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Lateral Medullary Syndrome from brainstem infarction: A case report

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ABSTRACT

Background: Lateral medullary syndrome (LMS) is a rare posterior circulation stroke caused by thrombosis or dissection of the vertebral or posterior inferior cerebellar artery (PICA). It results in neurological deficits such as ipsilateral facial pain and temperature deficits, contralateral body pain and temperature deficits, ipsilateral ataxia, vertigo, nystagmus, slurred speech, dysphagia, and hiccups.

Case Reports: We report a case of 50-year-old male with uncontrolled hypertension presented with sudden severe vertigo, ataxia, swallowing difficulties, slurred speech, and sensory deficits. He had right-sided facial pain and left-sided body pain.

Conclusions: Neurological examination suggested LMS, confirmed by radiological imaging.

Keywords: brainstem infarction, lateral medullary syndrome, Wallenberg syndrome, PICA

Abbrevations: LMS (lateral medullary syndrome), PICA (posterior inferior cerebellar artery)

INTRODUCTION

Wallenberg's syndrome develops when the posterior inferior cerebellar artery (PICA) or the vertebral artery induce injury to the lateral region of the medulla. The neurological disorders in Wallenberg's syndrome is evolve from the damage of lateral segment of the medulla posterior to the inferior olivary nucleus. In another case, lateral medullary syndrome can be also caused by the atherothrombotic occlusion of superior, basilar, and middle cerebellar artery. In rare case, Wallenberg's syndrome may associate to infections for example base osteomyelitis or tropical neurocysticercosis, demyelenation in multiple sclerosis, and autoimmune disease such as Sjogren's syndrome. The most typical LMS symptom is a sensory deficit in pain and temperature on the ipsilateral side of the face and the contralateral side of the remainder of the body. Dysphagia, nausea, diplopia, ipsilateral headache, hiccups, opposite cerebellar signs, Horner's syndrome, ipsilateral palatal and vocal cord weakness, dysarthria, ataxia, nystagmus, and vertigo are among the additional symptoms and indicators [1,2].

Lateral medullary syndrome (LMS) also known as Wallenberg's syndrome or posterior inferior cerebellar artery (PICA) syndrome was first described in 1808 by Gaspard Vieusseux and the description more detailed by Adolf Wallenberg in 1895. Wallenberg's syndrome predominantly affects men in their 50s or sixth decade. Large artery atherothrombosis is responsible for approximately 75% of cases, while cardioembolism accounts for 17% and vertebral dissection for 8% [1-3].

The homolateral intracranial vertebral artery or the posterior inferior cerebellar artery may become occluded by arteriosclerotic-thrombotic processes, leading to lateral medullary syndrome, also known as Wallenberg syndrome. It was uncommon and frequently misdiagnosed so the features are very important to know and understand.[3] We report a case of lateral medullary syndrome from dorsolateral medullary region infarction in 50 years old male accompanied by clinical and radiological findings.

CASE REPORT

A 50-year-old male with a history of uncontrolled hypertension presented to the emergency room more than 24 hours after the onset of his symptoms. He had been referred from another hospital, where he experienced persistent vertigo and ataxia

that did not improve with betahistine and flunarizine. Upon arrival, his symptoms included a sudden onset of right facial sensory deficit, numbness, left-sided body numbness, vertigo, and slurred speech. On a numeric pain scale, he rated his discomfort as 7-8 out of 10, with the most severe pain localized to the right occipital region. He reported feeling dizzy and noted a tendency to fall to the right when attempting to get out of bed. Despite these significant symptoms, he denied experiencing any weakness in any part of his body. Although he had difficulty speaking and his speech was slurred, he was able to understand spoken words and sentences without any issues.

A comprehensive neurological examination revealed that the patient was fully conscious, though he presented with very high blood pressure (180/110 mmHg) despite being on a 5 mg daily dose of amlodipine treatment. His pulse and respiratory rate were within normal limits. The examination also noted altered sensorium, numbness, and tingling in the right side of his face and the left side of his body. His muscle strength was assessed at 5/5 in both sides, indicating normal muscle tone and strength across all four limbs. Both abdominal and bilateral plantar reflexes were intact, and deep tendon reflexes were graded at 2+ without clonus bilaterally. Aside from his uncontrolled high blood pressure, his personal and family medical history were unremarkable.

