Case Report

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Lateral Medullary Syndrome: Wallenberg

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Abstract

Lateral Medullary Syndrome (Wallenberg) is a severe neurological condition associated with intracranial pathology, particularly the occlusion of the posterior inferior cerebellar artery branch. Symptoms include Horner's syndrome, ipsilateral gait ataxia, and hypoalgesia, and ipsilateral thermoanesthesia of the face. A 52-year-old male patient presented with complaints of nausea, vomiting, and unsteady gait. The patient had undergone cardiac angioplasty 4 months ago but had not adhered to his prescribed medical treatment nor attended follow-up cardiology appointments. His symptoms, which began around noon, were further complicated by difficulty swallowing, hoarseness, and hiccups. As approximately 8 hours had passed since the onset of symptoms, thrombolytic therapy could not be administered. He was admitted to the neurology ward for further evaluation. Lateral Medullary Syndrome (Wallenberg) can present with diffuse and subtle symptoms. Early hospitalization is crucial, especially for patients with a history of cardiac angioplasty, as timely intervention can be vital for improving outcomes.

Keywords: Lateral medullary syndrome, vertebral arteries occlusion, wallenberg

Introduction

Lateral medullary syndrome (Wallenberg) is caused by the occlusion of the vertebral artery and, typically, its branch, the posterior inferior cerebellar artery (PICA) (1). Clinical findings include ipsilateral ataxia resulting from damage to the inferior cerebellar peduncle and cerebellum, ipsilateral Horner's syndrome due to the involvement of intrinsic sympathetic axons descending to the cervical cord, and nausea, vomiting, and vertigo due to damage to the vestibular nuclei. Dysphagia and dysarthria occur as a result of ipsilateral pharyngeal and laryngeal dysfunction caused by damage to the nucleus ambiguus. Ipsilateral trigeminal nerve involvement leads to facial sensory loss, and damage to the spinothalamic tract results in decreased pain and temperature sensation on the contralateral side (1-3). The occlusion of the intracranial segment of the vertebral artery is more common compared to PICA occlusion (1,4), which accounts for 2.5% of brain infarctions (5). Possible risk factors include hypertension, diabetes, smoking, atrial fibrillation, history of ischemic heart disease, and prior radiation therapy to the neck region (6). The syndrome was named after Adolf Wallenberg, a famous neurologist. Compared to anterior circulation ischemia, posterior circulation ischemia has generally been diagnosed later. While computed tomography (CT) was indispensable before the mid-1980s, the advent of magnetic resonance imaging (MRI) has significantly facilitated diagnosis (7).

Case Report

We evaluated a 52-year-old male patient who presented to our emergency room with complaints of nausea, vomiting, dysphagia, and unsteady gait. The patient, who arrived at the emergency department at 8:00 PM, reported that his symptoms had started at noon with dysphagia and hoarseness occurring simultaneously. He stated that nausea, vomiting, and hiccups developed later in the evening, and unsteadiness while walking appeared within the last hour before his presentation. However, approximately 8 hours had elapsed since the onset of the initial symptoms. The patient's vital signs were as follows: blood pressure (BP): 135/85 mmHg, heart rate (HR): 84/min, oxygen saturation (O_2 Sat): 98%, respiratory rate (RR): 22/min, and body temperature: 36.8°C. On physical examination, the oropharynx appeared normal, and no uvular deviation was observed. Lung auscultation revealed no additional sounds. The abdomen was soft and nontender. Left-sided ptosis was noted, and the

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left pupil was miotic. Nausea and vomiting were persistent. No nystagmus was observed. Hoarseness and hiccups were present. There was no muscle weakness in the extremities, and the Babinski reflex was bilaterally negative. No headache was reported. However, ataxia was evident, and the patient performed poorly on cerebellar function tests. The patient was alert, oriented, and cooperative, with a Glasgow Coma Scale (GCS) score of 15. He reported numbness on the right side of his body, particularly insensitivity to painful stimuli and to hot and cold sensations. From his medical history, it was learned that he had undergone coronary angioplasty 4 months earlier. However, he had not attended follow-up appointments and had not taken his medications regularly.

The patient's vascular access was established, and blood samples were collected for complete blood count (CBC), biochemistry, and cardiac markers. Ondansetron, at a dose of 4 mg, was administered intravenously alongside a saline infusion. An electrocardiogram (ECG) revealed a normal sinus rhythm. Laboratory results showed a blood glucose level of 125 mg/dL, with normal urea, creatinine, electrolyte levels, and liver enzymes. The troponin level was measured at 6.1 ng/L, and the creatine kinase (CK) level was 261 U/L. A brain computed tomography (CT) scan revealed no abnormalities. However, a brain magnetic resonance imaging (MRI) scan detected a left cerebellar infarction, visible on a single slice (Figure-1). The patient was urgently referred to a neurologist and admitted to the neurology department for further evaluation and treatment.

