

Case report

Bilateral medial medullary infarction due to bilateral vertebral artery dissection

Masafumi Fukuda*, Toyotaka Aiba, Sho Takahashi

Department of Neurosurgery, Shibata Prefecture Hospital, Shibata City, Niigata, Japan

Received 27 January 2003; received in revised form 4 November 2003; accepted 20 November 2003

Abstract

We describe a 52-year-old woman who experienced transient motor weakness and numbness of the left extremities and presented 2 days later with severe hemiparesis and sensory impairment of the right extremities and right lingual palsy. Magnetic resonance imaging (MRI) revealed bilateral upper medial medullary infarction, primarily in the left ventral portion. The findings of both three-dimensional (3D) computed tomographic and conventional angiography suggested dissection of both intracranial vertebral arteries (VAs). Medial medullary infarction is generally caused by atherosclerosis within a VA or anterior spinal artery. This is the first report of bilateral medial medullary infarction due to dissection of both intracranial VAs.

© 2003 Elsevier B.V. All rights reserved.

Keywords: Bilateral; Medial medullary syndrome; Infarction; Vertebral artery dissection; 3D-CT angiography; MRI

1. Introduction

Medial medullary infarction, which occurs rarely compared to lateral medullary infarction, is characterized by contralateral hemiparesis sparing the face, hemisensory loss of the posterior column, and ipsilateral weakness of the tongue. In most patients with medial medullary infarction, atherosclerosis of the vertebral artery (VA) is the predominant underlying vascular pathology [1–4]. We describe what may be the first case in the literature of bilateral VA dissection resulting in bilateral medial medullary infarction.

2. Case report

A 52-year-old woman with a history of hypertension experienced sudden onset of motor weakness and numbness in her left extremities and was transported to our hospital by ambulance on February 2002. Upon admission, her blood pressure was 179 mmHg/110 mmHg and her pulse was 82

beats per minute. Electrocardiography showed no abnormalities. The left-sided hemiparesis and sensory impairment had disappeared completely and no other neurological deficits were seen. The patient appeared to have suffered a transient ischemic attack due to inadequate blood supply in the right cerebral hemisphere and was kept under observation. On the 2nd day of hospitalization, she complained of headache in the left frontal area and vomited. On the 3rd day, she showed motor weakness and numbness of the right extremities and face. Her visual fields were normal and her pupils were equal and reactive to light, and ocular movements were normal with no nystagmus. Her tongue deviated to the right. She had severe hemiparesis affecting her right extremities. Impaired proprioception, and tactile and pain sensation of her right extremities were noted. Impairment of pain sensation was also noted in her right face with intact sensation to touch. Brain computed tomography (CT) showed no abnormalities, but T2-weighted magnetic resonance imaging (MRI) of the brain showed high intensity areas in the upper medulla oblongata extending to the pontomedullary junction (Fig. 1A). The lesion involved the bilateral medial medulla oblongata, primarily in the left ventral portion (Fig. 1B). Three-dimensional CT angiography (three-dimensional (3D) CTA) showed tapered occlusion of the left VA and severe stenosis of the right VA (Fig. 2A). A diagnosis of bilateral medial medullary infarction due to bilateral VA dissection

* Corresponding author. Present address: Department of Neurosurgery, Brain Research Institute, Niigata University, 1-757 Asahimachi-dori, Niigata City 951-8585, Japan. Tel.: +81-25-227-0653; fax: +81-25-227-0819.

E-mail address: mfuku529@bri.niigata-u.ac.jp (M. Fukuda).

