CASE REPORT

Lateral Medullary Syndrome: An Unusual Central Cause for Unilateral Vocal Cord Palsy

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Abstract
Lateral medullary syndrome, also known also as Wallenberg syndrome, remains an interesting clinical entity with varied presentations of cerebrovascular accidents. The area of the brain stem involved is the posterolateral part of the medulla oblongata receiving arterial blood supply from the posterior inferior cerebellar artery. We present a 60-year-old man presenting with unilateral vocal cord palsy due to an unusual central cause, being part of lateral medullary syndrome. We reviewed the neuroanatomy and clinical aspects of this condition.

Key words: Lateral medullary syndrome, Wallenberg syndrome, PICA syndrome, Vocal cord palsy.

Introduction

Lateral medullary syndrome (LMS) remains an interesting clinical entity with a wide range of clinical presentations of cerebrovascular accidents (CVA). Few case reports have been published on the atypical presentations of lateral medullary syndrome, possible complications, management and outcome. These reports aim to improve prompt recognition, diagnosis, and management of such uncommon but serious condition (1,2). The area of the brain stem involved in LMS is the posterolateral part of the medulla oblongata (3), which is the portion receiving arterial blood supply from the posterior inferior cerebellar artery (PICA) (1,2). The latter is a branch of the vertebral artery; that is the artery that initiates the vertebrobasilar (posterior) system of blood supply to the brain. Occlusion or hemorrhage involving PICA would impede the blood supply to that part of the medulla, causing dysfunction of several nuclei (Figure 1) (4). Few case reports have been published on the atypical presentations of lateral medullary syndrome, possible complications, and how to manage them, aiming to improve prompt recognition, diagnosis, and management of such serious patients. To this end, we report this case and review the literature.

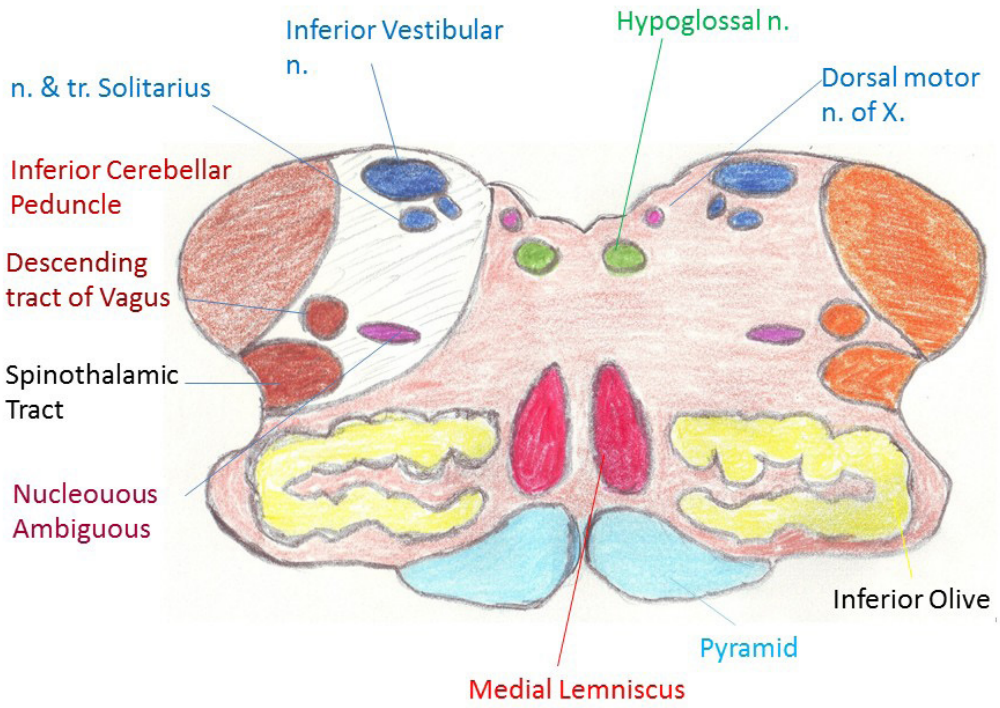

Figure 1. The neuroanatomy of the brain stemm region demostrating the tracts and nuclei affected in LMS (4).

