

Lateral Medullary Infarction Presenting Contralateral Palatal Paresis

Chien-Ming Chen and Tzu-Ying Huang

Abstract- Palatopharyngeal paresis has never been reported to be contralateral in the lateral medullary infarction (LMI). A 65-year-old lady with acute dorsolateral infarction in the left medulla presented mild hoarseness, mild dysphagia, mild gait ataxia along with marked hypalgesia and thermoanesthesia on the right limbs. To our surprise, palatal weakness was on the right side, instead of being on the left side as expected in typical Wallenburg syndrome. The palatal paresis was noted during voluntary phonation but turned into normal movement while gag reflex was induced. An involvement of corticobulbar fibers before synapsing into the ipsilateral caudal medulla or a selective involvement of the peri-ambigular reticular formation and/or its post-synaptic connection fiber destined to the contralateral swallowing center presumably underlies such a rare contralateral palatal paresis.

Key Words: Soft palate paresis, Lateral medullary infarction, Nucleus ambiguus, Peri-ambigular reticular formation, Disconnection dysphagia

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INTRODUCTION

Lateral medullary infarction (LMI) is usually secondary to the occlusion of the vertebral artery⁽¹⁾, or less frequently (up to 30%) to a lesion confined only to the territory of the posterior inferior cerebellar artery (PICA), particularly in large inferodorsolateral infarcts⁽²⁾. The triad of Horner syndrome, ipsilateral ataxia and contralateral hypalgesia clinically identifies patients with LMI⁽³⁾. However, unless the paresis of the ipsilateral vocal cord or pharynx is present, other findings do not unequivocally localize the lesion to the later-

al medulla⁽⁴⁾. Clinically soft palate paresis not only localizes a lesion to the lateral medulla but also endorses the lesion side. Such a clinical presentation has never been reported to be contralateral in LMI. We present a patient with such a unique condition along with mild hoarseness, mild gait ataxia and marked contralateral brachiorural hypalgesia and thermoanesthesia.

CASE REPORT

A 65-year-old lady with uncontrolled atrial fibrillation developed swallowing disturbance and sensory loss

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over the right upper limb following vertigo and unsteadiness beginning 4 days earlier. At admission, blood pressure was 130/88 mmHg, heart rate 94/min, respiratory rate 18/min and body temperature 36.0 °C. Neurological examinations indicated mild hoarseness, mild dysphagia (oral feeding feasible despite incapable of drinking a large mouthful of water), markedly diminished thermal and pinprick sensations with preserved light touch and proprioceptive sensations over the right arm and leg. All sensations over the face were intact. Soft palate paresis was evident on the right while attempting phonation saying “ah”, and disappeared when a tongue depressor was applied to induce gag reflex (Fig. A). Tonsils and palatopharyngeal tissue were not swollen nor injected and there was no pain on swallowing. She swayed on tandem walking but did not fall. There was no limb ataxia. Other neurological and physical examinations were unremarkable.

Brain MRI performed the following day disclosed a

high-signal lesion in the dorsolateral aspect of the left middle medulla on T2-weighted, fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted images. Magnetic resonance angiography showed an occlusion of the left vertebral artery (Fig. B). She was treated as ischemic stroke, which was favored by the clinical course--the gait ataxia, hoarseness and dysphagia abated in 3 days with normalization of the palatal movements, although the deficits of superficial sensations over the right limbs persisted.

DISCUSSION

In 2005, a single case of contralateral pharyngeal paralysis, presumably supranuclear origin, caused by a medial medullary infarction was reported. But the soft palate was normal in position and motion although the soft palate reflex was absent on either side⁽⁹⁾. Our patient differed in the soft palate movement that was poor on

