Two patients with abducens nerve palsy and crossed hemiplegia (Raymond syndrome)

KATSUHIKO OGAWA, YUTAKA SUZUKI, and SATOSHI KAMEI Department of Neurology, Nihon University School of Medicine, Nerimahikarigaoka Hospital, Tokyo, Japan

Abstract

We report 2 patients of alternating hemiplegia with abducens nerve palsy. One patient was also complicated by contralateral supranuclear facial nerve palsy. MRI showed an infarct involving the paramedian and lateral area of the pontine basis in both patients. We considered that corticospinal tract and infraabducens nerve fibers were involved in both patients, and that the corticobulbar tract was involved in 1 patient with supranuclear facial nerve palsy. Although supranuclear facial nerve palsy was not found in the other patient, we speculated that supranucler facial nerve fibers might pass mainly through the aberrant pyramidal tract in this patient.

Key words: Raymond syndrome; supranuclear facial nerve palsy; aberrant pyramidal tract; MRI.

Introduction

Raymond syndrome is one of the crossed paralyses and consists of lateral abducens nerve palsy and contralateral hemiparesis (Silverman *et al.*, 1995, Marx *et al.*, 2009). However, there have been few reports of MRI findings of Raymond syndrome (Satake *et al.*, 1995, Ogawa *et al.*, 2008). We reported a patient with Raymond syndrome demonstrating supranuclear facial nerve palsy, due to an infarct of the pontine basis (Ogawa *et al.*, 2008). We then encountered another patient with Raymond syndrome due to pontine infarction. We studied the neuroradiological correlation of the previously reported patient (Ogawa *et al.*, 2008) and the newly encountered patient.

Case report

Patient 1 (Ogawa *et al.*, 2008). A 73-year-old man complained of hemiparesis on the left side and double vision, and was admitted to our hospital. On admission, oculomotor examinations showed that

abduction of the right eye was severely impaired, whereas adduction of the left eye was intact, when he gazed to the right side. He did not show vertical gaze palsy or horizontal gaze palsy to the left in both eyes. He also showed left supranuclear facial nerve palsy, dysarthria, and left hemiparesis. Head MRI showed an infarct located in the paramedian and lateral area at the base of the caudal pons on the right side (Fig. 1A, B). MRA showed mild stenosis of the basilar artery.

Patient 2. A 61-year-old man complained of right hemiparesis and double vision, and was admitted to our hospital. On admission, oculomotor examinations showed that abduction of the left eye was severely impaired, whereas adduction of the right eye was intact, when he gazed to the left side. He did not show vertical gaze palsy or horizontal gaze palsy to the right in both eyes. He also showed mild hemiparesis on the right side. Head MRI showed an infarct that was located in the paramedian and lateral area in the base of the caudal pons on the left side (Fig. 2). MRA findings were normal.



FIG. 1. – Axial T2-weighted head MRI showed an abnormal high intensity area in the paramedian and lateral area in the base of the caudal pons on the right side (arrow) (A). Sagittal T1-weighted head MRI showed an abnormal low intensity area in the base of the caudal pons (arrow) (B).



Fig. 2. – Head MRI (T2-weighted image) showed an abnormal high intensity area in the paramedian and lateral area in the base of the caudal pons on the left side (arrows).

Discussion

The lesions of these 2 patients were located in the paramedian and lateral area in the base of the caudal pons. Corticospinal tract and corticobulbar tract run through the ventromedial area at the caudal pons. The corticobulbar tract decussates at the caudal pons and projects to the contralateral facial nerve nucleus. The abducens nerve nucleus is located in the dorsomedial area of the caudal pons. Infraabducens nerve fibers leave the abducens nerve nucleus and run in the ventral direction, and then pass through the lateral area at the caudal pontine base. Hemiparesis and abducens nerve palsy in these 2 patients were considered due to ischemia of the corticospinal tract and infraabducens nerve fibers in the paramedian and lateral area of the caudal pontine base. Supranuclear facial nerve palsy was found in Patient 1. We considered that the corticobulbar tract was also involved in Patient 1.

Aberrant pyramidal tract (APT) is reported to be a collateral pathway of the pyramidal tract, and supplies the motor nuclei of the cranial nerves (Yamashita *et al.*, 2001). Yamashita *et al.* reported that APT leaves the pyramidal tract within the crus cerebri and passes in the medial lemniscus of the pons through the upper medulla oblongata (Yamashita *et al.*, 2001). Supranuclear facial nerve palsy was absent in Patient 2, although the ventromedial area of the caudal pons was involved. We speculated that ABT was the main pathway of the supranuclear facial nerve fibers in Patient 2.

We reported here 2 patients with Raymond syndrome, and considered that investigating the

MRI findings for Raymond syndrome is important to determine the pathogenesis of supranuclear facial nerve palsy caused by caudal pontine lesion.

REFERENCES

- Silverman IE, Liu GT, Volpe NJ, Galetta SL. The crossed paralyses: the original brain-stem syndromes of Millard-gubler, Goville, Weber, and Raymond-Cestan. Arch. Neurol. 1995;52: 635-8.
- Marx JJ, Thömke F. Classical crossed brain stem syndromes: myth or reality? J. Neurol. 2009;256:898-903.
- Satake M, Kira J, Yamada T, Kobayashi T. Raymond syndrome (alternating hemiplegia) caused by a small haematoma at the medial pontomedullary junction. J. Neurol. Neurosurg. Psychiatry, 1995;57:261.
- Ogawa K, Tougou M, Oishi M, Kamei S, Mizutani T. A case of pontine infarction causing alternating hemiplegia with ipsilateral abducens nerve palsy and contralateral supranuclear facial nerve palsy. Clin. Neurol. 2008;48:135-8.
- Yamashita M, Yamamoto T. Aberrant pyramidal tract in the medial lemniscus of the human brainstem: normal distribution and pathological changes. Eur. Neurol. 2001;45:75-82.

Katsuhiko Ogawa, M.D. Division of Neurology, Department of Medicine, Nihon University School of Medicine, 30 -1 Oyaguchi-kamimachi, Itabashi-ku, Tokyo 173-8610, Japan E-mail: ogawak@med.nihon-u.ac.jp