

Delayed Central Respiratory Dysfunction After Wallenberg's Syndrome

—Case Report—

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Abstract

A 68-year-old man presented with Wallenberg's syndrome consisting of ataxia, dysphagia, hypesthesia on the left side of the body, and Horner's syndrome on the right. Magnetic resonance (MR) imaging revealed a right lateral medullary infarction and small multiple lacunae scattered in the upper medulla. Neurological symptoms improved in a week and the patient was discharged with mild residual hypesthesia on the left side. However, 31 days later, he was emergently admitted after suddenly becoming apneic and losing consciousness. MR imaging detected no new lesion. The patient was placed under ventilation support for 48 hours before regaining normal respiratory function. Medullary infarction sometimes causes catastrophic respiratory failure, but Wallenberg's syndrome caused by lateral medullary infarction is rarely associated with central respiratory dysfunction, and delayed onset of central respiratory dysfunction is extremely unusual. Delayed onset of central respiratory failure is a life-threatening complication of the medullary infarction causing Wallenberg's syndrome, which in general is not recognized.

Key words: Wallenberg's syndrome, central respiratory dysfunction, brain stem, infarction

Introduction

Brain stem infarction affecting the respiratory center located in the medulla can result in various types of respiratory dysfunction.^{1,3)} However, lateral medullary syndrome also known as Wallenberg's syndrome, is most commonly caused by occlusion of the posterior inferior cerebellar artery, and is rarely associated with central respiratory dysfunction. We treated a patient with Wallenberg's syndrome who developed central respiratory dysfunction unusually late, 38 days, after the onset. Unusual magnetic resonance (MR) imaging findings were important to infer the underlying mechanism of this rare event.

Case Report

A 68-year-old man with a 3-year history of hypertension suddenly experienced dysarthria and numbness on the left side of his body and visited a clinic. Examination revealed Wallenberg's syndrome which included dysphagia, ataxia, left dissociated hemihypesthesia, and Horner's syndrome on the

right. MR imaging revealed a small infarction in the right lower medulla oblongata and multiple small infarctions in the upper medulla (Fig. 1). MR imaging showed no indication of vertebral artery dissection. The neurological symptoms improved over a week, and he was discharged from the hospital with a prescription of low-dose aspirin.

Thirty-one days later, he was admitted emergently to our hospital after sudden apnea followed by loss of consciousness. He did not complain of any chest pain or shortness of breath prior to the event. Blood gas analysis showed moderate hypercapnia: PO₂ 42 mmHg, PCO₂ 69 mmHg, and pH 7.328. Alveolar-arterial gas pressure difference was 21.7 mmHg. Chest radiography demonstrated a mild left lower lobe infiltrate which could not explain the symptom. Respiratory failure continued even after the airway was secured, indicating that central respiratory dysfunction was the underlying mechanism. Therefore, he was intubated and was placed under ventilation support starting with synchronized intermittent mandatory ventilation at 20 times/min. He was weaned off ventilation support over the next 48 hours, and the endotracheal tube was removed 5 days after the admission without further respiratory

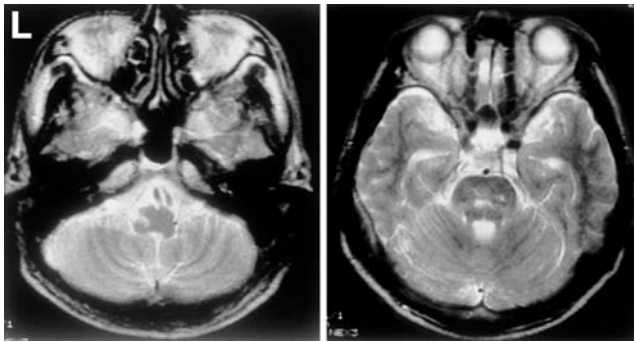


Fig. 1 Magnetic resonance images at the first admission demonstrating a small infarction in the right lower medulla (left) and multiple small infarctions in the upper medulla (right).

