

Ipsilateral Hemiplegia and the Wallenberg Syndrome

Surjit K. Dhamoon, MD; Javaid Iqbal, MD; George H. Collins, MD

● We documented the anatomic basis for hemiplegia occurring on the same side of the body as the lateral medullary infarction. Extension of the zone of ischemia into the rostral spinal cord involves corticospinal fibers after they cross from the opposite side.

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The occurrence of pyramidal tract signs as part of the syndrome associated with lateral medullary infarction is unusual.¹ Autopsy studies of most of these cases found that the medullary pyramid ipsilateral to the lateral medullary infarction was involved.² In some cases, however, the pyramidal signs have been ipsilateral to the medullary infarction.³⁻⁶ Most of these cases have been clinical reports without autopsy. In only one report did pathologic studies verify that the ischemic effect extended caudally into the cervical spinal cord and that it involved the corticospinal fibers caudal to the pyramidal decussation.⁶ Because of the rarity of such an occurrence and the lack of complete photographic documentation of this phenomenon, we present this case.

REPORT OF A CASE

A 78-year-old man with a history of hypertension, diabetes, and angina was admitted to the hospital with complaints of nausea, vomiting, and hoarseness. Two weeks prior to admission, he had begun to

have a productive cough without fever or chest pain. He was recovering from these symptoms until the evening of his admission, when nausea, vomiting, and hoarseness suddenly developed. He was unable to get out of bed and was brought to the emergency room.

On admission, his vital signs were normal, but his BP was 190/100 mm Hg. Extraocular movements were normal, and his pupils were equal in size and were reactive to light and accommodation. A low-pitched bruit in the right carotid area and a grade 2/6 systolic murmur in the aortic area were heard. The neurologic examination found that the patient was verbal and responsive but drowsy. Cranial nerves II, III, IV, and VI were intact. Facial sensation and jaw mobility were not evaluated. A flattened nasolabial fold was noted on the right side of the face. The patient had no gag reflex bilaterally, and his voice was hoarse. There was no tongue deviation, but weakness of head turning to the left was found. A right hemiparesis was present with symmetric tendon reflexes and a bilateral Babinski's sign. Sensory (including hearing) and cerebellar functions could not be tested.

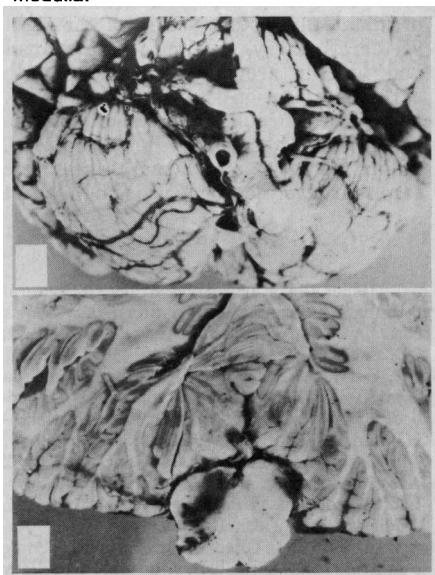
Findings from routine studies, including blood chemistry and enzymes, were normal except for a blood glucose level of 274 mg/dL. Chest roentgenogram showed mild cardiomegaly and increased markings at the base of the right lung. An ECG showed left bundle-branch block and a normal sinus rhythm. No mural thrombus was found from M-mode and two-dimensional echocardiography.

A neurologist found a right central facial palsy and a dense right hemiplegia (arm and leg weakness was equal). The patient's speech was slightly slurred. A deep left cerebral infarct was suggested, but the computed tomographic scan was normal. The patient's condition was stable for two days. On the third hospital day, respiratory arrest developed. He was intubated and resuscitated, but was found without respiration or pulse on the 12th day. He died the following day.

General autopsy studies found atherosclerosis of the coronary artery; a healed infarct of the anterior wall of the left ventricle; left ventricular hypertrophy (590 g); and nodular hemorrhagic areas of the left lower lobe consistent with pulmonary infarcts.