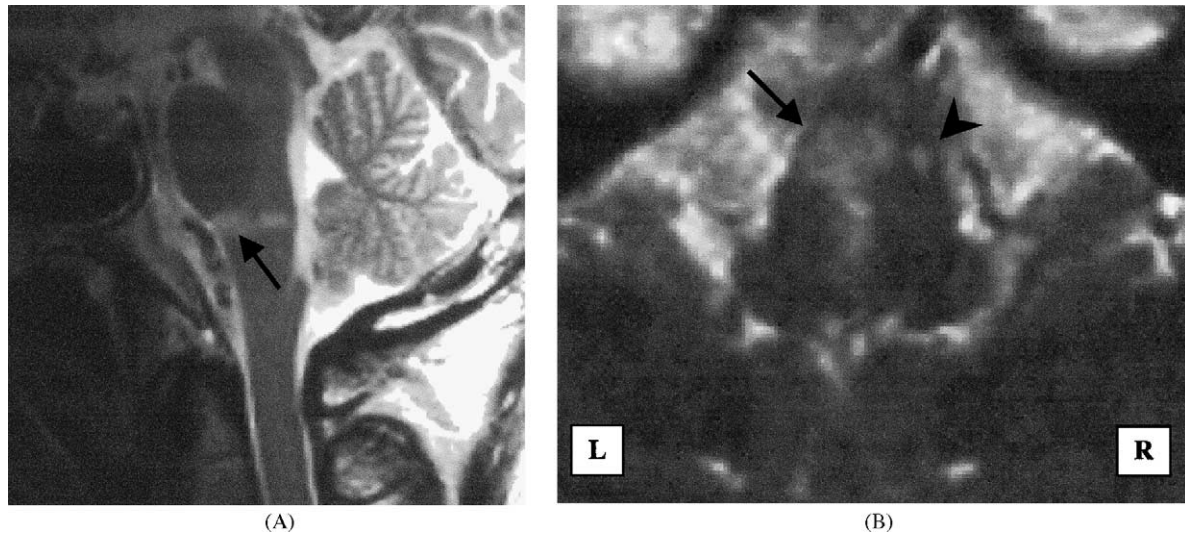


Fig. 1. Magnetic resonance images of the medulla oblongata (A) T2-weighted sagittal image shows high signal intensity at the pons-medullary junction (black arrow), (B) T2-weighted horizontal image shows high signal intensity at the left medial and ventral medulla (black arrow) and the right ventral medulla (black arrowhead).

was made from these findings. Although the hemiparesis ameliorated gradually with conservative therapy, a second 3D CTA on the 14th day showed occlusion of both VAs (Fig. 2B). On the 26th day after onset, we performed conventional cerebral angiography to determine the hemodynamics of the vertebrobasilar arterial system. The left VA was tapered and occluded immediately distal to the origin of the left posterior inferior cerebellar artery (Fig. 3A). Right ver-

tebral angiography revealed the pearl and string sign from the intracranial VA segment to the union (Fig. 3B). The basilar artery and main branches were visible via the right posterior communicating artery from the anterior circulation (Fig. 3C). The third 3D CTA approximately 2 months after onset showed that both VAs were re-canalized despite their wall irregularity still remained partly (Fig. 2C). The patient does not require daily living assistance, although the degrees

