

Case Report

Partial Wallenberg Syndrome with Contralateral Bulbar Palsy

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Abstract

Lateral medullary syndrome, also called Wallenberg syndrome, is a brainstem stroke with features like ipsilateral Horner's syndrome, dysphagia, crossed sensory deficits, vertigo, and ipsilateral ataxia. Incomplete or atypical variants of lateral medullary syndrome may present with selective or crossed neurological deficits, delaying diagnosis.

We report a case of a 52-year-old male presenting with dysphagia and right hemisensory impairment. Neurological examination revealed left partial ptosis and miosis, along with an absent gag reflex on the right side. MRI demonstrated an acute infarct at the left lateral pontomedullary junction, consistent with the atypical presentation of lateral medullary syndrome. Treatment with anti-platelet therapy, statins, and swallowing rehabilitation led to significant clinical improvement.

This case illustrates the variability in presentations of lateral medullary syndrome and underscores the need to correlate focal neurological findings with neuroimaging for early recognition of atypical medullary infarcts.

Keywords: Lateral medullary syndrome; Wallenberg syndrome; Dysphagia; Horner syndrome; Pontomedullary infarction

Abbreviations

LMS: Lateral Medullary Syndrome; PICA: Posterior Inferior Cerebellar Artery; MRI: Magnetic Resonance Imaging; FLAIR: Fluid-Attenuated Inversion Recovery; DWI: Diffusion-Weighted Imaging; ADC: Apparent Diffusion Coefficient; LMN: Lower Motor Neuron; UMN: Upper Motor Neuron; PARF: Parvicellular Reticular Formation

Introduction

Lateral medullary syndrome commonly involves the posterior inferior cerebellar artery territory, with classic presentations such as dysphagia, ipsilateral Horner's syndrome, ipsilateral facial numbness, crossed sensory deficits, vertigo, and ataxia. It usually results from atherothrombotic disease of the vertebral artery or Posterior Inferior Cerebellar Artery (PICA). Dysphagia occurs in 51% to 94% of LMS cases and often predominates [1,2]. Isolated or predominant dysphagia can pose a diagnostic challenge because patients may lack the full constellation of neurological signs [3]. Incomplete or atypical LMS variants, with partial or crossed cranial nerve deficits, are uncommon. Pediatric and trauma-related LMS strokes are rare. This limited literature shows the clinical variability of LMS and the importance of recognizing isolated symptoms like dysphagia for timely diagnosis and management.

Case Presentation

A 52-year-old man presented to our hospital with a history of dysphagia for four days. The dysphagia was sudden in onset, predominantly affected liquids, and was not associated with pain. The patient also reported nasal regurgitation of food. He described numbness and tingling on the right side of the body for 3 days, with numbness on the right side of the chest as well. He reported no limb weakness, visual changes, diplopia, hearing loss, vertigo, loss of consciousness, or bowel or bladder involvement. He had a 9-year history of hypertension treated with telmisartan and an 8-year history of diabetes mellitus treated with oral hypoglycemic agents. He had a 15-year history of alcohol use disorder; his family reported a recent binge episode 20 days before admission. There is no significant family history.

On examination, the pulse was 94 beats per minute, and the blood pressure was 180/100 mmHg. Other system examinations were unremarkable. He was conscious, alert, and oriented. Cranial nerve examination showed left ptosis and miosis (Figure 1), with normal extraocular movements. Examination of cranial nerves IX and X showed deviation of the uvula to the left, reduced right palatal movement, and a poor gag reflex on the right side (Figure 2). Motor exam revealed no abnormality. Sensory exam showed reduced touch and cold sensation in the right lower limb. The rest of the neurological examination was unremarkable.