Discussion

Lateral medullary syndrome (Wallenberg) presents with symptoms such as vertigo, Horner's syndrome, numbness on the same side of the face, pain and loss of temperature sensation on the opposite side of the body. Hemiparesis and muscle weakness may also occur. Additional findings can include double vision and uvulopalatal deviation. The goal of early diagnosis in these patients is to initiate treatment promptly if the patient's condition allows. Thrombolytic therapy is administered within 90 minutes of symptom onset. The recommended dose of tissue plasminogen activator (t-PA), Alteplase, is 0.9 mg/kg, not exceeding 90 mg. Of this, 10% is given as an initial bolus, while the remaining 90% is infused over the first hour (8). Unfortunately, thrombolytic therapy could not be initiated in our patient due to a significant delay in presentation, despite the patient meeting other eligibility criteria, including age, blood pressure, and the absence of diabetes, fever, hypotension, hyperglycemia, and hypoxia. The posterior circulation system supplies the cerebellum, brainstem, thalamus, medial temporal lobes, and occipital lobes via the vertebral-basilar arteries and their branches (8,9). Pathologies affecting this system, such as vascular occlusion, trauma, or aneurysm, may have a diffuse and subtle onset. This can delay the patient's recognition of the severity of their symptoms and their presentation to a healthcare facility, ultimately leading to delays in treatment.

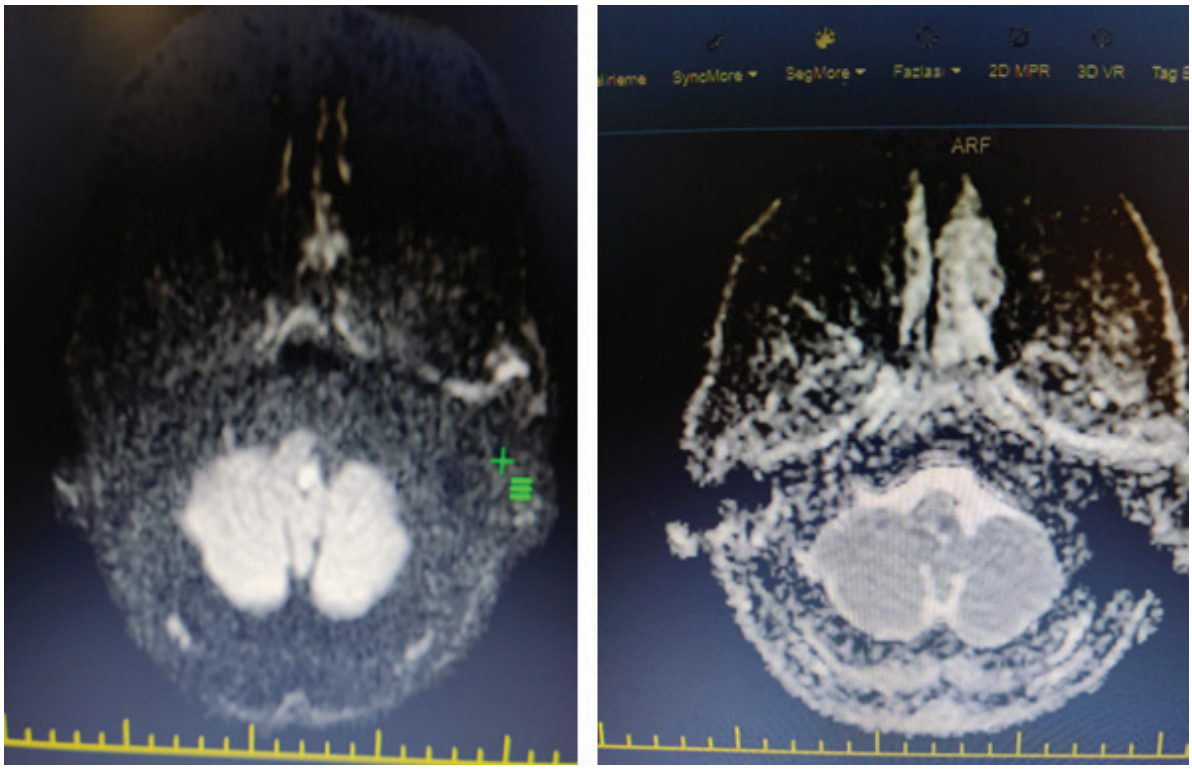

Figure 1. Brain MRI scan detected a left cerebellar infarction

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Thrombolytic therapy is the only approved emergency treatment for acute ischemic stroke if the patient meets the necessary criteria (10,11). It is also important to consider that emboli of cardiac origin can often be silent but may lead to severe neurological deficits. Common sources of cardioembolism include acute myocardial infarction, left ventricular aneurysm, cardiac arrhythmias such as atrial fibrillation, and balloon angioplasty (11). Hiccups, which may accompany the clinical presentation of patients, are reflex-originated activities caused by intermittent contractions of the diaphragm and the glottis (12). They are observed in 12–36% of cases during the course of lateral medullary syndrome (13). There may also be variants, which include several different clinical conditions, such as Opalski syndrome (14,15). This syndrome can be associated with various diseases, including autoimmune conditions like connective tissue diseases and multiple sclerosis (16,17).

Conclusion

Patients with lateral medullary syndrome may present with a wide variety of clinical findings. The patient's clinical presentation can range from a simple upper respiratory tract infection to more serious neurological symptoms. Lateral medullary syndrome should be considered in the differential diagnosis during patient evaluation. Emergency room monitoring and early diagnosis facilitate timely treatment. Treatments implemented in recent years have been satisfactory, but timely intervention is essential.

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