The formaldehyde-fixed brain weighed 1,140 g. On gross inspection, cerebral gyri and sulci appeared normal in size and configuration. Examination of the cerebral vessels showed thrombosis and occlusion of the terminal 3 cm of the right vertebral artery that included the origin of the posterior inferior cerebellar artery (PICA), 1.5 cm from the vertebrobasilar junction (Fig 1, top). The left vertebral artery was sclerotic, dilated, and patent. The basilar artery was severely atherosclerotic. Arteries of the circle of Willis were also atherosclerotic, with a 50% narrowing of the

Fig 1.—Top, Ventral aspect of brain stem and cerebellum showing tortuous and thrombotic right vertebral artery. Bottom, Cross section of infarcted zone in right superolateral aspect of medulla.



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From the Departments of Neurology (Drs Dhamoon and Iqbal) and Pathology (Dr Collins), State University of New York, Upstate Medical Center, Syracuse.

Reprint requests to Department of Pathology, Upstate Medical Center, 766 Irving Ave, Syracuse, NY 13210 (Dr Collins).

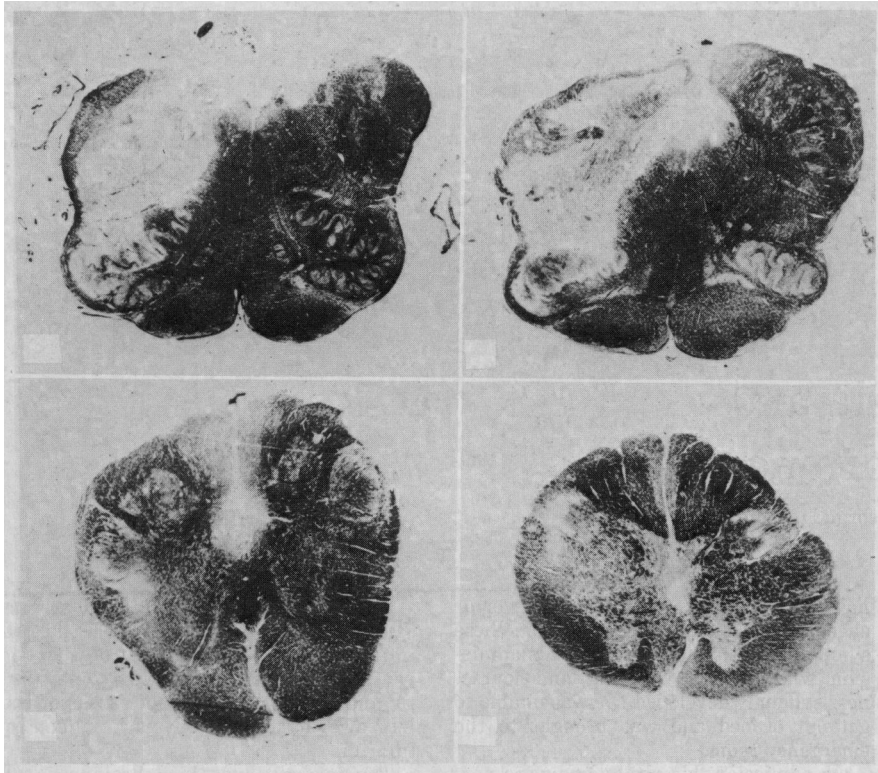


Fig 2.—Infarct distribution in cross sections of high, middle, and low medulla (top left, top right, and bottom left). Bottom right, Pale area of myelinated fiber loss affecting decussated fibers of pyramidal tract on right side at level of junction of medulla and spinal cord (Klüver-Barrera stain).

middle and anterior cerebral arteries. On coronal sectioning, the lateral ventricles appeared normal in size and shape. The left cerebral hemispheric white matter had a small (5 × 2 mm) cavity in the border zone between the cortical and central circulation. A similar cavity, 2 × 2 mm, was seen in the anterior limb of the right internal capsule. Midbrain and pons sections appeared normal. Coronal sections of the medulla showed infarction in the right lateral medullary region, but not in the pyramids nor in the remaining paramedian tissue (Fig 1, bottom). Coronal sections of the cerebellum appeared normal. In the proximal and distal sections of the right vertebral artery, severe atherosclerosis and a recent thrombus were present with early organization of the thrombus. A right lateral medullary infarction was seen in the rostral medulla (Fig 2, top left). Pallor, necrosis, and hemorrhages were seen that involved the nucleus and tract of

cranial nerve V, the ambiguous nucleus, the solitary tract, the spinocerebellar and spinothalamic tracts, and other structures in this region (Fig 2, top left and right). Paramedian structures were spared. In the caudal medulla and medullocervical region, the right lateral medullary infarct persisted (Fig 2, bottom left and right). The crossed pyramidal tract was affected by the infarct immediately at the point of decussation from the left pyramid to the right lateral white columns in the rostral cervical cord (Fig 2, bottom right).

