

**LETTERS TO THE EDITOR****TO THE EDITOR****Isolated Dysphagia after a Small Posterolateral Medullary Infarct: A Case Report**

Dysphagia is a common complication of cerebral infarction and is associated with aspiration pneumonia. According to previous studies, the frequency of dysphagia in patients with lateral medullary infarction (LMI) and medial medullary infarction (MMI) is 35% to 69%<sup>1,2</sup> and 11% to 78%<sup>1-3</sup>, respectively. Nevertheless, dysphagia as the only presentation of medullary infarction has rarely been reported. We report a case of isolated severe dysphagia after a small posterolateral medullary infarct.

**CASE PRESENTATION**

A 67-year-old, right-handed, African American female presented to the emergency department with the chief complaint of sudden onset swallowing difficulty. The night before presentation, the patient was awakened shortly after midnight by a choking sensation and realized that she was unable to swallow her saliva. On the way to the bathroom she also noticed a vertigo sensation and tendency to fall towards the left. The episode lasted about five minutes after which she regained her ability to swallow and noticed improvement in her dizziness. The patient went back to her bed and was awakened again by similar choking sensation after two hours. This time, as the symptom persisted, she decided to come to the hospital. Her past medical history was significant for high blood pressure and borderline diabetes. She had a history of gastroplasty several years ago. Her home medication consisted of Amlodipine, Benazepril, and Hydrochlorothiazide. She had a remote ten pack-year history of tobacco use.

The review of system was negative for coughing, chest pain, halitosis, regurgitation, odynophagia, hoarseness, dysarthria, and adenopathy or swelling in neck. She also denied any myalgia, visual or hearing disturbances, facial weakness, motor weakness, and anesthesia. She denied any dizziness, vertigo, and incoordination, however, after further questioning she disclosed that she had an abrupt episode of vertigo one day prior to her presentation which resolved gradually after a few hours. The review of system was also negative for mood lability, hallucinations, and delusions. She otherwise reported inability to swallow followed by choking sensation, and chronic intermittent heartburn.

The examination revealed a slightly overweight woman, calm, with slightly elevated blood pressure. No lymphadenopathy, masses, or thyromegaly were noted. Trachea was midline. Carotid pulses were present with no bruits. Buccal mucosa was moist and intact; however, she was unable to manage oral secretions. Tonsils were present. Chest expansion was symmetric and clear to auscultation. Heart was regular rate and rhythm with no adventitious sounds. Abdomen was soft, non-tender, without masses. Bowel sounds were present. In neurological examination, the language was appropriate and there was no dysphonia or dysarthria. The pupils were equal, round, and reactive to light. Extraocular movement was intact

with no diplopia or nystagmus. There was no ptosis, facial anesthesia, or weakness. The Weber test was negative for lateralization. The tongue protrusion was midline and palate elevation was symmetric. There was no tongue atrophy or fasciculation. The exam however showed mildly suppressed pharyngeal sensation and absent gag reflex bilaterally. The rest of our detailed neurological examination otherwise showed no focal neurologic deficits.

The computed tomogram (CT) scan of the head without contrast showed chronic white matter disease with no acute findings. A modified barium swallow (MBS) study was done which indicated a mechanical breakdown of esophageal phase of swallow with poor opening of upper esophageal sphincter (UES) (Figure 1). Nevertheless there was neither hypolaryngeal excursion nor aspiration. Following the results of MBS a contrast CT scan of the neck was performed which showed no obstructive or mass lesion consistent with the findings on MBS study. A magnetic resonance (MR) study of the brain was completed which showed a very small focus of restricted diffusion in the left posterolateral medulla at the junction of pons and medulla adjacent to the left anterior-inferior portion of the 4th ventricle (Figure 2). Magnetic resonance angiography of the head and



*Figure 1: Modified Barium Swallowing Study*

neck revealed mild intracranial atherosclerotic disease but it was negative for any significant stenosis. Both posterior inferior cerebellar arteries (PICA) were present.

