

Babinski-Nageotte Syndrome on Magnetic Resonance Imaging

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A 70-year-old woman developed left hypoglossal nerve palsy, a right hemiparesis sparing the face, and a typical left Wallenberg's syndrome. These symptoms resulted from a lesion in the left half of the medulla oblongata, suggesting Babinski-Nageotte syndrome, a rare cerebrovascular disease. This is the first case of ischemic infarction in the territory of the left vertebral artery and posterior inferior cerebellar artery demonstrated on magnetic resonance imaging. Severe bilateral lesions of the distal vertebral arteries demonstrated on digital subtraction angiography may have contributed to the development of this syndrome. (*Stroke* 1991;22:272-275)

In 1902, Babinski and Nageotte¹ first reported three autopsy cases characterized by symptoms due to an ischemic lesion of the medulla oblongata involving the unilateral lateral and medial areas of the medulla.^{2,3} The identity of this syndrome has not been clearly established, and the distribution of the vascular disease is still under discussion. Only a few cases have been described up to now.^{2,4-8}

We report a typical case of Babinski-Nageotte syndrome in a patient with an ischemic lesion in the medulla oblongata shown on magnetic resonance imaging (MRI). Cerebral angiography revealed severe stenosis and occlusion of the vertebral arteries.

Case Report

A 70-year-old woman who had suffered from hypertension and diabetes mellitus for 20 years was admitted to St. Mary's Hospital on April 8, 1989, because of a sudden headache, vomiting, and right-sided weakness. On admission, her blood pressure was 180/120 mm Hg and her pulse was regular at 72/min, but her breathing was severely irregular with markedly decreased movement of the right chest wall. Soon after arriving at the hospital, spontaneous respiration ceased and she lapsed into a coma. Artificial ventilation was started, and her level of consciousness quickly improved to drowsy. Her eyes deviated to the right for a few days, and the left corneal reflexes were diminished. Her pupils were

slightly anisocoric (left < right), but the light reflexes were prompt. She had a right hemiparesis with decreased deep tendon reflexes. The movement of her soft palate and tongue could not be examined. No sensory impairment was apparent.

On admission, her leukocyte count was 12,100/mm³, erythrocyte count 506×10⁴/mm³, hemoglobin concentration 16.1 g/dl, hematocrit 48.8%, platelet count 24.0×10⁴/mm³, and serum glucose concentration 281 mg/dl. Blood gas studies on room air revealed hypoventilation; PO₂ was 48.7 mm Hg, PCO₂ 40.8 mm Hg, and pH 7.336. Cardiomegaly was observed on a chest roentgenogram (cardiothoracic ratio 64%). Cranial computed tomography on day 3 showed a low-density area in the left dorsal hemisphere of the cerebellum. An additional infarcted area in the left half of the medulla oblongata was revealed by MRI on day 19. Obstruction or blood flow stagnation in the left vertebral artery was also suggested by MRI (Figure 1, top). There were no lesions in the bilateral pons or the bilateral cerebral hemisphere. Cerebral digital subtraction angiography on day 36 revealed severe stenosis of the right distal vertebral artery and complete occlusion of the left vertebral artery at the third segment, with retrograde blood flow from the right vertebral artery (Figure 2).

Her level of consciousness and respiratory condition gradually improved. A neurologic examination was repeated after successful extubation on day 46. She was alert, and her orientation was good. She had dysarthria and was hoarse. She revealed a left Horner's syndrome, including left facial hypohidrosis. Her ocular movements were full, with a horizontal nystagmus in both directions on horizontal gaze. The left soft palate was paralytic, and the left side of her tongue was atrophic with fasciculations, indicat-

