



Case Report

Tetraplegia Following Bilateral Medial Medullary Infarction

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Abstract

Bilateral medial medullary infarction is rare. We report a 54-year-old man who presented with a 3-day history of dysarthria and progressive weakness in all four limbs. He suffered from respiratory failure 10 days later. Brain magnetic resonance imaging showed acute infarction of the bilateral medial medulla, characterized by a typical heart-shaped sign. Intravenous heparin was given for his cerebral infarction; the patient survived but with tetraplegia. Respiratory failure caused by bilateral medial medullary infarction can be fatal. With improvements in diagnostic technology, and the application of appropriate respiratory care, the survival rate of patients with bilateral medial medullary syndrome has increased. Early diagnosis with adequate treatment of respiratory symptoms may improve the clinical outcome. (*Tzu Chi Med J* 2009;21(3):248–250)

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1. Introduction

Bilateral medial medullary infarction is rare (1). Typical clinical manifestations include tetraparesis (though not in the face), loss of deep sensation in all four limbs, dysphagia, dysarthria, and respiratory failure. Occlusion of the vertebral or anterior spinal artery—or their small branches—will cause infarction of the paramedian region of the medulla oblongata, which is consistent with the above symptoms (2). We report a patient with bilateral medullary infarction with a typical heart-shaped sign shown on brain magnetic resonance imaging (MRI) (3).

2. Case report

A 54-year-old man with hypertension and diabetes presented to our emergency department with progressive malaise, dysphagia, hiccoughs, and left

hemiparesis for 3 days. His blood pressure was 170/106 mmHg, pulse rate was 77 beats/minute, and respiratory rate was 18/minute. He was well oriented, with intact speech. There was no central type facial palsy, diplopia, vertigo, or hearing impairment. The muscle strength of the left upper and lower extremities was weak with a score of 4/5 on the Medical Research Council (MRC) scale. Generalized hyperreflexia (3+, symmetric) and bilateral positive Babinski signs were found. Vibration sensations over the four limbs were lowered. Pain and temperature sensations were intact.

MR diffusion-weighted imaging (DWI; Fig. 1) showed vague small hyperintense lesions at the bilateral pyramids of the middle medulla. Muscle weakness progressed over the following 3 days; the MRC score was 3/5 over the left limbs and 4/5 over the right limbs. Because brainstem infarction was suspected, intravenous heparin was started on the day of admission at 1000U/hour and was adjusted according to the

activated partial thromboplastin time (aPTT). The aPTT was checked twice daily to maintain a therapeutic aPTT target of 1.5–2.0 times control. Three days later, DWI showed hyperintensity over the bilateral medial medulla (Fig. 2). MR angiography (MRA) showed no significant occlusion of the basilar or vertebral arteries (Fig. 3). The patient's condition deteriorated. He became completely tetraplegic, and was respirator-dependent due to paralysis of the respiratory muscles and aspiration pneumonia. The patient, however, was alert during the 10-day hospitalization. Because the respiratory problems persisted—and in an attempt to avoid recurrent aspiration pneumonia—a tracheostomy was performed on the 14th day of hospitalization. After a 3-week hospitalization, the patient was discharged to a nursing home because

of respirator dependency and tetraplegia. He was bedridden and respirator-dependent on follow-up 1 year later.

3. Discussion

Medial medullary syndrome is characterized by ipsilateral hypoglossal nerve palsy, contralateral hemiparesis sparing the face, and impairment of contralateral deep sensation (4). Bilateral medial medullary infarction is rare and only a few cases have been reported (1,5).

In the first 3 days, our patient developed dysphagia—this is more common in patients with lateral medullary infarction, but not uncommon in medial medullary infarction (6). Brain MRI showed the infarct area was in the middle and upper level of the

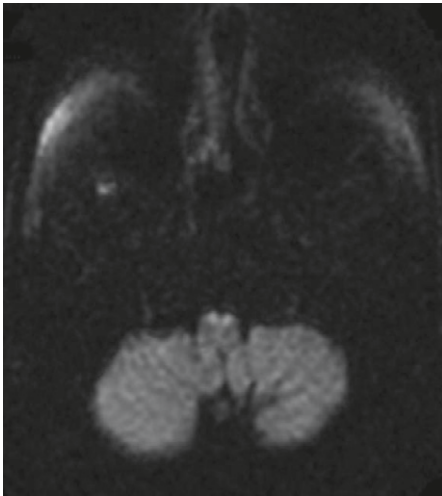


Fig. 1 — Magnetic resonance diffusion-weighted image demonstrating hyperintense signals in the bilateral pyramids over the upper medulla.



Fig. 3 — Magnetic resonance angiography showing no occlusion of the vertebral and basilar arteries.

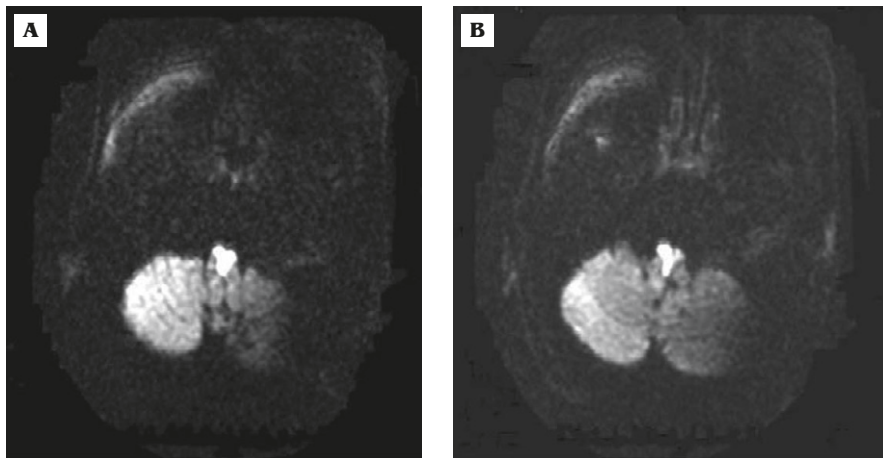


Fig. 2 — Magnetic resonance diffusion-weighted images showing hyperintense signals over the middle (A) and upper (B) medial medulla.

