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A Typical Lateral Medullary Syndrome

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1. Abstract

Lateral medullary syndrome (LMS) is a type of ischemic stroke which occurs due to disruption of the blood flow in vertebral artery, or posterior inferior cerebellar artery. LMS can present with various sign and symptoms, depending on the site of infarct at the medullary area. Typical LMS often affects the pain and temperature sensation over the contralateral extremities and ipsilateral face of the infarct area. We illustrate a case of LMS with predominant bulbar symptoms which is sparse the sensation and our treatment experience.

2. Introduction

Lateral medullary syndrome (LMS) or Wallenberg's syndrome is an unusual type of stroke and it typically caused by occlusion of the intracranial segment of the vertebral artery. Rarely, LMS can also occurs in posterior inferior cerebellar artery (PICA) or medullary artery thrombosis. There are variable presentations of LMS, depending on the area of infarct that involves the lateral medullary area. LMS typically characterized by sensory deficit (mainly pain and temperature) affecting the limbs on the contralateral side of infarction and face on the ipsilateral side of infarction. LMS may also present with vertigo, nystagmus, ataxia, bulbar symptoms such as dysarthria and dysphagia, and Horner syndrome. We report a case of atypical LMS presentation with predominantly bulbar symptom and vertigo, without the typical sensory deficit.

3. Case Presentation

A 69-year-old gentleman, without any known medical illness, presented with two days of dizziness, sudden onset of hoarseness of voice and dysphagia. He was unable to stand by himself due to unsteadiness. Otherwise, there was no diplopia, no fever, no headache, no chest pain and breathlessness. He was a chronic smoker (30-packs-year). On arrival to emergency department, he was alert and conscious with stable vital signs. Neurological examination showed he had nasal speech with his uvula deviated to the left and there was absent of gag reflex. Besides, he also had truncal ataxia and unable to perform a tandem-gait. Otherwise, there was no limb weakness, sensation was intact bilaterally over his face and extremities, without any other cerebellar sign and cranial nerves involvement. All deep tendon reflexes and plantar reflexes were normal.

Electrocardiogram showed sinus rhythm. Initial head computed tomography (CT) showed no obvious acute infarct or haemorrhage. A magnetic resonance imaging (MRI) brain was done the next day. It showed hypo intense signal on T1-weighted imaging, hyper intense signal on T2-weighted/ fluid-attenuated inversion recovery (FLAIR) imaging at right medulla oblongata with restricted diffusion on diffusion-weighted imaging (DWI), which were suggestive of acute infarction. Moreover, magnetic resonance angiography (MRA) brain showed loss of normal signal at V4 segment of right vertebral artery, likely suggestive of thrombosis. A nasogastric tube was inserted to initiate enteral feeding. Single antiplatelet and statin were started and patient was referred for stroke rehabilitation. Patient's vertigo improved after 2 weeks of stroke rehabilitation and was discharged with nasogastric tube. He was regularly follow up with speech therapist as well as rehabilitation and eventually he managed to tolerate oral feeding after 2 months of stroke event (Figure 1A & 1B, 2A & 2B, 3).

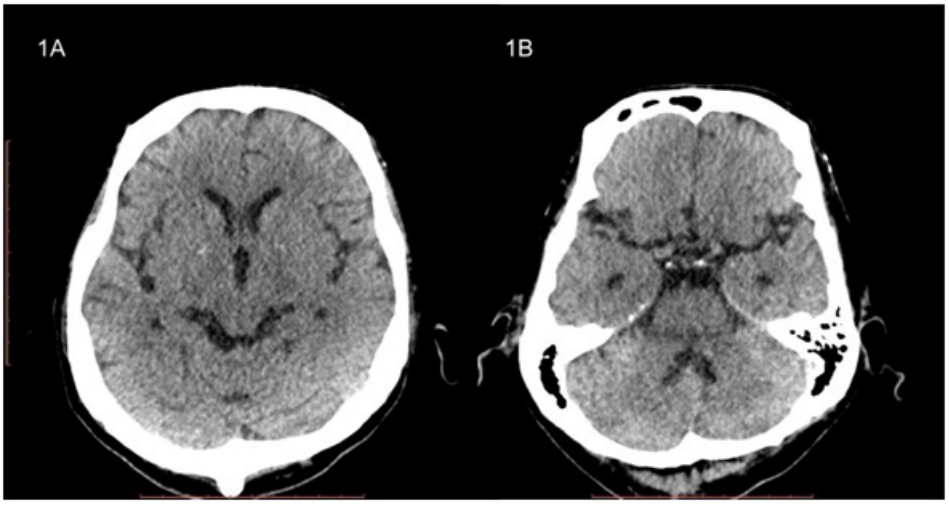

Figure 1A & 1B: Initial head computed tomography (CT) did not show any acute infarct or haemorrhage

