CASE REPORT
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A Case of Airway Obstruction Caused by Bilateral Vocal Cord Palsy in a Patient with Lateral Medullary Infarction

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Lateral medullary infarction can lead to central respiratory failure. However, in Wallenberg's syndrome, obstructive respiratory failure rarely develops. Here, we report a case of obstructive respiratory failure with stridor caused by unilateral lateral medullary infarction. A 74-year-old woman was admitted for acute right lateral medullary infarction. On the 12th day after the stroke, the patient developed respiratory failure necessitating endotracheal intubation. She could maintain good oxygen saturation and ventilation in intubated status without the assistance of mechanical ventilator, even during sleep. The pharyngo-laryngoscopy demonstrated the hypomobility of bilateral vocal cords. This case suggests that unilateral medullary infarction might be a cause of bilateral vocal cord palsy. (Korean J Stroke 2012;14:89-91)

KEY WORDS: Vocal cord paralysis, Brain stem infarctions, Medulla oblongata, Respiratory insufficiency

Introduction

Brain stem infarction can lead to various types of respiratory failure.[1-3] Lateral medullary infarction, so called Wallenberg's syndrome, can result in central (autonomic) respiratory failure. However, obstructive respiratory failure rarely develops because a unilateral medullary lesion is commonly associated with unilateral vocal cord palsy which does not cause a respiratory difficulty. We report a case of obstructive respiratory failure with stridor caused by unilateral acute lateral medullary infarction.

Case Report

A 74-year-old woman with a one-year history of hypertension was referred to our hospital with vertigo and unsteadiness during walking. She had no history of diabetes mellitus or other pulmonary disease, although she was currently a 20-pack-year smoker. On admission, the patient showed typical clinical features, compatible with right lateral medullary infarction; dysphagia, dysarthria, hoarseness, anisocoria, right ptosis, right facial hypalgesia, hypalgesia of the left-side of the trunk and extremities, and right limb ataxia. She also reported mild dyspnea with intermittent stridor, although the arterial blood gas analysis (ABGA) was in the normal range. Brain magnetic resonance imaging (MRI) showed a small infarction in the right lateral medulla with a focal stenotic lesion in the right proximal vertebral artery and left distal vertebral artery. Hyperintense signals in the bilateral central semiovale and corona radiata were also detected on T2 weighted image (Figure 1).

On the 12th day after the stroke, respiration failure developed, necessitating endotracheal intubation. The ABGA revealed a respiratory acidosis and CO_2 retention (pH 7.229, pCO_2 94.8 mmHg, pO_2 144.3 mmHg, O_2 saturation 99%). After the patient was placed on a mechanical ventilator, she had no complaint of dyspnea. The chest CT did not reveal any active lung disease, pulmonary thromboembolism, or compressive lesion affecting the respiratory system.

During the intensive care unit stay with intubation, she had no

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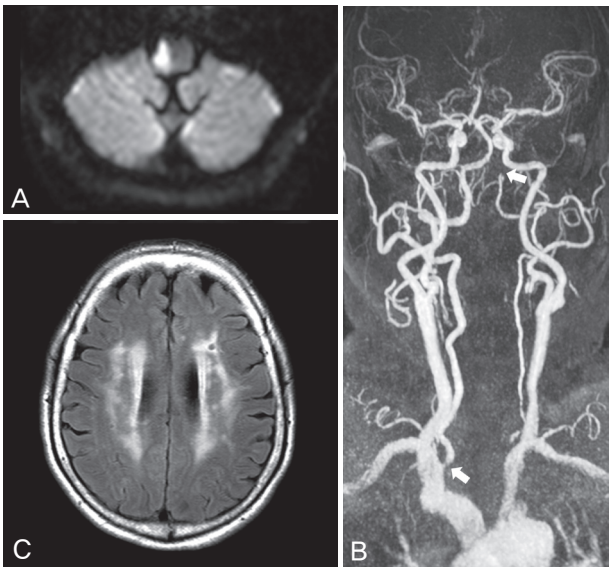

FIGURE 1. The brain MRI shows an acute infarction in the right lateral medulla (A) with bilateral vertebral artery stenosis (arrow) (B). Fluid-attenuated inversion recovery imagingshows old ischemic lesions in the bilateral centrum semiovale (C).

respiratory difficulties. She could maintain good saturation without the assistance of mechanical ventilator, even during sleep. Extubation was tried on the third day after intubation but the endotracheal tube was replaced because her respiratory difficulty re-developed shortly after extubation. Follow-up brain MRI (slice thickness 3 mm, interslice gap 0) did not show any newly developed lesion (Figure 2). The bronchoscopy also failed to detect any potential endobronchial lesions. It was unable to evaluate the vocal cord during bronchoscoping since the saturation dropped immediately after drawing out the endotracheal tube along with scope, necessitating the emergent re-intubation.