paramedian medulla—this caused severe dysphagia and might have damaged the nuclei of the bilateral hypoglossal nerve (7). Katoh and Kawamoto classified the affected areas of bilateral medial medullary infarction into type 1, with an infarction area from the medullary pyramid to the pontine medial longitudinal fasciculus, and type 2, with the infarction confined to the bilateral medullary pyramids. Our patient was classified as having type 1, which has a worse prognosis (5).

Occlusion of the vertebral or anterior spinal artery—or their branches to the paramedian region of the medulla oblongata—is the usual cause of bilateral medial medullary infarction (5). Vertebral artery dissection is another possible etiology (8). Because the MRA did not show vertebral or basilar artery obstruction, occlusion of the anterior spinal artery or small branches of vertebral artery may have been the cause of the stroke in our patient.

Aspiration pneumonia is the major cause of death in bilateral medullary infarction (5). Respiratory failure due to aspiration is fatal when airway management is inadequate. Recognizing the severity of the respiratory symptoms in these patients is critical to avoid recurrent aspiration pneumonia and to improve the outcome. In our case, a tracheostomy with appropriate respiratory care reduced the risk of aspiration pneumonia and associated complications in the acute phase of the stroke. In previous case reports, the effective treatment of bilateral medial medullary infarction was inconclusive. Intravenous or intra-arterial thrombolysis in patients with basilar artery occlusion may be beneficial (9). Recently, one study reported that a combination of intravenous thrombolytics and consecutive endovascular mechanical thrombectomy might be an option in this difficult condition (10). Early use of heparin in acute cerebral infarctions has been studied and the results are diverse (11). The most convincing study for heparin use was the International Stroke Trial, which enrolled almost 20,000 patients. It found that stroke recurrence was slightly reduced following heparin use (12). For effective anticoagulation in the treatment of acute cerebral infarction, the dosage of heparin should be adjusted to keep the aPTT 1.5–2 times the patient's pre-heparin aPTT, which is similar to treatment of acute myocardial infarction (13). Although there is a lack of strong evidence, anticoagulation with heparin is an acceptable and safe therapy in acute cerebral infarction (14).

A combination of bulbar symptoms and progressive tetraplegia sparing the face suggests a lower

brainstem lesion, possibly bilateral medial medullary infarction. Brain MRI is a useful and sensitive tool for early detection of this condition. Although it has a grave prognosis, a correct diagnosis with aggressive treatment of respiratory problems may improve the survival rate in patients with bilateral medial medullary infarction.

References

1. 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.
2. Gan R, Noronha A. The medullary vascular syndromes revisited. *J Neurol* 1995;242:195–202.
3. Maeda M, Shimono T, Tsukahara H, Maier SE, Takeda K. Acute bilateral medial medullary infarction: a unique 'heart appearance' sign by diffusion-weighted imaging. *Eur Neurol* 2004;51:236–7.
4. Kim JS, Choi KD, Oh SY, et al. Medial medullary infarction: abnormal ocular motor findings. *Neurology* 2005;65:1294–8.
5. Katoh M, Kawamoto T. Bilateral medial medullary infarction. *J Clin Neurosci* 2000;7:543–5.
6. Aydogdu I, Ertekin C, Tarlaci S, Turman B, Kiylioglu N, Secil Y. Dysphagia in lateral medullary infarction (Wallenberg's syndrome): an acute disconnection syndrome in premotor neurons related to swallowing activity? *Stroke* 2001;32:2081–7.
7. Kim H, Chung CS, Lee KH, Robbins J. Aspiration subsequent to a pure medullary infarction: lesion sites, clinical variables, and outcome. *Arch Neurol* 2000;57:478–83.
8. Kataoka S, Terasawa H, Tohji H. Sequential bilateral medullary infarction due to vertebral artery dissection. *Cerebrovasc Dis* 2007;24:309–12.
9. Lindsberg PJ, Mattle HP. Therapy of basilar artery occlusion: a systematic analysis comparing intra-arterial and intravenous thrombolysis. *Stroke* 2006;37:922–8.
10. Pfefferkorn T, Mayer TE, Opherck C, et al. Staged escalation therapy in acute basilar artery occlusion: intravenous thrombolysis and on-demand consecutive endovascular mechanical thrombectomy: preliminary experience in 16 patients. *Stroke* 2008;39:1496–500.
11. Gubitz G, Sandercock P, Counsell C. Anticoagulants for acute ischaemic stroke. *Cochrane Database Syst Rev* 2004;CD000024.
12. Saxena R, Lewis S, Berge E, Sandercock PA, Koudstaal PJ. Risk of early death and recurrent stroke and effect of heparin in 3169 patients with acute ischemic stroke and atrial fibrillation in the International Stroke Trial. *Stroke* 2001;32:2333–7.
13. Samuels MA. *Manual of Neurologic Therapeutics*, 7th edition. Philadelphia: Lippincott Williams & Wilkins, 2004: 369–87.
14. Merwijk G, Lodder J. Presumed basilar artery thrombosis and anticoagulation: a survey of 61 cases. *Cerebrovasc Dis* 1991;1:227–30.