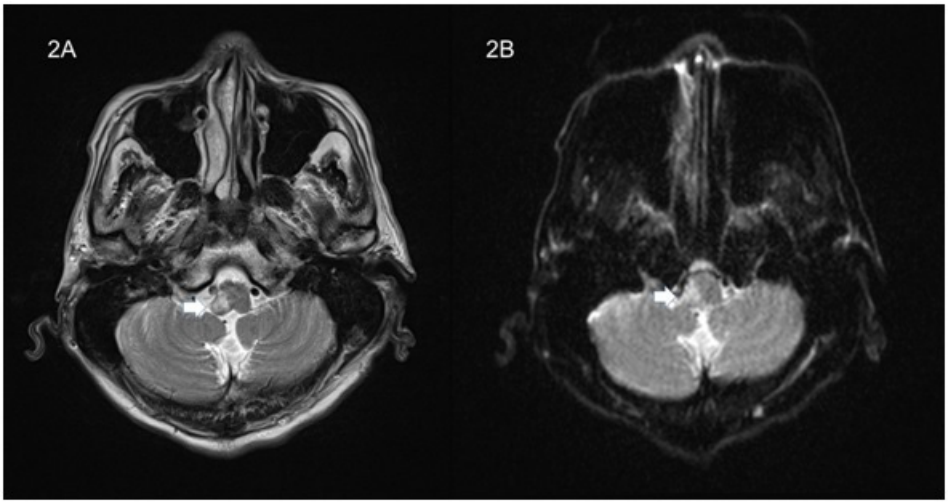

Figure 2A: Hyperintensity seen (arrow) during T2W/ fluid-attenuated inversion recovery (FLAIR) at right lateral medulla oblongata.
Figure 2B: Restricted diffusion seen (arrow) during diffusion-weighted imaging (DWI) at right lateral medulla oblongata, indicates acute infarct.

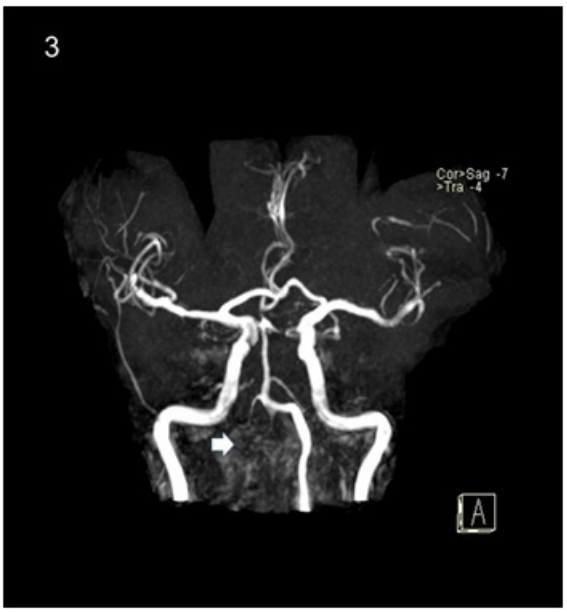

Figure 3: Filling defect over right vertebral artery (arrow) indicates right vertebral artery thrombosis.

4. Discussion

LMS typically presented with loss of pain and temperature sensation over the ipsilateral side of the face and contralateral side of the body, due to the insult involve at spinothalamic tract. Other clinical presentation includes dysphagia and dysarthria, which is due to involvement of nucleus ambigus; nystagmus and vertigo due to involvement of vestibular nuclei; Horner's syndrome due to involvement of the sympathetic nervous system; and ataxia due to damage of the inferior cerebellar peduncle. In our case report, the patient's main symptoms were dysphagia and nasal speech, associated with vertigo and ataxia, without any limb weakness and sensory deficit, which is not the typical presentation of LMS as dysphagia is not the main symptom at onset of LMS [1].

Deglutination is controlled by a group of central pattern generator (CPG), which is located at the nucleus tractus solitarius adjacent to the medullary reticular formation. In order to perform a deglutination process, the CPG will trigger the nucleus ambigus and vagal dorsal motor nucleus, which are then coordinate and innervate the muscles around the distal pharynx, upper esophageal sphincter and proximal esophagus [2]. The severity of dysphagia is worse and this disability can last longer in LMS compared to hemispheric stroke patients, except in those with multiple cerebral infarct [3]. In a case report written by Vigderman, et, al. (1998) [4], they suggest that bilateral swallowing centers at the brainstem function as an integrated center; and a complete loss of swallowing may occur even if there is only unilateral lesion occur at the swallowing center[4]. There are three phases in deglutination, namely oral phase, pharyngeal phase and esophageal phase. In LMS, the main abnormality was found during pharyngeal phase of the deglutination, as well as absence of the upper esophageal sphincter and proximal esophagus contraction during esophageal phase, which result for swallowing difficulty [5]. As for patient with hemispheric stroke, the swallowing difficulty happens during oral phase [5].

The prognosis of LMS is merely depends on the etiology and well-controlled of the risk factors. Treatment for LMS is mainly symptomatically approach. Most of the dysphagia occur in LMS will eventually recover spontaneously within 1 to 2 months after the stroke event[5]. As for our patient, apart from single antiplatelet and statin for secondary stroke prevention, antiemetic and anti-vertigo medications were prescribed for his vertigo. He was also discharged with nasopharyngeal tube for feeding purpose and was reviewed by speech therapist regularly. He manages to tolerate oral feeding 2 months after his stroke event.

5. Conclusion

LMS can present with dysphagia, vertigo and ataxia as main symptom without sensory deficit. Brainstem infarct should always be considered as a differential diagnosis for patient presented with acute bulbar symptom and vertigo. MRI is a superior imaging tool to diagnose LMS compared to CT. Dysphagia in LMS may recover in 1-2 months after the stroke and initial feeding often requires the aid of nasopharyngeal tube.

6. Consent

The authors certify that they have obtained all appropriate patient's consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

7. Conflict of Interest

The authors declared that they have no conflict of interests regarding publication of this article.

8. Ethical Approval

Ethical approval is not required at our institution to publish an anonymous case report.

9. Acknowledgement

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