Further examination of the cranial nerves revealed slight right-sided ptosis, mouth deviation, and sensory loss on the right side of the face. Laboratory analysis was largely normal, except for elevated low-density lipoprotein (LDL) levels at 156 mg/dL. A brain CT scan as in (Figure 1) revealed a right medullary infarct consistent with lateral medullary syndrome (LMS). Additional diagnostic tests, including an electrocardiogram and an echocardiography, yielded normal results. However, a Brain Transcranial Doppler as in (Figure 2) showed severe stenosis in the basilar artery. Given these findings, the patient was admitted to the neurology unit.

Upon admission, the patient received a loading dose of clopidogrel (300 mg) to manage the risk of further thrombotic events. He was also prescribed aspirin (100 mg), atorvastatin (40 mg), gabapentin (100 mg) for pain management, and folic acid. His antihypertensive medication regimen, including amlodipine, was continued and closely monitored. During his eight-day hospital stay, the patient received comprehensive medical treatment, which resulted in significant improvement in his condition.

Upon discharge, the patient was scheduled to receive physiotherapy to support his recovery and help him regain his balance and coordination. Six months later, follow-up evaluations indicated that he continued with antiplatelet treatment, including aspirin and clopidogrel, as well as antihypertensive medications, specifically amlodipine and candesartan. Gabapentin was also continued for managing post-stroke neuropathic pain. His modified Rankin scale score was 1, indicating that he was largely independent and could perform daily activities with minimal assistance. However, he continued to experience residual symptoms, including tingling and numbness on the left side of his body and the right side of his face.

This case highlights the complexity and severity of lateral medullary syndrome, particularly in patients with uncontrolled hypertension. The timely and comprehensive management, including antiplatelet therapy, statins, and physiotherapy, played a crucial role in the patient's recovery. Continued follow-up and management of his hypertension and cholesterol levels are essential to prevent recurrence and ensure long-term health. Despite the significant neurological deficits at presentation, the patient's improvement over six months demonstrates the potential for recovery with appropriate medical intervention and rehabilitation.

DISCUSSION

We report a case of a 50-year-old man who visited the emergency room complaining of vertigo, slurred speech, hiccups, and a sensory abnormality on the left side of his face. He had a history of uncontrolled hypertension. The patient had been experiencing these symptoms for more than 24 hours before being referred to our hospital. He was diagnosed with a brainstem stroke in the less common form known as lateral medullary syndrome (LMS). LMS, also known as Wallenberg's syndrome, is brought on by the of the posterior inferior cerebellar artery's (PICA) blockage. The blockage can result from thrombosis, atherosclerosis, or emboli originating from another source. Additional causes of this syndrome include hemorrhage, cavernous angioma, malignancies, and vertebral artery dissection [4,5].

Patients with LMS often present with crossed hemisensory or motor impairments, such as ipsilateral facial symptoms and contralateral body symptoms. These symptoms are often accompanied by vertigo, ipsilateral Horner syndrome,

nausea, vomiting, ataxia, dysphagia, hiccups, and dysarthria or slurred speech. Other, less frequent neurological symptoms include nystagmus, diplopia, and possibly palatal myoclonus. Because this condition can resemble other types of brainstem strokes, early detection is especially crucial. In our case, the patient was diagnosed late because of the uncommon presentation of stroke symptoms and signs [6,7].

For a clinical diagnosis of Wallenberg's syndrome, the standard symptoms and signs, including crossed hemisensory deficits and cranial nerve involvement, are often sufficient. Other conditions that might present similarly include Millard-Gubler syndrome, which manifests as ipsilateral 6th and 7th nerve palsy along with contralateral hemiplegia due to a lesion in the ventral caudal pons. Avellis syndrome, which involves Horner's syndrome, cranial nerve 10 involvement, a tegmentum of the medulla lesion, and spinothalamic tract involvement, is another potential differential diagnosis. Additionally, Tapia syndrome, which causes unilateral paralysis of the tongue and vocal cords due to simultaneous extracranial paralysis of the recurrent laryngeal branch of the vagus nerve and the hypoglossal nerve, should be considered [8].