Magnetic Resonance Imaging (MRI) of the brain revealed a wedge-shaped lesion in the lateral aspect of the upper medulla at the junction of the medulla and pons on the left side, appearing T1 isointense, T2- and Fluid-Attenuated Inversion Recovery (FLAIR)-hyperintense (Figure 3), with diffusion restriction on Diffusion-Weighted Imaging (DWI) and corresponding low Apparent Diffusion Coefficient (ADC), and no contrast enhancement. MR angiography was normal. Laboratory investigations (Table 1) showed polycythemia, with a hemoglobin level of 18 g/dL. The remaining laboratory investigations were within normal limits. The patient was treated with antiplatelets,

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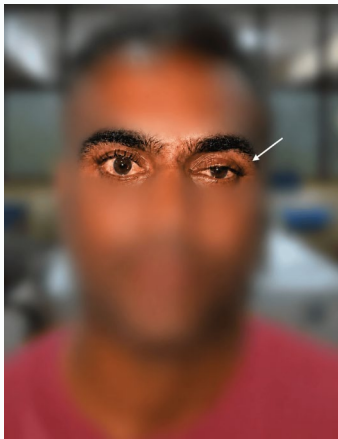


Figure 1: Left partial ptosis and miosis (arrow).



Figure 2: Gag reflex examination showing absent response on the right side (a) and preserved response on the left side (b).

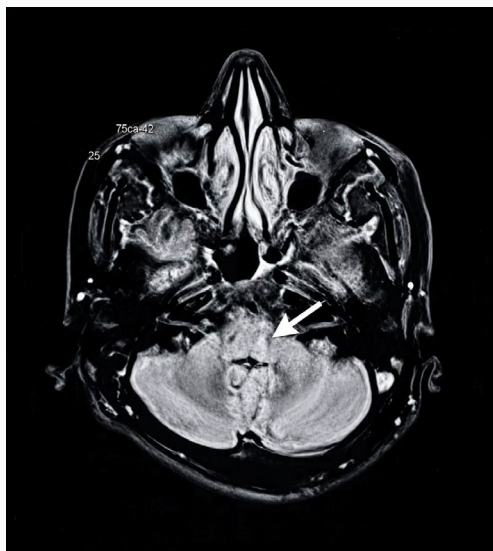


Figure 3: Magnetic resonance imaging of the brain showing the left lateral pontomedullary infarct (arrow).

statins, and other supportive care. Speech and swallow therapy was initiated, and the patient recovered significantly over 10 days.

Discussions

Lateral Medullary Syndrome (LMS), or Wallenberg syndrome, is the most common brainstem stroke syndrome and typically results from occlusion of the intracranial vertebral artery or posterior inferior cerebellar artery. The classical clinical constellation includes ipsilateral facial sensory loss, contralateral loss of pain and temperature over the body, ipsilateral Horner syndrome, cerebellar ataxia, vertigo, nystagmus, dysphagia, dysarthria, and palatal weakness. However, the syndrome demonstrates considerable anatomical and clinical heterogeneity depending on the rostro caudal level and extent of the infarction.

The present case demonstrates an atypical rostral lateral medullary infarction with predominant bulbar dysfunction and an unusual pattern of cranial nerve involvement. In addition to dysphagia at onset, ipsilateral Horner's syndrome, ipsilateral palatal weakness, and contralateral hemisensory impairment, the patient exhibited a rare and well-documented contralateral impairment of glossopharyngeal and vagal (IX-X) function, manifesting as a poor gag reflex on the contralateral side.

Chakraborty, et al. [4] described several clinical variants and incomplete forms of LMS in the literature (Table 2), reflecting heterogeneity in the infarct's extent and location. For instance, the Opalski syndrome variant includes ipsilateral hemiparesis due to caudal extension of the infarct involving corticospinal fibres after pyramidal decussation. In contrast, Babinski-Nageotte syndrome is characterized by hemiparesis associated with involvement of corticospinal fibres and can occur as an incomplete lateral medullary syndrome variant [4-7]. Furthermore, atypical presentations such as partial or incomplete Horner's syndrome and crossed cranial nerve findings can pose diagnostic challenges [8,9]. These variations highlight the complex neuroanatomical organization of the lateral medulla and the diversity of vascular territories involved. Understanding incomplete or crossed patterns is essential for accurate clinico-radiological correlation and timely management.