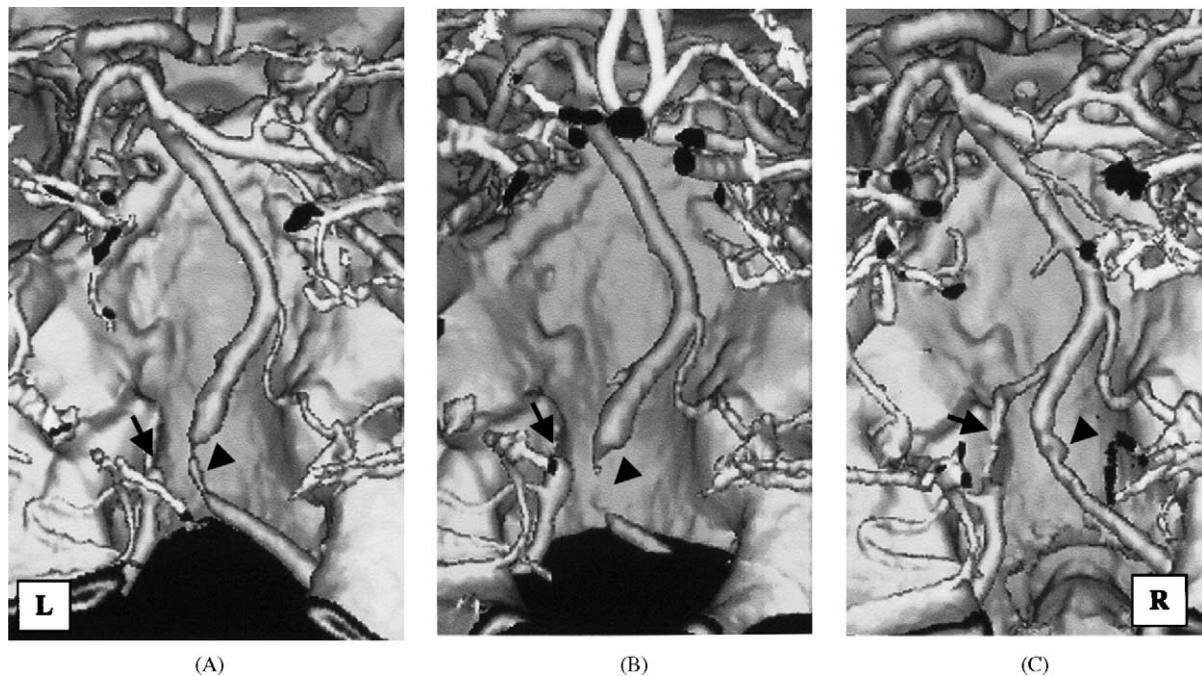


Fig. 2. Three-dimensional computed tomographic angiograms (A) the first angiogram shows tapered occlusion of the left vertebral artery (VA) (black arrow) and severe stenosis of the right VA (black arrowhead), (B) the second angiogram (day 14) shows occlusion of both VAs (left: black arrow; right: black arrowhead), (C) the third angiogram (2 months after onset) shows both VAs are re-canalized but some irregular narrowing is present (left: black arrow; right: black arrowhead).