Case History

We report a 60 years old Indian male, who presented to the emergency department (ED) with acute hoarseness of voice, right-sided body anasthesia, nausea and vertigo for two days. He also lost sweating on one side of his face. The patient is a known hypertensive, heavy smoker for the past 40 years, has atherosclerotic coronary artery disease (CAD) with PTCA done 10 years ago. In addition, he reported history of a previous admission to the hospital for an ischemic stroke 1 month prior to this presentation. Brain imaging at that time showed right internal capsule lacunar infarct, The patient was started on multi-drug regimen (antiplatelets, beta-blocker, statin, calcium channel blocker and ARB) then he was discharged. He visited another hospital 2 days prior to presentation, and a laryngoscopy showed left vocal cord palsy. Peripheral causes of vocal cord palsy were investigated; a chest x-ray as well as CT chest showed no abnormality. However, the possibility of a central cause for the vocal cord palsy was overlooked at that time. On examination, patient was stable with normal BP, but had a clearly hoarse voice and unilateral absence of the gag reflex; on the left side of the pharynx. Patient states he has been tilting his head towards the right side when swallowing liquids. He adopted this maneuver to avoid choking and coughing. Neurological examination showed right-sided body loss of pinprick and temperature sensations, as well as left-sided facial loss of pinprick and temperature sensations. There was clinical picture of Horner's syndrome on the left side of the face: partial ptosis, miosis, and anhydrosis (Figure 2). Tests for cerebellar functions revealed some dysdiadochokinesia of the left hand and foot, and moderate ataxia. Chest exam showed reduced air entry over both lung bases with fine crepitations heard over the left base. CT scan of the head showed no acute infarct or hematoma. Patient was hyperglycemic, HbA1c = 7.8%, and troponins were negative. Fasting lipid profile showed hypertriglyceridemia and low HDL [Total serum cholesterol was 4.86 mmol/L; HDL: 0.92 mmol/L; LDL: 2.64mmol/L; Triglycerides: 2.84mmol/L and Chol/HDL: 5.3]. ECG showed sinus rhythm, Q waves in the inferior leads and T-wave inversion in the lateral leads. MRI was sought, but patient gave history of working with metallic sawdust and possibility that his body contains some of it. The MRI machine did give a warning signal before starting imaging, indicating a metallic body in the eyes region. X-RAY orbits done next day showed a 1.8 mm radiopaque shadow in the left orbital region (Figure 3). Therefore, a repeat CT head instead was done 48 hours later showed a small ill-defined hypodensity in the left dorsal lower pons/upper medulla consistent with lacunar infarct, and an ectatic left vertebral artery (Figure 4). Speech-Language Pathology assessment showed no

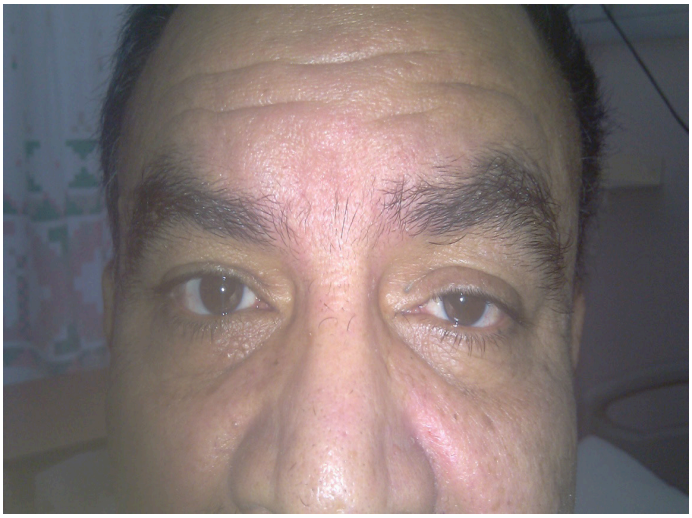

Figure 2. The clinical appearnce of the patient second day of admission to hospital. Note the partial ptosis in the left upper eye lid. Patient's consent taken for publishing this photo.

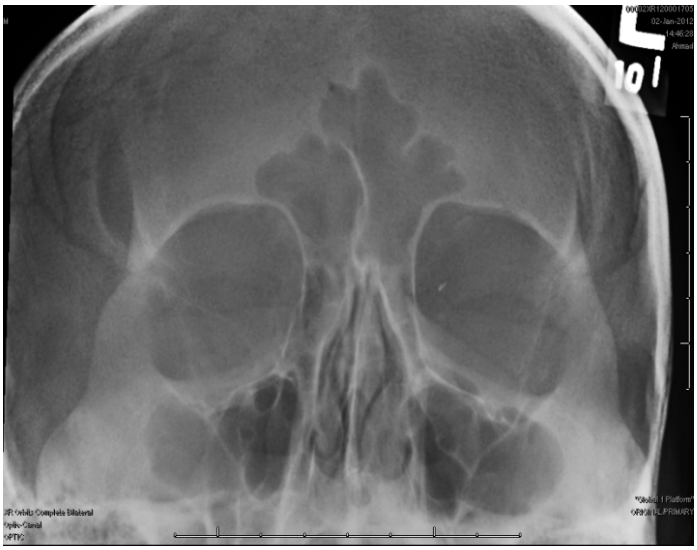

Figure 3. Plain X-RAY of the orbits. Note the very small, 1.8 mm radiopaque shadow in the left orbital region, representing a metallic foreign body.

significant dysphagia and no risk of aspiration. Patient continued to receive Aspirin, Clopidogrel, Bisoprolol, Rosuvastatin, Valsartan-Amlodopine that he was taking before admission, in addition to metformin and Omega-3 fatty acids supplementation. A fibrate wasn't added to the statin for the hypertriglyceridemia because of the added risk of muscle breakdown. Upon discharge, he was firmly counseled on quitting smoking; as it is the persistent risk factor for the ongoing atherosclerosis in his cerebral and coronary circulations.