The most frequent cause of Wallenberg's syndrome is atherothrombotic blockage of the vertebral artery.[8] The primary risk factors for Wallenberg's syndrome in adults include hypertension, diabetes, and coronary artery disease, all of which contribute to atherosclerosis. The symptoms that typically bother patients the most include slurred speech, problems swallowing, numbness or pain, and vertigo. Although dysphagia initially necessitates nasogastric tube feeding support, patients subsequently demonstrate remarkable progress and can resume oral feeding within 1-2 months following the stroke [9,10].

Wallenberg's syndrome, also known as lateral medullary syndrome, involves injury to the vestibular nucleus, resulting in a range of vestibular and ocular symptoms. Patients typically present with horizontal and rotational nystagmus, double vision (diplopia), oscillating vision (oscillopsia), dizziness, nausea, and vomiting. The rapid phase of rotatory nystagmus often causes the upper border of the iris to move towards the side of the lesion. A larger amplitude, slower horizontal nystagmus is observed when the patient looks towards the affected side, whereas a smaller amplitude, quicker nystagmus occurs with gaze directed to the opposite side. [10]

One of the most incapacitating manifestations of Wallenberg's syndrome is ipsilateral ataxia, stemming from infarction in the inferior cerebellar peduncle.

Additionally, vertigo results from damage to the vestibular nuclei. When the spinocerebellar tract is affected, patients may experience limb ataxia and a sensation of falling towards the side of the lesion. The descending sympathetic fibers, located adjacent to the spinothalamic tract in the lateral tegmentum of the brainstem, can also be affected, resulting in ipsilateral Horner's syndrome. This syndrome is characterized by partial ptosis, miosis, and occasionally anhidrosis. Autonomic dysfunctions, such as diaphoresis, tachycardia, bradycardia, and orthostatic hypotension, may also manifest [10].

Manifestation of difficulty swallowing (dysphagia), hoarseness, and a diminished gag reflex is a result from paralysis of the palate and vocal cords, involving the 9th (glossopharyngeal) and 10th (vagus) cranial nerves. Damage to the nucleus and tractus solitarius is linked to loss of taste. The nucleus ambiguus, located dorsally adjacent to the inferior olivary nucleus, sends branchial motor fibers via the vagus nerve to the muscles of the palate, pharynx, and larynx, and via the glossopharyngeal nerve to the stylopharyngeus muscle. Infarction affecting this nucleus and the exiting fibers of cranial nerve IX can lead to breathy hoarseness, dysphagia, reduced gag reflex on the affected side, and ipsilateral vocal cord paralysis, as confirmed by laryngoscopy [10].

Hiccups (singultus) represent an uncommon but significant symptom of lateral medullary infarction. While the exact anatomical mechanism behind hiccups remains unclear, they often occur due to lesions in the middle and dorsolateral regions. Hiccups involve repetitive, involuntary spasms of the diaphragm followed by abrupt closure of the glottis, resulting in the characteristic "hic" sound [11].

Damage to the lateral spinothalamic tract results in contralateral deficits in pain and temperature sensation from the body. The cuneate and gracilis nuclei, which are responsible for numbness in the ipsilateral arm, trunk, and leg, can also be impacted. According to a study conducted at the Stroke Centre between 1983 and 1989, which evaluated 33 consecutive patients with lateral medullary syndrome, the triad of Horner's syndrome, ipsilateral ataxia, and contralateral hyperalgesia effectively identifies individuals with lateral medullary infarction. Although facial weakness and ocular symptoms are common, they do not necessarily indicate extension of the infarction beyond the lateral medulla. Moreover, vascular imaging or isonization studies confirmed vertebral artery disease in 73% of patients. Cerebellar infarcts were rarely concurrent with lateral medullary syndrome, suggesting that the majority of the

posterior inferior cerebellar artery (PICA) territory remains unaffected despite frequent occlusion of the vertebral artery [11,12].