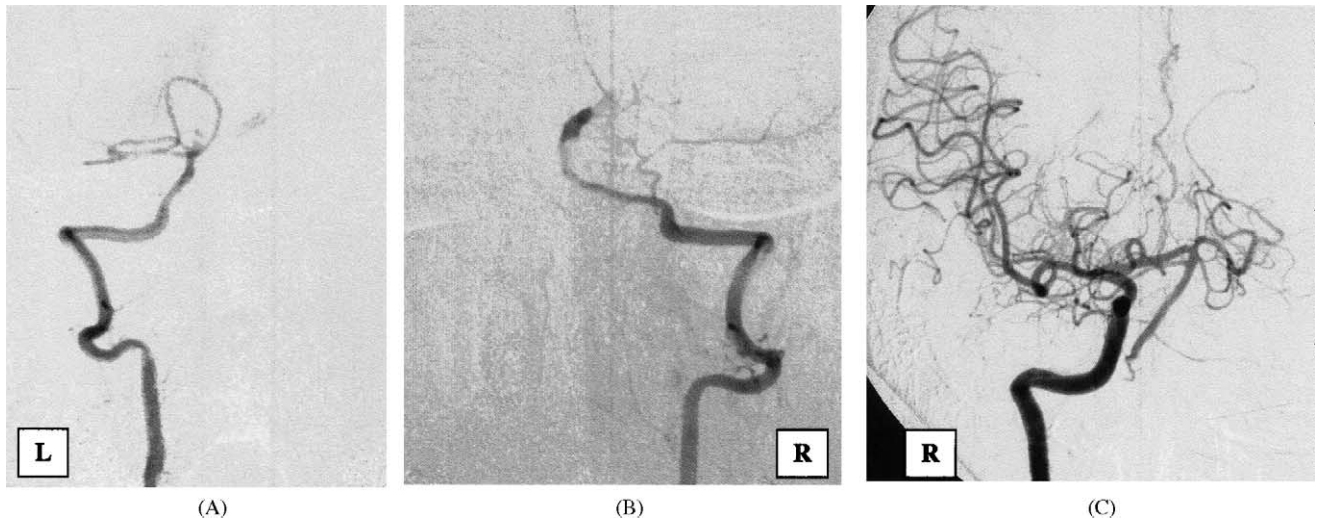


Fig. 3. Conventional angiogram (A) left vertebral angiogram shows tapered occlusion of the left VA at the part immediately distal to the origin of the left posterior inferior cerebellar artery, (B) right vertebral angiogram shows the pearl and string sign from the intracranial segment to the union, (C) right carotid angiogram reveals the basilar artery and main branches via the right posterior communicating artery.

of her right hemiparesis and sensory impairment have not been changed 18 months after the onset.

3. Discussion

Medial medullary infarction has been a relatively rare occurrence and only autopsy descriptions of the disease have been reported; however, MRI now allows us to diagnose living patients. A recent study, showed that medial medullary infarction detected by MRI accounts for approximately 0.5% of all acute brain infarctions [4]. Traditionally, medial medullary infarction is associated with the clinical triad of ipsilateral lingual palsy, contralateral hemiparesis, and lemniscal sensory loss (Déjérine's syndrome). However, some reported medial medullary infarctions identified by MRI were more heterogeneous in each case depending upon differences in the extent of the medullary ischemic lesion.

Hemiparesis due to involvement of the pyramidal tract is the most common clinical manifestation of medial medullary infarction. Although hemiparesis is usually contralateral to the lesions, as in the present case, some authors have described patients with ipsilateral hemiparesis in which infarction extended to the medullocervical junction disrupting the pyramidal tract below its decussation [1,2]. Hemisensory disturbance of the posterior column type is also common in cases of medial medullary infarction. However, in several patients previously described, the sensation of pain in addition to sensations of vibration and position were disturbed even when MRI showed that the lesion did not extend into the lateral spinothalamic pathway [1,2]. Our patient also presented with impaired pain sensation in the right hemibody. MRI depicted the infarction within the left medial medullary region, although the obtained axial slice of the medulla oblongata may not have revealed the whole extension of the

lesion. Impairment of pain sensation in medial medullary infarction can be explained by involvement of the fibers in the dorsal columns and medial lemniscus as well as the adjacent medial medullary reticular formation carrying nociceptive information [5].

Ipsilateral lingual palsy is a classic symptom of medial medullary infarction. Nonetheless, in the Kim et al. series of 18 patients, it was seen in only two [2]. Infrequent ipsilateral lingual palsy in medial medullary syndrome may be attributed to the anatomical characteristics of the hypoglossal nucleus extending longitudinally within the medulla oblongata. A lesion in a small portion of it may not cause detectable symptoms. Besides, several patients previously described had tongue weakness contralateral to the infarct [4]. Because the hypoglossal nucleus does not extend to the upper medulla adjacent to the pontomedullary junction, the upper medullary infarction as well as in our patient is likely to give rise to contralateral lingual palsy due to involvement of the corticobulbar tracts. These findings suggest that we may diagnose a medial medullary infarction even in patients presenting only motor weakness and sensory disturbance in right or left extremities without ipsilateral lingual palsy, especially when MR imaging does not depict hemispheric lesions.

The most common etiology of medial medullary infarction is atherothrombosis of the VA and anterior spinal artery [1–4]. Dissection of the VA has not been named as a common cause of medial medullary infarction [6], however, there are several reports of just such a cause (Table 1). In the previous reports, VA dissections were extracranial [1,7], or they were caused by head injury [4]. To our knowledge, ours is the first report of medial medullary infarction caused by spontaneous intracranial VA dissection. Although we could not show the intramural lumen in the dissected artery by MRI, conventional angiography depicted the tapered occlusion of

