

Bilateral Medial Medullary Infarction Manifested as Sensory Ataxia : A case Report and Review of the Literature

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Medial medullary infarction is usually manifested as hypoglossal palsy, limb weakness, impairment of proprioception, and oculomotor disturbance. We report a case with the unusual presentation of sensory ataxia. A 71 year-old male presented with ataxia and disequilibrium. Bilateral dysmetria, truncal ataxia, Upbeat nystagmus, and impaired vibration and position sense were the clinical features. However, weakness of the limbs, tongue, or face was not definite. MRI revealed bilateral lesion in the medullary tegmentum, and cortical potentials of somatosensory evoked response were absent. Recovery was fair after treatment. According to the literature, motor weakness is a cardinal manifestation in medial medullary infarction, and there has been no reported case presented as sensory ataxia preserving motor power. Current development of diagnostic tools would contribute to define a variety of clinical manifestations, topography, vascular concomitants, and prognosis in medial medullary infarction.

Key Words: Cerebral infarction, Cerebrovascular disorders, Medulla oblongata, Medial medullary syndrome, Ataxia

INTRODUCTION

Infarction of the medial medulla has been known to be a rare clinical entity of cerebrovascular diseases (Davison, 1937; Davison, 1944). It is ascribed to occlusion of the anterior spinal artery (ASA), or penetrating branches of vertebral or basilar arteries (VBA). Well-known manifestations are ipsilateral weakness of the tongue, contralateral hemiparesis, contralateral impairment of proprioception, and oculomotor disturb-

ance. However, it may be difficult to make a clinical diagnosis if some of these features are missing. Modern development in MR imaging has contributed to the detection of smaller lesions of the brainstem (Sawada et al., 1990), and the detection of cases with medial medullary infarction is increasing. The clinical features of our patient were atypical compared with previous reports, so the case here is presented with an extensive review of previous literature (Table 1). A new classification of medial medullary syndrome based on vascular territory, cause, symptoms, and its prognosis will be discussed.

CASE REPORT

A 71 year-old right-handed man was admitted due to severe ataxia and disequilibrium. Two days

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before admission, he had felt numbness on both upper extremities. Next morning, he had had difficulties in standing and walking due to severe disequilibrium. He had a history of right hemiparesis and hemianesthesia 5 years before. Some medications had been prescribed for hypertension, angina pectoris, chronic azotemia and hyperuricemia. He has been

smoking about a pack of cigarettes per day for the past 20 years. His father and brother died from strokes. On initial physical examination, the heart rhythm was regular and murmurs were absent. He was alert and oriented. Pupils were isocoric and light reflex was prompt. Extraocular movements were full, but up-beating vertical nystagmus was present during

Table 1. Vascular territories and neurologic deficits in medial medullary infarction (analysis of 15 reported cases)

Author, year	Vascular territory	Motor weakness	Tongue weakness	Proprioceptive dysfunction	Oculomotor disturbance	Course
Davison (1937)	R)ASA	L)Hemi R)FP	(+)	(+)	Horizontal(N) B)CGP*	Died 10 months later
Davison (1937)	L)ASA	R)Hemi	(-)	(+)	Unchecked	Died 8 months later
O'Brien et al. (1945)	ASA	Quadri	(+)	(+)	Vertical(N) PMDG*	Died on day 12
Brown et al. (1961)	BA(?)	L)Hemi L)FP	(+)	Unchecked	Unchecked	Static
Meyer et al. (1962)	ASA	Quadri R)FP	(+)	(-)	(D)	Died at 7th week
Fisher et al. (1965)	R)VA	L)Hemi	(-)	Unchecked	Absent	Died on day 25
Chokroverty et al.(1975)	Undefined	L)Hemi L)FP	(-)	(-)	Absent	Died 5 weeks later
Leestma et al. (1976)	Undefined	R)Hemi R)FP	(-)	(-)	Absent	Improved
Ropper et al. (1979)	R)VA(?)	L)Hemi L)FP	(-)	(+)	Horizontal(N) Vertical(D)	Improved
Mizutani et al. (1980)	ASA	Quadri	(+)	(+)	Upbeat(N)	Died 8.5 months later
Ho et al. (1981)	Undefined	R)Hemi R)FP	(-)	(-)	Absent	Improved
Kase et al. (1983)	ASA	Quadri B)FP	Unchecked	(+)	Upbeat(N) Bobbing	Died on day 12
Paulson et al. (1986)	ASA	Quadri	Unchecked	(+)	Unchecked	Improved
Sawada et al. (1990)	Undefined	L)Hemi	(-)	(+)	Absent	Improved
Toyoda et al. (1992)	ASA	Quadri R)FP	(+)	(-)	Horizontal(N) Ophthalmoplegia	Static
Present case (1994)	Undefined	Absent	(-)	(+)	Upbeat(N) (D)	Improved