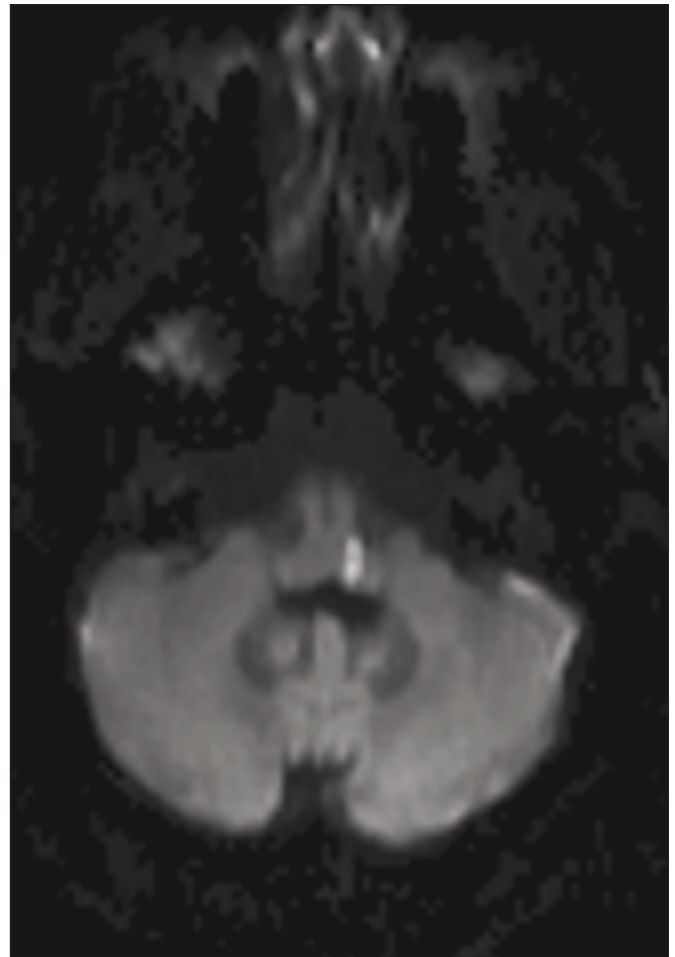
The repeat gag reflex examination, after five days, showed that the patient regained her gag bilaterally but it was decreased on the left side. The second MBS study showed no improvement in the pharyngoesophageal phase of swallowing. Our patient hence underwent percutaneous endoscopic gastrostomy (PEG) tube placement and was later discharged home.

## DISCUSSION

The etiology of dysphagia in the medullary infarction can generally be explained by the involvement of the nucleus ambiguus which innervates upper alimentary tracts as well as various thoracic and abdominal viscera. Previous studies indicate that dysphonia and particularly dysarthria are related to dysphagia due to the involvement of the nucleus ambiguus.<sup>1</sup> Several observations and animal experiments indicate that rostral lesions to the nucleus ambiguus are associated with increased frequency and severity of dysphagia.<sup>4,5</sup> Yet, the characteristics of dysphasia are different between LMI and MMI. The patients with LMI usually have problems on range of hypolaryngeal excursion; however, among MMI patients, the most observed phenomenon is problems on timing of hypolaryngeal excursion.<sup>1</sup> That observation indicates that dysphagia in MMI may be due to involvement of corticobulbar tract innervating the nucleus ambiguus rather than damage to this structure.<sup>1</sup>

On the other hand, our patient surprisingly did not have any problems on hypolaryngeal excursion. The MBS results showed that dysphagia was mainly due to insufficient opening of UES. In addition, she had neither dysarthria nor dysphonia. That can be explained according to animal experiments<sup>5</sup> demonstrating that the nucleus ambiguus in the rat consists of a dorsal division representing the special visceral efferent component, and a ventral division representing the general visceral efferent component. The dorsal division further comprises a rostral esophagomotor compact formation, an intermediate pharyngolaryngomotor semicompact formation, and a caudal laryngomotor loose formation. Therefore phonatory muscles are innervated by intermediate and caudal medulla and can stay intact in a medullary infarction presenting with dysphagia. Considering the organization and synaptology of the nucleus ambiguus, we can conclude that our patient suffered from a small lacunar infarction involving a part of rostral esophagomotor compact formation of the nucleus ambiguus. This conclusion is also consistent with the finding on diffusion-weighted images. The reason for early absence of bilateral gag reflex remained unclear.

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**Figure 2:** Diffusion Weighted Image

## REFERENCES

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