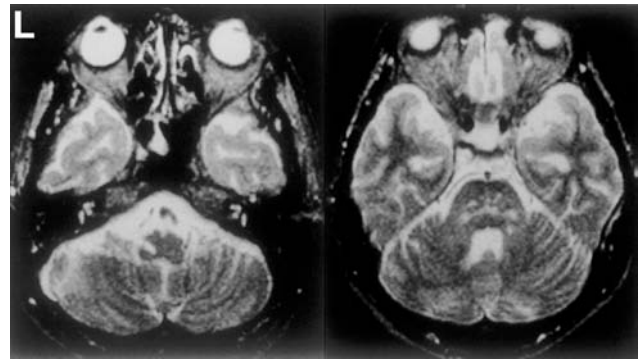


Fig. 2 Magnetic resonance images at the second admission revealing no significant changes compared to the findings at the first admission.

problems. His respiration remained stable thereafter. MR imaging obtained 6 days after the onset of the respiratory failure demonstrated no new lesion compared to the first MR imaging (Fig. 2). Cerebral angiography performed on the second day detected no signs of stenosis, occlusion, or dissection of major vessels including the vertebral and basilar arteries (Fig. 3). Follow-up examination 4 months later found he was in good condition, with no new episodes of respiratory distress.

Discussion

Three main groups of neurons located in the brain stem are involved in the regulation of respiratory function: the nucleus solitarius (dorsal respiratory group), the nucleus ambiguus (ventral respiratory group), and the nucleus parabrachialis medialis.⁶⁾ Ischemic damage to these neurons could lead to central respiratory dysfunction, but inconsistencies between the radiological findings and the symptoms suggest that complex networks between the neurons could compensate for the damage to some extent.⁵⁾ For instance, bilateral medial medullary infarction involving the nucleus parabrachialis medialis does not always cause respiratory failure.^{2,8)} Unilateral lateral medullary infarction is one of the smallest lesions known to cause central respiratory dysfunction,^{1,7)} and also causes Wallenberg's syndrome. However, previous cases of central respiratory dysfunction associated with Wallenberg's syndrome occurred in the acute stage. The delayed onset at 38 days following the first ischemic attack in our case was extremely unusual. MR imaging obtained after the initial event visualized an infarct in the right lower medulla oblongata and scattered multiple

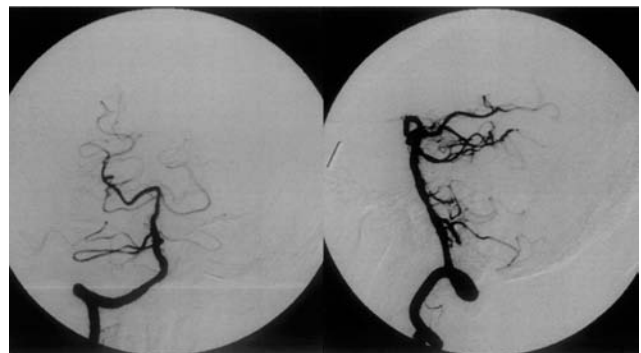


Fig. 3 Right vertebral angiograms at the second admission demonstrating no abnormalities such as occlusion or dissection of the vertebral and basilar arteries.

high-intensity spots in the bilateral upper medulla. These findings were unchanged after the second event, indicating that new infarction was unlikely to be the cause of the respiratory failure. Furthermore, angiography did not show occlusion or dissection of the vertebral or basilar artery, which is a common cause of small multiple infarctions leading to step-wise aggravation of neurological deficits.

Based on the absence of evidence for vertebral artery occlusion on angiography or MR imaging, we presumed that microembolism from an unknown source was the most likely cause of the initial event leading to Wallenberg's syndrome. We cannot exclude the possibility that another such event had caused the respiratory dysfunction as well, but MR imaging did not show any new lesions following respiratory failure. Pulmonary lesion was probably not the direct cause either, because the alveolar-arterial gas pressure difference (21.7 mmHg) was

within the normal limits for the age of this patient⁴⁾ and strongly indicated that the hypercapnia was caused by central alveolar hypoventilation.

The possibility of the late developing respiratory disturbance after brain stem infarction is important to recognize, because this can be fatal if not treated appropriately and immediately. Careful instruction of patients and carers on the possibility and measures to assist respiration is recommended to prevent any serious outcome caused by such a rare complication of medullary infarction manifesting as Wallenberg's syndrome.

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