

Bilateral Medial Medullary Infarction With Oculomotor Disorders

Kazunori Toyoda, MD; Yasuhiro Hasegawa, MD; Toshiro Yonehara, MD;
Jiro Oita, MD; and Takenori Yamaguchi, MD

Background and Purpose: We describe the first case of bilateral medial medullary infarction demonstrated by magnetic resonance imaging. We discuss the relation between this lesion and the oculomotor signs that were observed clinically.

Case Description: A 71-year-old man initially presented with pure motor hemiparesis, which progressed to complete quadriplegia. He also developed nearly complete vertical and horizontal ophthalmoplegia. Magnetic resonance imaging revealed upper medial medullary infarctions bilaterally that extended to the pontomedullary junction.

Conclusions: We propose that the vertical oculomotor disorders resulted from involvement of the oculomotor system in the caudal brain stem, especially the caudal paramedian pontine reticular formations on both sides. (*Stroke* 1992;23:1657–1659)

KEY WORDS • cerebral infarction • cerebrovascular disorders • magnetic resonance imaging • medulla oblongata • ocular disease

Infarction of the medial medulla oblongata has been rarely reported, even during the era of magnetic resonance imaging (MRI), a technology that can detect small lesions in the posterior fossa.¹ Because of the rarity of this syndrome, clinical features such as disturbance of oculomotor control are still controversial. We report the first case, to our knowledge, in which bilateral medial medullary infarction was clearly demonstrated by MRI and discuss the relation between this anatomic lesion and the oculomotor signs appearing in this patient.

Case Report

A 71-year-old man with a history of hypertension and smoking awoke with a right hemiparesis. He was admitted to our hospital the day after symptoms began. On admission, his pulse rate was 80 beats per minute, blood pressure was 202/110 mm Hg, and respirations were 18 per minute and regular. He was alert, with normal higher cortical function. His speech was dysarthric. The visual fields were intact; pupils were equal, with a normal light reflex; and ocular movements were full and smooth, without nystagmus. A mild right hemiparesis was present involving the face and tongue. The right-sided deep tendon reflexes were slightly increased. There were no sensory deficits to touch, pain, temper-

ature, vibration, or position sense. There was no ataxia on either side. Laboratory studies showed no abnormal findings. No abnormality was demonstrated on brain computed tomography (CT) repeated in the acute stage.

A diagnosis of pure motor hemiparesis due to lacunar infarction was initially made. From day 5, his neurological status began to deteriorate gradually. In addition to progression of the right hemiparesis, weakness on the left side was noted. Ultimately, he progressed to a complete quadriplegia on day 17. Cheyne-Stokes respirations that began on day 10 were followed by apnea 1 week later, and he needed mechanical ventilation thereafter.

Abnormalities of eye movement began with horizontal nystagmus of lateral gaze on day 9. Horizontal saccadic and smooth pursuit were disturbed bilaterally on day 14 and vertically on day 16. On day 19 he could neither direct his gaze voluntarily nor follow a target in any direction except for slight downward gaze. Oculocephalic maneuvers elicited both horizontal and vertical eye movements. Caloric nystagmus was normally provoked, and Bell's phenomenon was preserved. Jerk nystagmus with the quick phase to upward, leftward, and rightward was observed when trying to gaze to each direction.

A brain MRI with a 1.5-T superconducting magnet performed on day 10 revealed a low-intensity area on T1-weighted spin-echo imaging, which was enhanced with gadolinium-DTPA in the upper medulla oblongata extending to the pontomedullary junction. The lesion involved the medial medulla oblongata bilaterally, mainly the ventral half with partial extension dorsally. Small lesions without gadolinium-DTPA enhancement, suggesting old lacunar infarcts, were present in the subcortices of both cerebral hemispheres. The junction

From the Cerebrovascular Division, Department of Medicine, National Cardiovascular Center, Suita, Osaka, Japan.

Supported in part by Research Grant for Cardiovascular Diseases 2A-2 from the Ministry of Health and Welfare, Tokyo, Japan.

Address for correspondence: Kazunori Toyoda, MD, Department of Cerebrovascular Disease, Institute of Neuroscience, St. Mary's Hospital, 422, Tsubuku-Honmachi, Kurume, Fukuoka 830, Japan.

Received February 17, 1992; final revision received June 11, 1992; accepted July 28, 1992.

