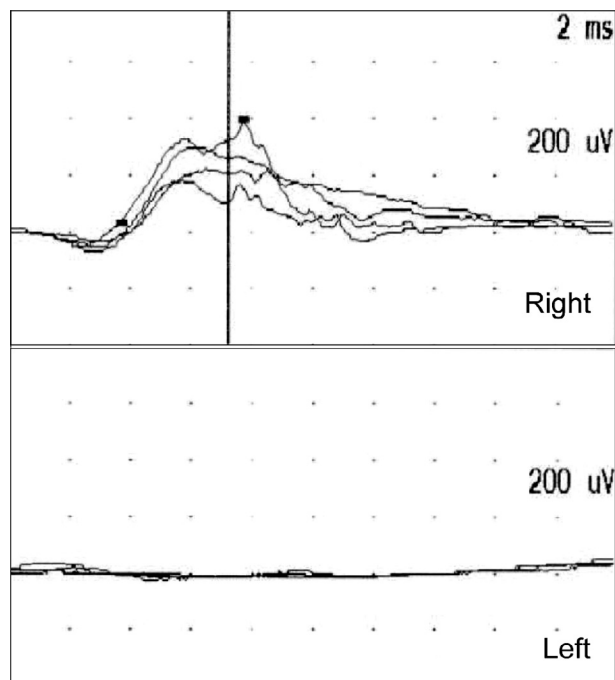
**Fig. 1.** Pretreatment diffusion weighted imaging (DWI) axial MRI showing (A) moderate high signal intensity lesions in the right basis pontis, right cerebellum and left pontine tegmentum, (B) right vertebral artery angiogram, anteroposterior view, showing mid-basilar artery occlusion, (C) DWI MRI showing a residual left pontine tegmentum infarct, two days after the onset of symptoms, and (D) left vertebral artery angiogram, anteroposterior view, showing the mid-basilar artery occlusion, successfully recanalized without residual clot.



**Fig. 2.** (A) Photograph of the face of the patient showing severe muscle wasting on the left side above and below the zygoma and (B) axial T2-weighted facial MRI showing marked atrophy of the left temporalis muscle. (This figure is available in colour at [www.sciencedirect.com](http://www.sciencedirect.com).)

tions and positive sharp waves and scanty motor units with a fast firing rate, while the volitional activity was markedly reduced. The masseter reflex test revealed loss of waveform from the left side in

comparison with the normal configuration of the right side (Fig. 3). Finally, the blink reflex test and facial nerve conduction study were normal.



**Fig. 3.** Masseter reflex electromyography test findings showing normal latencies on the right side and no waveform on the left side.

### 3. Discussion

This patient demonstrates isolated unilateral pure trigeminal motor neuropathy with preserved trigeminal sensory nerve function. We observed both objective evidence of trigeminal motor neuropathy on examination, and electrophysiological studies showing denervation changes, in addition to muscle atrophy on MRI. We suggest that only the trigeminal motor nerve was selectively damaged in the pons based on the findings of normal blink reflex and corneal reflex, without sensory change on the face.

In most cases, trigeminal neuropathy is caused by a peripheral trigeminal nerve lesion. But, rarely, ischemic or hemorrhagic lesions of the brainstem also cause isolated cranial nerve palsies

through the involvement of the intra-axial segments of the respective nerves.<sup>4–8</sup> Thus, we should consider the possibility of a brainstem lesion as the cause of an isolated trigeminal neuropathy. In our patient, the temporal sequence of cerebral infarction followed by progressive wasting of the masseter and temporalis muscles and the fact that the trigeminal neuropathy is consistent with the lesion location support the infarction in left pontine tegmentum as the cause.

To our knowledge, trigeminal motor neuropathy combined with wasting of masseter muscle and caused by ischemic stroke has only been reported twice.<sup>7,8</sup> Lesions in our patient and the two previously reported cases were confined to the pontine tegmentum. Our case is unique because a sensory deficit was not combined with the trigeminal motor neuropathy. The motor nucleus of the trigeminal nerve is situated at the midportion of the pons, medial to the main sensory nucleus of the trigeminal nerve and near the floor of the fourth ventricle. We presume that the lesion in our patient did not involve the tract or the nucleus related to the sensory system, because the lesion was relatively small and located in the dorsomedial side of the sensory trigeminal nucleus.<sup>4</sup> We presume the delayed atrophic changes of the left masseter and temporalis muscles were due to axonal degeneration.

In summary, we suggest that a small infarction in the pontine tegmentum can cause isolated trigeminal motor neuropathy with masticatory muscle atrophy and weakness.

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