Abbreviations : BA=Basilar artery(or branches) ; ASA=Anterior spinal artery ; VA=Vertebral artery ; Quadri=Quadripareisis ; Hemi=Hemiparesis ; FP=Facial palsy ; R)=Right ; L)=Left ; B)=Both ; (N)=Nystagmus ; (D)=Diplopia ; PMDG=Poor maintenance of depression of gaze ; CGP=conjugate gaze palsy

upward and lateral gaze. Facial expression, sensation, and soft palate movement were symmetrical. Tongue deviation or atrophy was absent. Muscle tone was normal and strength was greater than MRC grade IV. Vibration sense was decreased on both sides and joint position sense was impaired on the right toe. Tendon reflexes were symmetrically increased, but Babinski reflex was flexor response. Bilateral dysmetria and clumsiness were observed on finger-to-nose test, heel-to-shin test, and rapid alternating movement. Romberg's test was positive. Gait was impossible due to severe truncal ataxia. The blood test showed increased levels of blood urea nitrogen, creatinine, and uric acid. Electrocardiography and echocardiography demonstrated no characteristic findings except LVH. Brain MRI revealed a well demarcated bilateral linear increased T_2 -signals in the rostral medullary tegmentum (Fig. 1). In MR subtraction angiography, basilar and vertebral arteries were competent, but both internal carotid arteries were tortuous, and the right middle cerebral artery was irregularly narrowed. Cortical potentials were absent bilaterally in the median nerve sensory evoked potential (MNSEP) study. After admission, neurologic de-

ficits did not progress any more. And within two weeks, he was able to stand and walk with the conservative treatment.

DISCUSSION

Clinical manifestations of medial medullary infarction can be summarized into three categories (Table 2). Hemiparesis or quadriplegia was present unexceptionally. Tongue weakness was observed in about half (6/13), and most of them (5/6) in ASA syndrome. Typical alternating hemiparesis with ipsilateral hypoglossal palsy and contralateral limb weakness was rare (1/13). Mild facial weakness was also common (9/15) and this was relatively frequent in VBA syndrome. In our case, motor weakness of the limbs, tongue, or face was not observed, which means that the corticobulbar and corticospinal tracts were not seriously affected. Ataxia shown in our patient was presumed

Table 2. Symptoms and signs in medial medullary infarction (analysis of 15 reported cases)

Symptoms and Signs	Number of cases	Frequency(%)
Motor Weakness	15/15	100
Limb	15/15	100
Hemiparesis	9/15	60
Quadriplegia	6/15	40
Tongue	6/13*	46
Face	9/15	60
Proprioceptive Dysfunction	8/13**	62
Loss of vibration or position sense	8/13	62
Sensory ataxia	0/13	0
Oculomotor disturbance	7/12***	58
Upbeat nystagmus	4/12	33
Horizontal nystagmus	3/12	25
Ocular bobbing	1/12	8
Gaze paresis (upward or lateral)	2/12	17
Poor maintenance of depression of gaze	1/12	8
Diplopia	2/12	17

* No description or comment about tongue weakness in other 2 cases.

** No description or comment about sensory examination in other 2 cases.

*** No description or comment about oculomotor findings in other 3 cases.



Fig. 1. T2-weighted magnetic resonance imaging without gadolinium-DTPA enhancement (done at 5 day from onset). On sagittal section of the rostral medulla, bilateral linear high signal intensity lesion is present in the tegmentum (black arrow).

to be sensory type because it was definitely aggravated by eye closure, and position sense was severely impaired. The main cause was supposed to be involvement of bilateral medial lemniscus. Findings of MNSEP which may assess the electrophysiological integrity of the medial lemniscus (Sawada et al., 1990) were compatible with the anatomical location of lesions in MRI. Although the loss of vibration or position sense was common in medial medullary infarction (8/13), moderate to severe limb weakness was always combined, therefore an exact evaluation of limb ataxia or Romberg's test might be difficult. Our patient was an exception because his motor power was sufficient for such tests even during the early stage of the disease. According to the literature (Table 1), occlusion of ASA was detected in eight out of eleven cases with defined vascular territories, and perforating branches of VBA in three cases (Brown and Fang, 1961; Fisher and Curry, 1965; Ropper et al., 1979). But we think that the four undefined cases might be ascribed to the occlusion of penetrating branches rather than ASA. It is our suggestion to reclassify the medial medullary syndrome as "classical" ASA syndrome and VBA syndrome. In our case, no potential source of cardiac or large arterial embolism could be found. But the history of hypertension, and the location and topography of infarction are highly suggestive of a small-vessel disease. The prognosis of medial medullary infarction is usually known to be poor, especially in the infarction of ASA territory which often result in respiratory distress and severe motor weakness. However, the prognosis was variable according to our review. Extensive infarction of ASA territory due to embolism resulted in a graver outcome, while small deeply located lesions ascribed to small-vessel disease showed only minor neurologic deficits and more favorable outcome as in our case. In conclusion, we suggest that VBA syndrome due to small-vessel disease will probably result in better prognosis than the embolic ASA syndrome, which can be differentiated by its clinical features and MRI findings.

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