Atypical findings in the current case can be explained by involvement of contralateral corticobulbar pathways, swallowing, and the gag reflex, although traditionally considered to have predominantly ipsilateral control, anatomical evidence suggests that they involve bilateral and partially decussating influences, and selective damage to specific functional subregions may therefore produce atypical or crossed cranial nerve findings. Another mechanism is variability in the vascular supply of the medulla, primarily through the posterior inferior cerebellar artery and vertebral artery, which can result in highly focal ischemia.

Recognition of atypical or incomplete presentations depends on correlating clinical features with MRI findings. Rostral lesions are more likely to produce bulbar symptoms (dysphagia, dysarthria). Caudal lesions produce more vestibular dysfunction and cerebellar signs. Crossing or bilateral involvement of neural pathways may produce complex infarct patterns and atypical neurological findings [7,9].

Conclusion

Atypical or incomplete lateral medullary infarcts present diagnostic challenges due to selective symptomatology. Effective management depends on precise clinico-radiological localization

Table 1: Laboratory investigations.

Parameter	Patient value	Reference range (adult male)	Interpretation
Haemoglobin (Hb)	18 g/dL	13.5-17.5 g/dL	Elevated
Red Blood Cell (RBC) Count	$5.4 \times 10^6/\mu\text{L}$	$4.5-5.9 \times 10^6/\mu\text{L}$	Normal
White Blood Cell (WBC) Count	$4.5 \times 10^3/\mu\text{L}$	$4.0-11.0 \times 10^3/\mu\text{L}$	Normal
Blood Sugar (Fasting)	87 mg/dL	70-99 mg/dL	Normal
Total Cholesterol	178 mg/dL	<200 mg/dL	Normal
LDL Cholesterol	67 mg/dL	<100 mg/dL	Normal
HDL Cholesterol	42 mg/dL	>40 mg/dL	Normal
Triglycerides	132 mg/dL	<150 mg/dL	Normal

Table 2: Reported clinical variants of lateral medullary syndrome.

Study	Year	Clinical features	Structures affected	Vessel involved	Comments
Renjen, et al. [1]	2021	Ipsilateral partial Horner's, ipsilateral lateral rectus palsy, ipsilateral LMN facial palsy, contralateral hemianesthesia	Left facial colliculus + left lateral dorsal medulla infarct	Normal	Recovered over 2-3 weeks
Khyati, et al. [2]	2022	Ipsilateral hemi cranial headache, dizziness, ipsilateral face numbness, dysphagia, ipsilateral eye ptosis	Right posterolateral medulla	Right vertebral stenosis	LMS with headache
Chen, et al. [3]	2008	Contralateral ataxia + anaesthesia and palatal palsy	Left dorsolateral medullary infarct	Left vertebral artery occlusion	Involvement of corticobulbar fibres or PARF proposed
Chakraborty, et al. [4]	2019	Ipsilateral ataxia, ipsilateral UMN facial palsy, contralateral hemiparesis, partial Horner's	Left dorsal medulla	Normal	Babinski-Nageotte syndrome with UMN facial palsy
Mahale, et al. [5]	2015	Vertigo + ipsilateral hemiataxia + ipsilateral face and body hypoesthesia	Left dorsolateral medulla infarct with caudal extension	Left vertebral artery not visualised	Ipsilateral thalamic pattern sensory involvement noted

and understanding of anatomical variants. Awareness of crossed or selective cranial nerve findings, particularly isolated dysphagia without other brainstem signs, should prompt consideration of focal medullary infarction. Early diagnosis facilitates timely therapy, reduces the risk of aspiration, and improves neurological recovery.

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