

# Alternating Hemiplegia with Ipsilateral Supranuclear Facial Palsy and Abducens Nerve Palsy Caused by Pontine Infarction

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## Abstract

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A 62-year-old right-handed man was diagnosed with a cerebral infarction in the ventromedial region of the left lower pons. He showed left abducens nerve palsy, left-sided supranuclear palsy of the lower part of the face and right hemiparesis. We hypothesized that the mechanism underlying the patient's ipsilateral supranuclear facial palsy involved the corticofacial fibers after they crossed the midline.

**Key words:** pontine infarction, alternating hemiplegia, facial palsy, corticofacial fibers

(Intern Med 55: 2073-2075, 2016)

(DOI: 10.2169/internalmedicine.55.6603)

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## Introduction

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Alternating hemiplegia is caused by a lesion on the opposite side and is accompanied by the additional paralysis of a motor cranial nerve on the same side as the lesion. It is usually caused by lesions of the basal portion of the caudal pons that are extensive enough to involve the corticospinal tract and include the fibers of abducens and facial nerves. Medial pontine syndrome and Millard-Gubler syndrome (MGS) are characterized by ipsilateral sixth and seventh nerve fascicular palsy with contralateral hemiplegia. The conditions are caused by a lesion in the basis pontis, which is usually a stroke that involves the paramedian arteries from the basilar artery (1). In previously reported cases, patients have shown peripheral facial paralysis but not supranuclear facial palsy. We report a case of pontine infarction that caused ipsilateral abducens nerve palsy, ipsilateral supranuclear facial palsy and contralateral hemiparesis.

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## Case Report

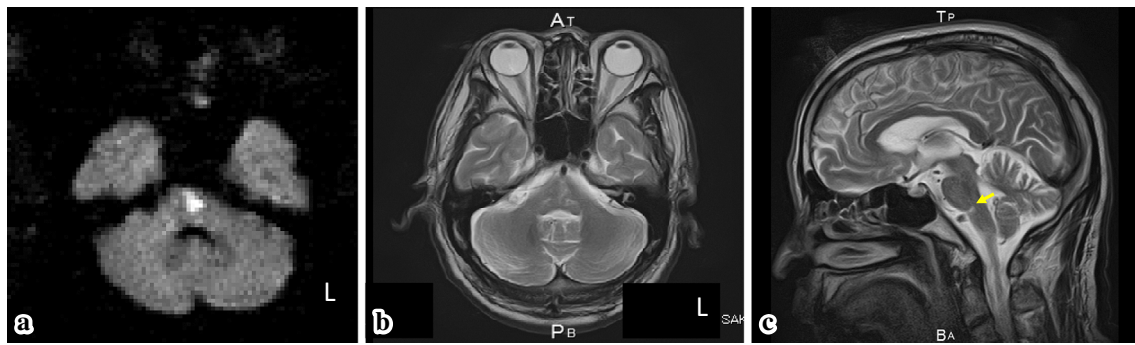
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The patient was a 62-year-old right-handed man with a history of hypertension and diabetes mellitus but no history of neurological problems. After experiencing a sudden onset

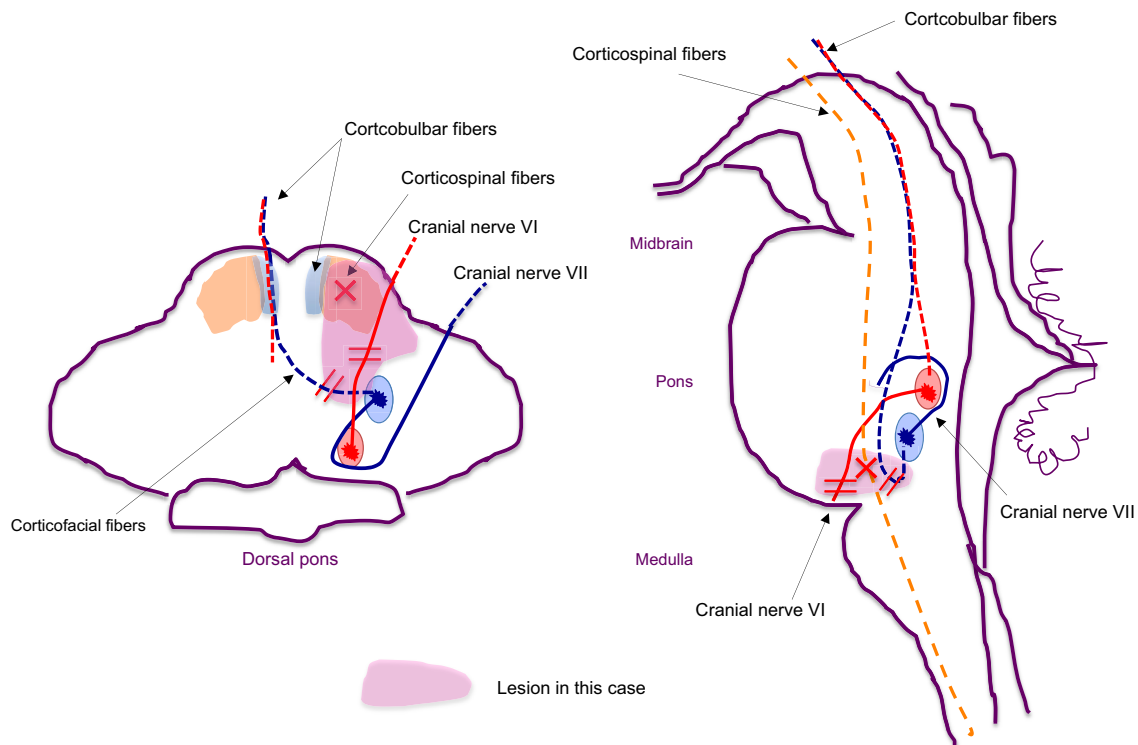
of vertigo and diplopia and right hemiplegia upon waking, he was taken to a local hospital by ambulance and diagnosed with a cerebral infarction. The patient was transferred to our hospital for rehabilitation after undergoing 3 weeks of conservative treatment. Our examination revealed left abducens nerve palsy, left-sided palsy of the lower part of the face and the right upper and lower limbs and right hemiparesis. The pupils were equal, round and reactive to light and accommodation. He did not exhibit ptosis. There was no muscle tenderness. The patient's sensation was normal and intact. His cerebellar coordination was normal on the left side but limited on the right side due to weakness.