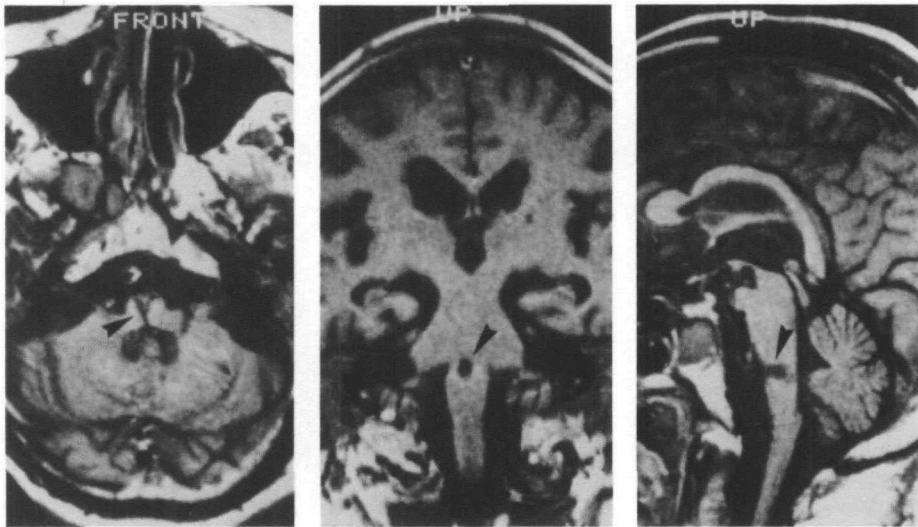


FIGURE 1. T1-weighted magnetic resonance imaging (spin-echo; repetition time, 600 msec; echo time, 20 msec) without gadolinium-DTPA enhancement on day 109, showing axial (left panel), coronal (middle panel), and sagittal (right panel) sections. Infarct is clearly demonstrated in medial medulla oblongata bilaterally (arrows).

of the vertebral arteries was clearly seen at the level of the lower pons. Follow-up MRI studies (days 68 and 109) showed essentially the same findings except for disappearance of gadolinium-DTPA enhancement (Figure 1).

Cerebral angiography on day 18 delineated a wall irregularity of the left vertebral and basilar arteries, with a small aneurysm at the middle of the basilar artery. The right vertebral artery was hypoplastic and severely sclerotic. The anterior spinal artery could not be identified (Figure 2). The right internal carotid artery was occluded, but fairly good collateral flow through the right ophthalmic artery and the anterior communicating artery was noted and confirmed by single-photon emission computed tomography (SPECT).

The disturbance of ocular movement gradually improved, and only a mild horizontal gaze limitation was detected on day 60, but improvement of the quadriplegia was not observed.

Discussion

Although there have been a few reports of unilateral medial medullary infarction verified by MRI,¹ we could not find prior studies describing bilateral infarction. Such a lesion is presumed to occur mainly because of an

occlusion of the anterior spinal artery, which we could not identify angiographically in this patient. The infarcted area demonstrated by MRI included the pyramidal tracts, inferior olivary nuclei, medial lemnisci, and medial longitudinal fasciculi (MLF) bilaterally. Involvement of the rostral brain stem was not detected on MRI, and supratentorial significant lesions were not demonstrated by MRI or SPECT.

The clinical features of medial medullary infarct are known to be contralateral hemiplegia and posterior column sensory deficits in addition to ipsilateral tongue weakness. Several patients, however, have been observed to have pure motor hemiplegia with slight facial palsy.² The lesion in our patient was isolated pyramidal involvement at a level higher than that of the hypoglossal nucleus. Initially, the pure motor hemiparesis of this patient was difficult to distinguish from that seen with lacunar infarct of the internal capsule or the pontine base.

The deterioration of his clinical status was assessed to be an extension of the ischemic lesion. Respiratory failure was produced by bilateral involvement of the medullary reticular formation.³⁻⁵ Oculomotor disorders are infrequent in medial medullary lesions and are mainly limited to upbeat nystagmus.⁶ Gaze palsy is