Table 1
Medial medullary infarction due to vertebral artery dissection

Case no.	Reference	Age/sex	Risk factor	Site of infarct	Site of dissection	Symptoms
1	[4]	38/M	Head injury	Rt upper	Blt VA and BA	Somnolence, Lt hemiparesis
2	[1]	42/F	HT	Lt	Lt extracranial VA	Rt hemiparesis, sensory impairment, Lt XI–XII palsy, Nystagmus
3		48/F	–	Lt	Lt extracranial VA	Rt hemiparesis, sensory impairment, Lt XII palsy
4		30/M	HT	Lt	Lt extracranial VA	Somnolence, Lt hemiparesis, sensory impairment, Lt XII palsy, Nystagmus
5	[7]	61/F	–	Rt	Rt extracranial VA	Lt hemiparesis, sensory impairment, Rt XII palsy, Nystagmus, Dysarthria
6	Presented case	52/F	HT	Blt upper (Lt > Rt)	Blt VA (V4)	Lt transient hemiparesis, Rt hemiparesis, sensory impairment

Abbreviations: HT, hypertension; Blt, bilateral; Lt, left; Rt, right; VA, vertebral artery; BA, basilar artery.

the left VA and the pearl and string sign in the right VA. The 3D CTA findings also suggested that our patient's medial medullary infarction was caused by bilateral VA dissection. Recent study has shown the irregular luminal surface to be a characteristic of vertebrobasilar dissection and that follow-up 3D CTA allows for identification of changes in lesion morphology over time [8]. Three-dimensional CTA may be useful in the diagnosis and management of vertebrobasilar dissection.

It is intriguing that left and right hemiparesis occurred alternately in our patient. At first, she experienced transient left hemiparesis, but 2 days later she presented with motor weakness and numbness in the right extremities and face, which were followed by frontalgia and vomit. The first 3D CTA showed the tapered occlusion in the left VA and tapered but incomplete occlusion in the right VA. MRI revealed a very small infarction in the right upper medulla compared to the lesion in the left medulla. We speculate the following: the right VA dissection occurred initially, but because the patient had only a small ischemic lesion, the left hemiparesis improved quickly. The left VA was dissected and completely occluded, leading to severe hemiparesis and sensory impairment in the right extremities caused by left medial medullary infarction. Despite absence of or insufficient blood supply from both VAs, the intact basilar artery territory can prob-

ably be attributed to retrograde flow via the posterior communicating artery from the anterior circulation.

References

- [1] Bassetti C, Bogousslavsky J, Mattle H, Bernasconi A. Medial medullary stroke: report of seven patients and review of the literature. *Neurology* 1997;48:882–90.
- [2] Kim JS, Kim HG, Chung CS. Medial medullary syndrome report of 18 new patients and a review of the literature. *Stroke* 1995;26:1548–52.
- [3] Kumral E, Afsar N, Kirbas D, Balkir K, Ozdemirkiran T. Spectrum of medial medullary infarction: clinical and magnetic resonance imaging findings. *J Neurol* 2002;249:85–93.
- [4] Toyoda K, Imamura T, Saku Y, Oita J, Ibayashi S, Minematsu K, et al. Medial medullary infarction: analyses of eleven patients. *Neurology* 1996;47:1141–7.
- [5] Lu GW, Bennett GJ, Nishikawa N, Hoffert MJ, Dubner R. Extra- and intracellular recordings from dorsal column postsynaptic spino-medullary neurons in the cat. *Exp Neurol* 1983;82:456–77.
- [6] Caplan LR, Tettenborn B. Vertebrobasilar occlusive disease: review of selected aspects. Part 1. Spontaneous dissection of extracranial and intracranial posterior circulation arteries. *Cerebrovasc Dis* 1991;2:256–65.
- [7] Leepert D, Radue EW. Medial medullary syndrome due to vertebral artery dissection. *J Neurol Neurosurg Psychiatry* 2001;70:130–1.
- [8] Nakatsuka M, Mizuno S. Three-dimensional computed tomographic angiography in four patients with dissecting aneurysms of the vertebrobasilar system. *Acta Neurochir (Wien)* 2000;142:995–1001.