Hemiplegia is uncommon in Wallenberg's syndrome but can occur if there is involvement of the corticospinal tract within the medulla. This complication is observed in conditions such as vertebral artery dissection, also known as Opalski syndrome. A review of literature comparing hemimedullary syndrome in patients with vertebral artery dissection versus those with vertebral artery atherosclerotic disease indicated that vertebral artery dissection tends to cause a hemimedullary lesion at a lower level compared to atherosclerosis. This affects the medullary-penetrating branches supplying the medulla just below the pyramidal decussation [13,14].

The diagnosis of Wallenberg syndrome typically necessitates the presence of two main criteria: a lesion in the medulla oblongata causing dysarthria or dysphagia, and a lesion in the dorsolateral medulla oblongata resulting in dysesthesia, Horner's syndrome, and ataxia. According to these diagnostic criteria, our patient complete the criteria with dysarthria (slurred speech), dysesthesia (facial sensory deficit, numbness, left-sided body numbness), and ataxia. These clinical features, when considered alongside the findings from Brain Transcranial Doppler combined with MRI findings of a right medullary infarct, meets the established diagnostic criteria for Wallenberg syndrome. This comprehensive approach ensures an accurate diagnosis and facilitates appropriate management of the condition [15].

The primary method used to diagnose posterior circulation infarcts, including Wallenberg's syndrome, is diffusion-weighted imaging (DWI) on brain CT or MRI.[8,9] If the diagnosis is made as soon as feasible followed by prompt and appropriate therapy, generally leads to a favorable prognosis for patients with lateral medullary syndrome. If there are no contraindications, patients with lateral medullary syndrome who present to the emergency department in 4.5 hours after the symptom's onset, they can receive intravenous tissue plasminogen activator (tPA) therapy, similar to other types of ischemic stroke. Additionally, anticoagulation, antiplatelet therapy, and strict control of risk factors are essential as secondary prevention measures [15].

The patient in this case study that was admitted to our neurology department, a 50-year-old male patient presented with a constellation of symptoms, including vertigo, slurred speech, and sensory deficits, which were initially misattributed to less serious conditions. His high blood pressure was a significant contributing factor, complicating his clinical presentation and delaying an accurate diagnosis of

Wallenberg's syndrome. This case underscores the complexity and severity of lateral medullary syndrome, particularly in patients with uncontrolled hypertension.

CONCLUSION

We present a case of a 50-year-old male patient suffering from uncontrolled hypertension and the unusual Wallenberg's syndrome. In conclusion, lateral medullary syndrome, though uncommon, should be considered in patients presenting with a combination of vertigo, nystagmus, dysphagia, slurred speech, or ipsilateral face sensory impairments particularly when these symptoms occur in the context of high-risk factors such as hypertension and atherosclerosis. Early recognition and treatment are critical to improving outcomes and minimizing long-term disability. This case highlights the importance of a thorough and systematic approach to diagnosis and management, ensuring that even rare stroke syndromes are accurately identified and treated promptly.

Patient consent:

The authors certify that they have obtained verbal patient consent. The patient identity cannot be identified.

Conflict of interest:

We certify that none of the writers of this work have any conflicts of interest. The conduct, preparation, collecting, analysis, interpretation, and writing of the report were all done without any financial assistance.