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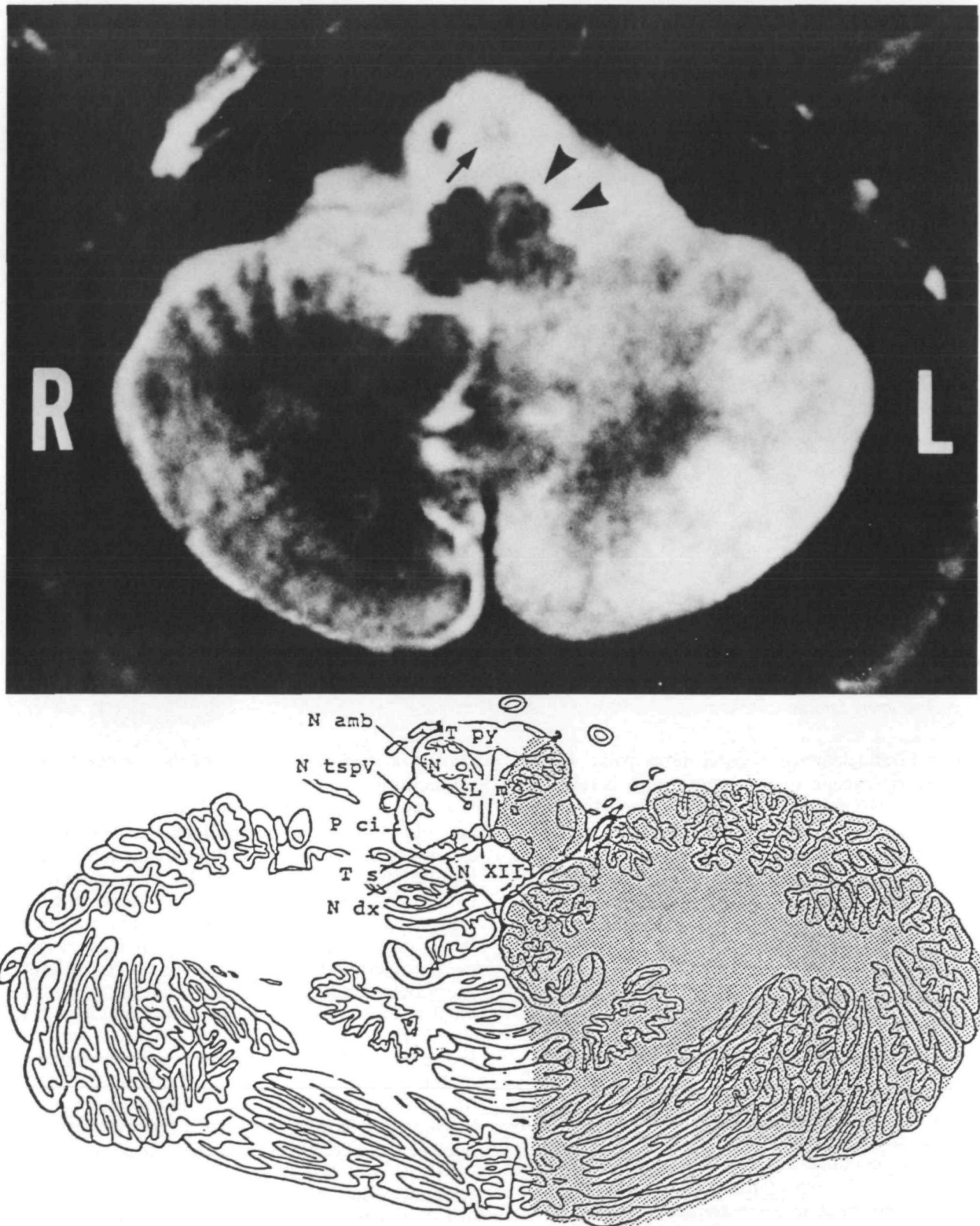


FIGURE 1. Top: T2-weighted magnetic resonance imaging scan (repetition time 2,000 msec; echo time 90 msec) of axial section of medulla oblongata and cerebellum in 70-year-old woman. Areas of hyperintense signal in left half of medulla oblongata (arrowheads) and ipsilateral left dorsal cerebellar hemisphere are demonstrated. Although lumen of right vertebral artery shows flow void signal, lumen of left vertebral artery shows hyperintense signal (arrow), suggesting stagnant blood flow. R, right; L, left. Bottom: Schematic illustration of cross-section of medulla oblongata and cerebellum. Anatomic correlates with affected areas are shaded. T py, tractus pyramidalis; N o, nucleus olivaris; L m, lemniscus medialis; N XII, nucleus nervi hypoglossi; N amb, nucleus ambiguus; N tspV, nucleus tractus spinalis nervi trigemini; P ci, pedunculus cerebellaris inferior; T s, tractus solitarius; N dx, nucleus dorsalis nervi vagi.