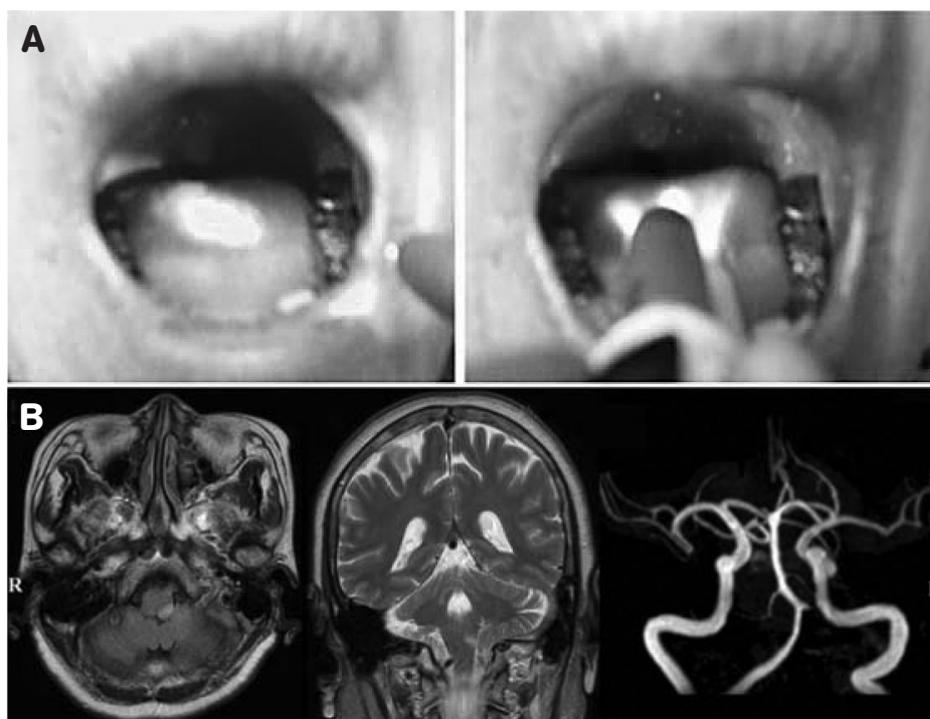


Figure. (A) Palatal elevation was markedly impaired on the right side while attempting phonation saying “ah” (left). It became normal when a tongue depressor coupled with a light pen to illuminate, was applied upon her tongue (right). (B) Cranial MRI: axial fluid attenuated inversion recovery (FLAIR, left) and coronal T2-weighted (middle) images revealed a dorsolateral infarct in the left middle medulla. The left vertebral artery is occluded on magnetic resonance angiography (right).

voluntary phonation but normal on the gag reflex and in the lesion locating at the dorsolateral medulla, in which contralateral palatal palsy has never been reported.

In 33 cases of LMI reported by Kim et al, rostral lesions were usually associated with more severe dysphagia/hoarseness and the presence of ipsilateral facial paresis for the involvement of the nucleus ambiguus (NA) and the looping corticobulbar fibers destined to the facial nucleus respectively. Caudal lesions were correlated with more marked vertigo, nystagmus and gait ataxia by the involvement of the vestibular nuclei, vestibulo-cerebellar pathway and restiform body or the spinocerebellar pathway. Nausea/vomiting and Horner sign were common regardless of the lesion location without rostro-caudal difference⁽⁶⁾.

Bulbar dysfunction is not particular to brainstem lesions. In Venketasubramanian's study addressing the issue of vocal cord paresis in acute ischemic stroke, palatolaryngeal paresis was noted in 20.4% (11 out of 54) of patients, 100% (5/5) in the lateral medullary stroke and 12.2% (6/49) in other strokes. Except in those patients with LMI, the palatolaryngeal paresis was exclusively contralateral. One LMI patient in this study had ipsilateral palatal but strangely contralateral vocal cord paresis, which they could not explain⁽⁷⁾.

Unilateral LMI usually produces bilateral palatal/pharyngeal/laryngeal dysfunctions. Severe bilateral pharyngeal paresis had been demonstrated by video-fluoroscopy and manometry in a patient with unilateral rostral dorsolateral medulla infarction⁽⁸⁾. As compared with hemispheric stroke and peripheral 9th and 10th cranial nerve palsies, dysphagia is more severe in LMI, extremely slow in the pharyngeal phase electrophysiologically. Unilateral LMI produces bilateral dysfunctions of the swallowing muscles, including the submental muscles which are not innervated at the medullary level. An acute disconnection syndrome was proposed for such severe swallowing disturbance⁽⁹⁾. There are extensive interconnections between bilateral major swallowing centers, the NA and nucleus tractus solitarius (NTS) and surrounding reticular formations, that either side coordinates the pharyngeal and esophageal phases of swallowing. Unilateral antegrade electrical stimulation of NTS elicited compound motor action potentials in bilateral

pharyngeal and hyoid muscles in dogs⁽¹⁰⁾. Likewise retrograde horseradish peroxidase injection to the laryngeal muscles labeled neurons in both NA after sectioning of left recurrent laryngeal nerve in rats⁽¹¹⁾. In squirrel monkeys, stimulating laryngeal cortical area while blocking excitatory neurotransmission by injection of the glutamate antagonist kynurenic acid demonstrated that the injection into the dorsal reticular nucleus of the caudal medulla ipsilateral to the stimulation site blocked vocal fold movements bilaterally; injections invading the major parts of the NA blocked vocal fold movements exclusively ipsilateral to the injection site; and injections centered on the parvocellular reticular formation bordering the NA blocked exclusively contralateral vocal fold movements⁽¹²⁾. Corticobulbar laryngeal pathway synapses in the ipsilateral dorsal reticular nucleus, where the two components divide, one runs directly to the ipsilateral NA and the other crosses to the contralateral NA after having synapsed in the ipsilateral peri-ambigular reticular formation (PARF)⁽¹²⁾. The latter predominates probably, as evidenced by the fact that palatolaryngeal paresis is exclusively contralateral in supratentorial stroke⁽⁷⁾.

Apart from the involvement of corticobulbar fibers before synapsing to the ipsilateral caudal medulla, an alternative to our patient is a "pure disconnection dysphagia" by selective involvement of the PARF and/or its post-synaptic connection fiber destined to the contralateral swallowing center⁽¹²⁾. Nevertheless, more similar cases are required to clarify this combination of clinical presentations.

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