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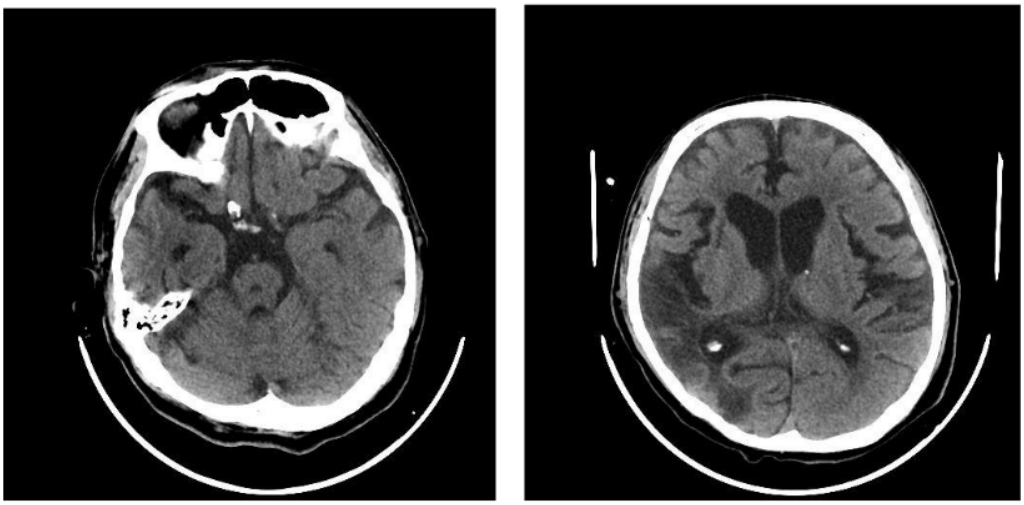
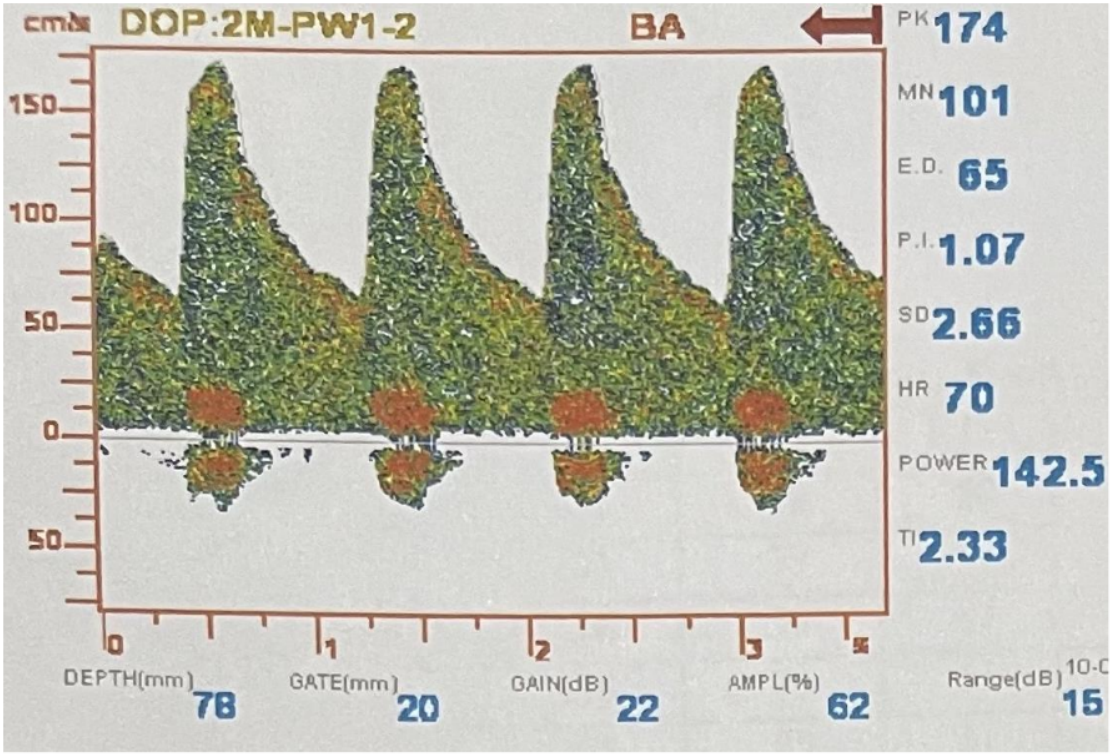


(Figure 1) The infarction in medulla and parieto-occipital region



(Figure 2) Severe stenosis in basilar artery (peak systolic velocity/PK > 90 m/s)