A magnetic resonance imaging (MRI) scan of the brain 3 days after the onset of symptoms revealed areas of high intensity in the ventromedial region of the left lower pons on diffusion-weighted imaging (Fig. 1a). An MRI T2-weighted image 2 months from the onset of symptoms revealed an abnormal area of high intensity involving the left ventromedial region of the left lower pons (Fig. 1b); a sagittal image showed an abnormal area of high intensity in the base of caudal pons (Fig. 1c). MR angiography of the head and neck revealed no stenosis or obstruction in the carotid artery or vertebral artery system.

He could walk without any assistance and he was discharged from hospital after 2 months; however, his facial



**Figure 1.** A magnetic resonance imaging (MRI) scan of the brain. An abnormal area of high intensity was detected in the ventromedial region of the left lower pons on diffusion-weighted imaging 3 days after the onset of symptoms (a). An axial brain MRI T2-weighted image 2 months after the onset of symptoms revealed an abnormal area of high intensity involving the left ventromedial region of the left lower pons (b). A sagittal image shows an abnormal area of high intensity in the base of the caudal pons (c).



**Figure 2.** The anatomic localization of infarctions. Ipsilateral supranuclear facial paresis was caused by damage to the corticofacial fibers from decussation to the facial nerve nucleus at the medioinferior pons.

and limb paresis persisted. Although the disturbance of his ocular movement gradually improved, he still had diplopia.

## Discussion

MGS is characterized by ipsilateral facial palsy involving the root fibers and contralateral hemiparesis which results from the involvement of the corticospinal tract (2). Most MGS patients present other associated neurological abnormalities because numerous nuclei or fibers exist next to the root fibers of the facial nerve.

Although ipsilateral peripheral facial palsy originating from nuclear fascicular involvement is occasionally observed in patients with pontine ischemia and mainly presents as a supplementary sign to contralateral hemiparesis in MGS, our patient showed ipsilateral supranuclear facial palsy (3).

The facial nucleus has dorsal and ventral divisions which contain the lower motor neurons which supply the muscles of the upper and lower face, respectively. Although the ventral division receives only contralateral input, the dorsal division receives bilateral upper motor neuron input. Thus, lesions of the corticobulbar tract between the cerebral cortex,

the pons and the facial nucleus impair or reduce the input to the ventral division; however, the ipsilateral input to the dorsal division is retained. As a result, supranuclear facial palsy is characterized by hemiparalysis of the facial expression muscles on the contralateral side but not the forehead.

Based on a transcranial magnetic stimulation study, Urban et al. (4) suggested that facial paresis due to brainstem lesions may present with findings similar to those of contralateral supranuclear facial paresis caused by lesions in the cerebral peduncle, pontine base, the aberrant bundle and the ventral medulla. Furthermore, they hypothesized that ipsilateral supranuclear facial paresis may result from a lesion in the lateral medulla, and that paresis of the peripheral facial nerve in the dorsolateral medulla may occur when a lesion involves the lower pons (4). In our patient with ipsilateral supranuclear facial palsy, we hypothesized that the supranuclear fibers formed a loop in the medulla before reaching the facial nucleus and that the fibers were affected after

crossing the midline, thus causing ipsilateral paresis (Fig. 2). Furthermore, the lower pontine lesion and the intra-axial infranuclear nerve fibers may be involved or the corresponding facial subnucleus may be affected.

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

## References

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