On the 12th day after intubation, her endotracheal tube was changed for a tracheostomy. She still had no respiratory difficulties and the ABGA no longer revealed CO_2 retention. On the 33rd day after the stroke, the pharyngo-laryngoscopy demonstrated hypomobility of bilateral vocal cords. After successful training of tube closure, the tracheostomy site was sealed off on the 44th day after the stroke. The ABGA showed a normal pCO_2 (pCO_2 30.3 mmHg). The patient was discharged home five days after the seal-off with mild dyspnea.

The patient visited the clinic one month later because of aggravated dyspnea. She had no newly developed neurologic deficit. Because follow-up pharyngo-laryngoscopy demonstrated the persistent hypomobility of bilateral vocal cords with only 30% patency, she received the laser cordectomy.

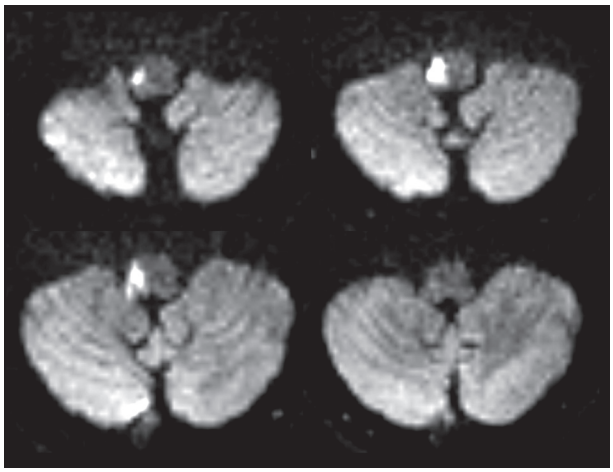

FIGURE 2. Follow-up brain MRI does not show any newly developed lesion.

Discussion

Unilateral medullary infarction can cause an isolated autonomic respiratory failure (Ondine's curse) or combined obstructive and autonomic respiratory failure during sleep.[1-4] However, our patient suffered from isolated obstructive respiratory failure. The patient tolerated being intubated without assistance of mechanical ventilation, even during sleep. Her respiratory failure re-developed immediately after extubation, which suggests her respiratory difficulties might have been caused by airway obstruction rather than decreased central respiratory drive.

In general, unilateral lateral medullary infarction is not associated with the obstructive respiratory failure. The lower motor neuron that originates in the nucleus ambiguus ipsilaterally innervates the intrinsic laryngeal muscles. Unilateral vocal cord palsy does not lead to respiratory failure, although this lesion can cause the hoarseness or dysphonia. Our patient had bilateral vocal cord palsy confirmed by pharyngo-laryngoscopy. Some explanations may be possible. A previous study using squirrel monkeys suggests that the corticobulblar laryngeal control pathway synapses in the ipsilateral dorsal reticular nucleus and is divided into two components. One component runs directly to the ipsilateral nucleus ambiguus, and the other crosses to the contralateral nucleus ambiguus after having synapses in the ipsilateral peri-ambigual reticular formation.[5] The simultaneous involvement of the nucleus ambiguus and peri-ambigual reticular formation in unilateral medullary lesion can cause simultaneous ipsilateral and contralateral vocal cord palsy. Secondly, previous old ischemic lesions at the contralateral hemisphere might

involve bilateral vocal cord palsy. The fibers innervating the vocal cords originate in the precentral gyrus and travel along the corticobulbar tract to enter the brainstem. The fibers cross at the upper portion of the medulla and then innervate the nucleus ambiguus (the majority innervate the ipsilateral nucleus ambiguus) bilaterally. In our patient, the old ischemic lesion in the bilateral centrum semiovale along with the acute medullary lesion might have contributed to her symptoms.

In conclusion, our patient shows that unilateral medullary infarction might be a cause of bilateral vocal cord palsy. It highlights the need for the awareness of the non-central component respiratory failure in the case of lateral medullary infarction.

Conflicts of Interest

The authors have no financial conflicts of interest.

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