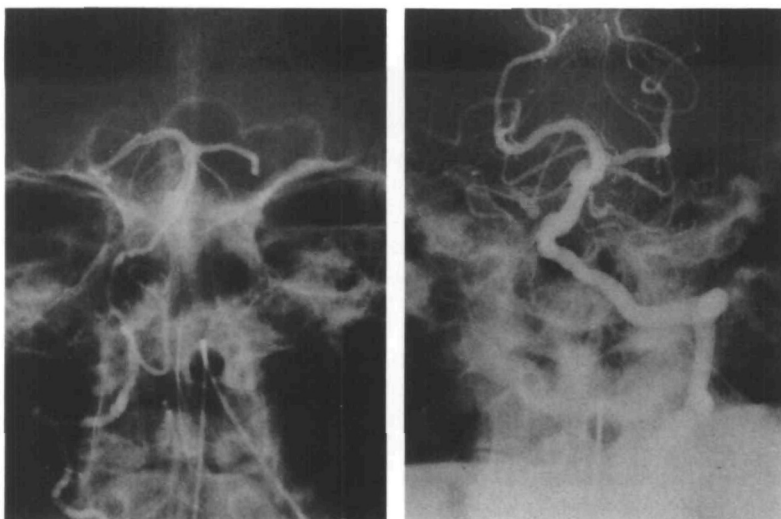


FIGURE 2. Conventional angiogram of right vertebral artery (left panel) and the left vertebral artery (right panel) on day 5. Right vertebral artery is hypoplastic and severely sclerotic. Anterior spinal artery is not identified.

usually caused by a lesion in the pons or midbrain, and vertical oculomotor abnormality is a classic feature of midbrain lesions, related to the rostral interstitial nucleus of the MLF.⁷ Three clinicopathologic reports on medial medullary infarction, however, described the presence of oculomotor disturbance^{3–5}: bobbing eye,³ upward and lateral gaze palsy,⁵ and poor maintenance of depression of gaze.⁴ All of the pathologically verified lesions were limited to the bilateral medial medulla, extending to the pontomedullary junction in one case⁵; the mechanism, however, was not detailed.

Although MRI did not show the pontine lesion in our patient, brain edema or incomplete ischemia might have extended to the lower pons continuously from the medulla. The high position of the vertebral artery junction suggested the presence of a blood supply from vertebral branches and anterior spinal arteries up to the lower pons. Upward extension of the lesion can be responsible for horizontal gaze palsy, but some previous reports indicated that there was also vertical gaze palsy.^{6,8} An animal experimental study⁹ suggested the relation of vertical saccade palsy to the lower pontine lesion. Bilateral damage of the caudal paramedian pontine reticular formations (PPRFs) ventral to the abducens nuclei leads to a severe disruption of vertical rapid eye movements in it. The mechanism was supposed to result from involvement of inputs from the caudal PPRF to the rostral interstitial nucleus of the MLF through the direct ascending anatomic pathway between them.^{6,10} Recovery of oculomotor palsy suggested transient damage of PPRFs due to edema or incomplete ischemia. Preservation of oculocephalic and caloric reactions indicated that both the oculomotor and abducens nuclei and fibers as well as vestibular systems

were spared, and also suggested an isolated insult of the PPRF.

We have attempted, in this provocative case of bilateral medial medullary infarction, to clarify the function of oculomotor system in the caudal brain stem.

Acknowledgments

The authors are grateful to Dr. Marc Fisher (Professor of Neurology and Radiology, University of Massachusetts Medical School, Worcester, Mass.) for editing the manuscript and providing valuable comments.

References

1. Fox AJ, Bogousslavsky J, Carey LS, Barnett HJM, Vinitski S, Karlik SJ, Vinuela F, Pelz DM, Hachinski V: Magnetic resonance imaging of small medullary infarctions. *AJNR* 1986;7:229–233
2. Ho KL, Meyer KR: The medial medullary syndrome. *Arch Neurol* 1981;38:385–387
3. Kase CS, Varakis JN, Stafford JR, Mohr JP: Medial medullary infarction from fibrocartilaginous embolism to the anterior spinal artery. *Stroke* 1983;14:413–418
4. O'Brien FB, Bender MB: Localizing value of vertical nystagmus. *Arch Neurol* 1945;54:378–380
5. Meyer JS, Herndon RM: Bilateral infarction of the pyramidal tracts in man. *Neurology* 1962;12:637–642
6. Bogousslavsky J, Meienberg O: Eye-movement disorders in brainstem and cerebellar stroke. *Arch Neurol* 1987;44:141–148
7. Buttner-Ennever A, Buttner U, Cohen B, Baumgartner G: Vertical gaze paralysis and the rostral interstitial nucleus of the medial longitudinal fasciculus. *Brain* 1982;105:125–149
8. Bogousslavsky J, Regli F: Convergence and divergence synkinesis: A recovery pattern in benign pontine hematoma. *Neuroophthalmology* 1984;4:219–225
9. Henn V, Lang W, Hepp K, Reisine H: Experimental gaze palsies in monkeys and their relation to human pathology. *Brain* 1984;107:619–636
10. Larmende P, Henin D, Jan M, Elie A, Gouaze A: Abnormal vertical eye movements in the locked-in syndrome. *Ann Neurol* 1982;11:100–102