COMMENT

Because the clinical information is incomplete in our case, many of the typical features of a lateral medullary infarction are lacking. Nevertheless, on clinical grounds alone, it is still possible to identify the lateral medulla as the target area despite some

unusual features, including right facial paresis, right sternocleidomastoid weakness, and right hemiplegia. The right facial paresis, an occasional occurrence in this syndrome,⁴ has been explained by involvement of corticobulbar fibers coursing from the pyramidal tract to the facial nucleus through the rostral medulla.⁷ Involvement of sternocleidomastoid muscle function implies caudal extension of the infarction to the level of the 11th cranial nerve, with an interruption of its motor fibers within the neuraxis. The right hemiplegia, which developed from an initial right hemiparesis, indicates an evolution of the primary process so as to involve either the contralateral pyramid or the ipsilateral corticospinal tract after decussation of the pyramids. The pathologic data clearly demonstrate involvement of the ipsilateral corticospinal tract.

We probably cannot determine the precise vascular mechanism responsible for such an occurrence because of the marked variability in the blood supply to the affected areas.⁸ However, because of the restricted nature of the infarct in this area, relative ischemia was probably a causal factor. The thrombosis in the vertebral artery occluded the lateral medullary branches, the PICA, and the anterior spinal artery. The distribution of the main body of the infarct can be explained by a vertebral artery effect, alone or in combination with the PICA. There was no evidence of tissue damage in the distribution of the anterior spinal artery at the level of the medulla, nor in the distribution of the posterior spinal artery at the level of the spinal cord. The area of spinal cord that was involved, however, can be regarded as a junctional zone between the anterior and posterior spinal artery and between the vertebral and spinal artery supplies.⁹⁻¹¹ We can thereby identify a potential mechanism for the occurrence of relative ischemia in this area that may account for this unusual extension of the infarct into the spinal cord.

References

1. Fisher CM, Karnes WE, Kubik CS: Lateral medullary infarction: The pattern of vascular occlusion. *J Neuropathol Exp Neurol* 1961; 20:323-379.
2. Currier RD: Syndromes of the medulla oblongata, in Vinken PJ, Bruyn GW (eds): *Handbook of Clinical Neurology*. Amsterdam, North-Holland Publishing Co, 1969, vol 2, chap 10.
3. Castaigne P, Lhermitte F, Gautier JC, et al: Arterial occlusion in the vertebral-basilar system: A study of 44 patients with post mortem studies. *Brain* 1973;96:133-154.
4. Currier RD, Giles CL, DeJong RN: Some comments on Wallenberg's lateral medullary syndrome. *Neurology* 1961;11:778-791.
5. Opalski A: Syndrome partiel de l'artere vertebro-spinale posterieure. *Paris Med* 1946; 1:214-220.
6. Osetowska E, Krasnicka Z: Sur le syndrome sous-bulbaire d'Opalski avec une etude anatomique (contributions aux aspects paradoxaux du syndrome de Wallenberg). *Rev Neurol* 1961; 104:314-320.
7. Kuypers HGJM: Corticobulbar connections to the pons and lower brain stem in man: An anatomical study. *Brain* 1958;81:364-390.
8. Loeb C, Meyer JS: *Strokes due to Vertebro-basilar Disease*. Springfield, Ill, Charles C Thomas as Publisher, 1965, p 53.

9. Chakravorty BG: Arterial supply of the cervical spinal cord and its relations to the cervical myelopathy in spondylosis. *Ann R Coll Surg Engl* 1969;45:232-251.
10. Gillilan LA: The correlation of the blood supply of the human brain stem with clinical brain stem lesions. *J Neuropathol Exp Neurol* 1964;23:78-108.
11. Lazorthes G, Gouaze A, Zadeh J: Arterial vascularization of the spinal cord. *J Neurosurg* 1971;35:253-262.