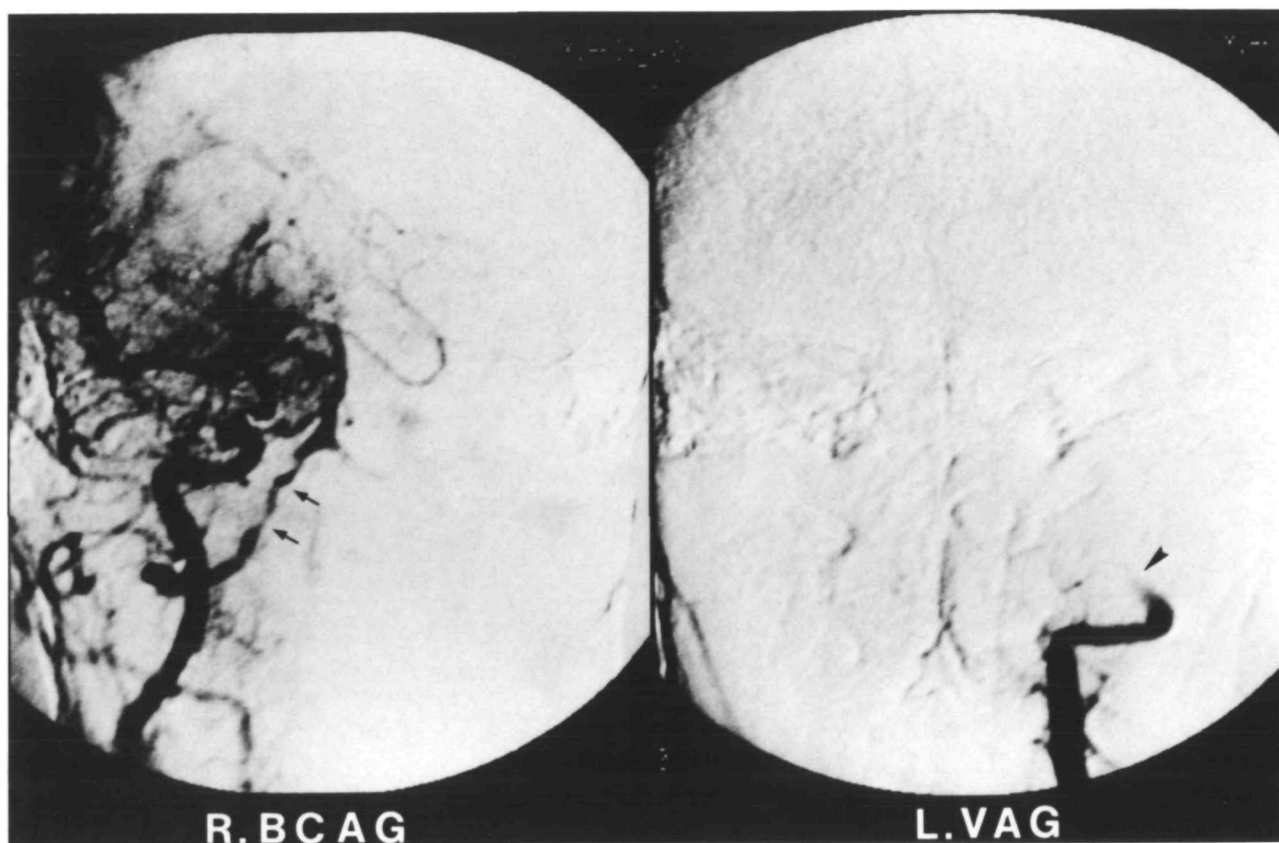


FIGURE 2. Digital subtraction angiograms of right brachiocephalic artery (R.BCAG) and left vertebral artery (L.VAG), anteroposterior view. Left: Severe stenosis of distal portion of right vertebral artery (arrows). Right: Occlusion of distal portion of left vertebral artery (arrowhead).

ing left infranuclear hypoglossal nerve palsy. Right hemiparesis, except for the face, and cerebellar ataxia of the left extremities were observed. Her deep tendon reflex was increased on the right side, and the planter reflex was normal.

The sensations of pain and temperature on the right side of her face and in her left limbs were diminished, and proprioception was bilaterally normal. These signs and symptoms were considered to be caused by the involvement of the left lateral and medial medulla oblongata and were compatible with those of Babinski-Nageotte syndrome.

Discussion

Babinski-Nageotte syndrome is described as a combined lateral and medial medullary syndrome, which is characterized by contralateral hemiplegia, tactile/proprioceptive sensory loss, and ipsilateral weakness and wasting of the tongue.² The arterial supply to the medulla is anatomically divided into four territories, namely, the median, paramedian, lateral, and dorsal areas, to which blood is perfused through the anterior spinal rami of the vertebral artery and basilar artery, branches from the short and long lateral or transverse arteries of the vertebral artery, penetrating branches of the short and long transverse arteries of the vertebral artery, and the

longest of the lateral arteries of the vertebral artery, respectively.^{3,9}

Because of such characteristic vascular distribution and blood supply, it seems rare that an ischemic lesion would involve the medulla oblongata. In addition to signs of lateral medulla involvement, our patient had ipsilateral weakness and atrophy of the tongue and contralateral hemiparesis (except for the face), suggesting both lateral and, at least in part, medial medullary infarction. Since contralateral proprioception was intact, the medial lemniscus, supplied by the anterior spinal arteries, might have been spared from the ischemic insult (Figure 1, bottom). In addition to those symptoms, conjugate deviation of the eyes and respiratory failure were observed during the acute stage; these symptoms disappeared thereafter, indicating a transient involvement of the pons or the right side of the medulla oblongata.

In our case MRI clearly demonstrated a diffuse ischemic lesion in the territory of the left vertebral artery and the posterior inferior cerebellar artery. Cerebral angiography on day 36 revealed severe stenosis of the right distal vertebral artery concomitant with occlusion of the left vertebral artery and posterior inferior cerebellar artery. We assume that these vascular lesions induced ischemia mostly in the territory perfused from the left distal vertebral artery and caused an infarct in the left half of the medulla

oblongata because collaterals from the basilar artery and anterior spinal artery might have been insufficient. To our knowledge, this is the first case of Babinski-Nageotte syndrome demonstrated by MRI and angiography.

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KEY WORDS • cerebral infarction • magnetic resonance imaging • medulla oblongata