

Short communication

Sensorimotor hemiparesis with secondary cervical dystonia following lateral caudal medullary infarction without signs and symptoms of Wallenberg syndrome

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Abstract

We report the case of an 84-year-old woman who suddenly developed motor and both superficial and deep sensory hemiparesis on the left side, and cervical dystonia with a head tilt to the right side. A brain MRI showed an infarct in the left lateral caudal medulla. It is clinically important to recognize that the lateral caudal medullary infarction appears without signs and symptoms of lower cranial nerve palsies commonly involved in Wallenberg syndrome.

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1. Introduction

Lateral medullary infarction is one of the most frequently encountered cerebral infarctions involving the brainstem, and often manifests as Wallenberg syndrome [1]. If the ipsilateral pyramidal tract is involved below the decussation by the caudal extension of the lateral medullary lesion, ipsilateral hemiparesis may occur, which is known as Opalsky's syndrome [2,3]. Recently, we have encountered a patient with left lateral caudal medullary infarction who presented sensorimotor hemiparesis and secondary cervical dystonia without symptoms and signs of lower cranial nerve palsies commonly seen in Wallenberg syndrome.

2. Case report

An 84-year-old Japanese woman with a medical history of cardiac ischemic attack (acute myocardial infarction), was admitted because of the sudden gait disturbance and dysarthria. On admission, her general physical condition was unremarkable. Neurological examination revealed that she was alert and well oriented. She had hemiparesis of the left

upper and lower extremities, as well as decreased pain and touch, and decreased temperature sensations on the left side of her face, body, and extremities. There was no weakness of her facial muscles. Her left sternocleidomastoid muscle was paretic. Decreased deep sensation was noted on the left side of the body, and extremities. She also complained of pain on the left side of the neck. Ten days after admission, cervical dystonia with a head tilt to the right side developed (Fig. 1A). There was an abnormal sustained muscle contraction of the right lateral neck muscles, including the sternocleidomastoid and anterior border of the trapezius muscle. Neuroradiological studies well demonstrated the responsible lesion for her symptoms. Diffusion-weighted imaging (DWI) and T2-weighted imaging (T2WI) showed an infarct located on the left lateral caudal medulla (Fig. 1B–F). MR-angiography revealed an absence of blood flow in the left vertebral artery. Her cervical dystonia gradually improved after several weeks; however, her left hemiparesis as well as the loss of deep sensation persisted, and she could not walk without a support.

3. Discussion

It is rare to see Opalsky's syndrome, but the clinical symptoms of our patient are rarer, since her did not

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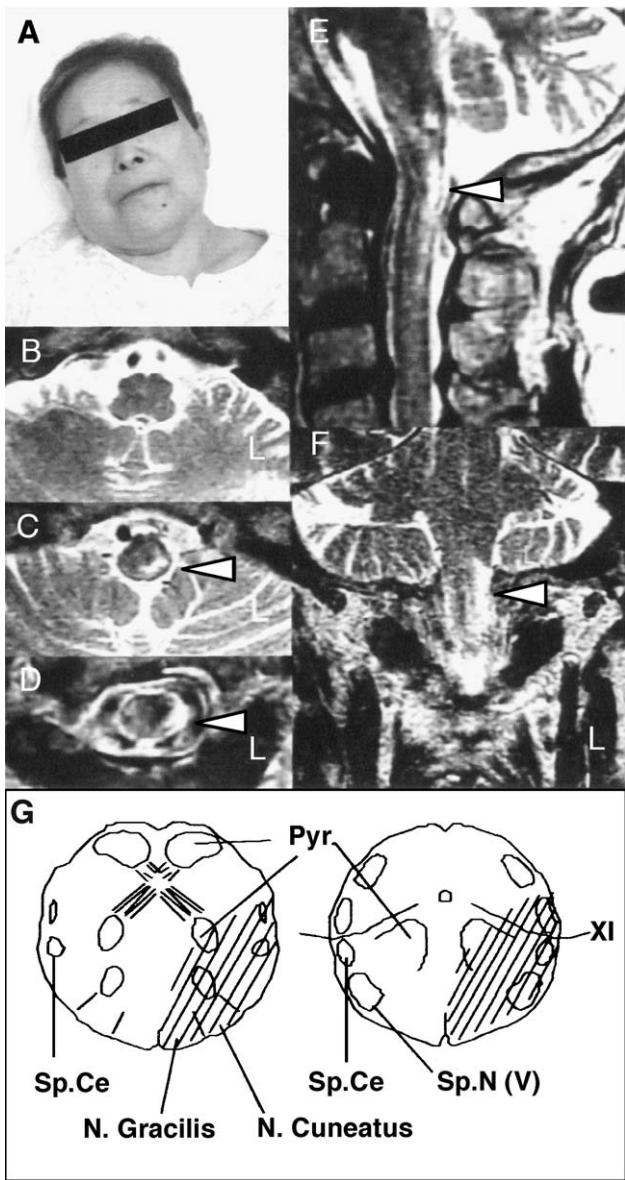


Fig. 1. (A) Cervical dystonia of the present patient is demonstrated. (B)–(F) Brain MRI of the present patient showed an infarct located on the left lateral caudal medulla. (G) The schematic representations of the location of the lesion of the present patient are demonstrated. The lesion may involve the pyramidal tract, the ascending tracts of deep sensation, spinocerebellar tract, the spinal tract nucleus of the trigeminal nerve, and the accessory nerve nucleus. Arrowheads indicate the lesion.

present lower cranial nerve palsies usually seen in lateral medullary infarctions as in Wallenberg or Opalsky's syndrome. Based on the findings of MR imaging, the responsible lesion of our patient probably involved the pyramidal tract, the ascending tracts of deep sensation in

which nucleus cuneatus and nucleus gracilis are located, spinocerebellar tract, the spinal tract nucleus of the trigeminal nerve, and the accessory nerve and its nucleus (Fig. 1G). The lesion of the present patient was more caudal than the lesions of Wallenberg or Opalsky's syndrome. This was the reason the present patient did not show lower cranial nerves palsies. It is clinically important to recognize that the occlusion of the vertebral artery causes the lateral caudal medullary infarction that presents with hemiparesis and the ipsilateral loss of deep sensation without signs and symptoms of Wallenberg syndrome.

Another interesting finding of the present patient is the association with cervical dystonia. On the basis of a recent review of secondary cervical dystonia [4], that is, structural lesions associated with cervical dystonia are most commonly localized to the brainstem and/or cerebellum; it is speculated that the dysfunctioning of the cerebellar afferent pathway, usually that of the olivocerebellar system, plays an important role in generating cervical dystonia [4]. In the present patient, it is unclear whether the patient had cerebellar symptoms because she had severe hemiparesis. However, very likely the lesion directly interrupted the spinocerebellar tract and modified the function of the olivocerebellar system, supporting such speculation. Currently, it is uncertain whether the associated secondary cervical dystonia is a chance phenomenon only seen in the present patient or a significant diagnostic pointer of this rare syndrome of the lateral caudal medullary infarction. Further accumulation of similar cases would be valuable to better understand this rare clinical syndrome.

Finally, when we see a sudden appearance of sensorimotor hemiparesis of the upper and lower extremities without facial involvement, the possibility of not only cervical lesion but also the lateral caudal medullary infarction should be kept in mind.

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