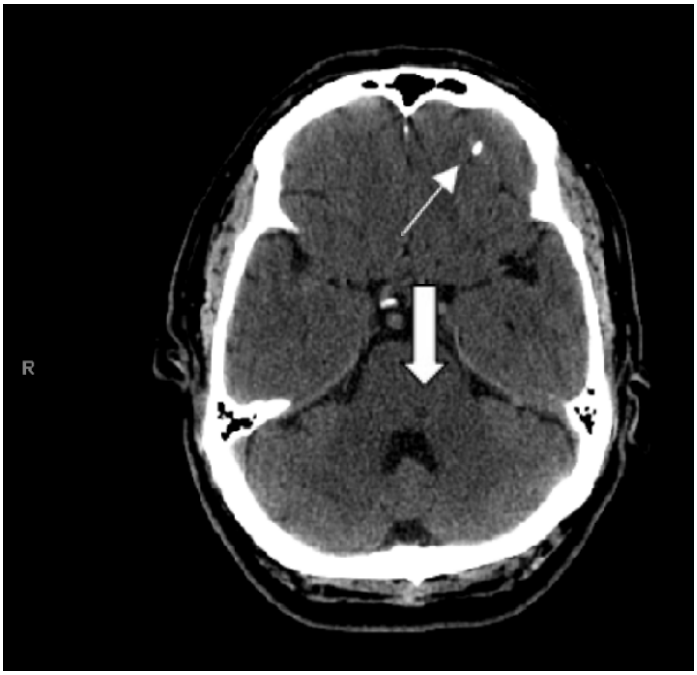

Figure 4. CT head without contrast done 48 hours from admission showing a small ill-defined hypodensity in the left dorsal lower pons/upper medulla consistent with lacunar infarct *(thick arrow)*, and an ectatic left vertebral artery. A small metallic foreign body is still seen in the left orbital region *(thin arrow)*.

Discussion

Lateral medullary syndrome is also known as Wellenberg's syndrome and PICA Syndrome. The first clinical description of lateral medullary syndrome was made by the German internist Adolf Wallenberg (1862–1949) in 1895 (5); soon followed by autopsy confirmation in 1901 (6). Reportedly, Adolf Wallenberg was renowned in his time for his careful history taking and neurological examination, and for his insistence on clinicopathological confirmation (7). However, the syndrome was first ever described by the Swiss physician Gaspard Viesseux (1746-1814), who rendered an exact description of his own disease in 1808 at the Société médicochirugicale de Genève and in 1910 at the Medical and Chirurgical Society of London (8).

The clinical presentation of this type of stroke allows some variation between different patients. Similar to any other stroke, LMS can be caused by thrombosis as in our case, embolism, or hemorrhage as has been reported in other LMS cases (3). Severity of the presentation, the need for emergency intervention and subsequent monitoring will depend on the cause and size of the area affected in the brainstem. For example, our patient presented with a stable status that required only secondary prevention by

Table 1. Clinical picture of lateral medullary syndrome and the likely corresponding anatomical location	
Presenting Symptoms and signs	**Likely anatomical location**
Contralateral loss of pain and temperature sensation.	Spinothalamic tract
Ipsilateral facial loss of pain and temperature.	Spinal trigeminal nucleolus
Dysphagia/dysphonia and depressed gag reflex.	Nucleus ambiguous (supplies cranial nerves IX and X)
Vertigo/diplopia/nystagmus/vomiting.	Inferior vestibular nucleus
Ipsilateral Horner's syndrome.	Sympathetic fibers
Palatal clonus.	Central trigeminal tract
Ataxia.	Inferior cerebellar peduncle

medications and lifestyle changes to stop his progressive atherosclerosis. On the other hand, Nicholson et al. (3) and Vaidyanathan et al. (9). Both reported LMS cases presenting with acute upper airway obstruction and stridor requiring intubation and precipitating respiratory failure and Saha et al (2) reported a patient with LMS due to an embolus from the heart, requiring cardiology attention and treatment of the underlying cardiac disease.

Despite starting extensive anti-stroke drug therapy for this patient after diagnosing his first lacunar infarct 3 months prior to this presentation, and ensuring compliance, patient continued to have residual risk, which resulted in his progression to a second stroke (10). In addition, smoking -to be emphasized more than all- remains the most important and constant risk factor in this patient and most others (3). The presence of most symptoms and signs of LMS alongside hoarseness of voice or stridor as has been reported (1,2), aids the prompt diagnosis (Table 1). However, when the presentation is limited to only the latter symptoms, clinicians should keep in mind that only central causes of vocal cord palsy would be associated with bulbar palsy (dysphagia).

Stroke remains one of the most common pathologies a physician may meet in clinical practice, with very numerous clinical presentations. We emphasize that central causes of unilateral vocal cord palsy are becoming increasingly common, and should be sought when peripheral causes have been roled out, so that anti-stroke therapy would be initiated as early as possible.

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