

LATERAL MEDULLARY SYNDROME (WALLENBERG SYNDROME) AND DYSPHAGIA – A CASE REPORT

DEEPIKA PERUMILLA^{1*}, MEGHANA GUNDRE¹, SYAM PRASAD TIRUMALA¹, UHAPRIYA MANDYAM¹, MADHAVI VALLETI²

¹ Pharm.D intern, Department of Pharmacy Practice, Sri Padmavathi School of Pharmacy, Tiruchanoor, Tirupathi-517503. ² Assistant professor, Department of Pharmacy Practice, Sri Padmavathi School of Pharmacy, Tiruchanoor, Tirupathi-517503.

Email: pdeepikadaddy@gmail.com

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ABSTRACT

Lateral Medullary Syndrome (LMS) or Wallenberg Syndrome or Posterior Inferior Cerebellar Artery (PICA) Syndrome is a rare prevailing cause of stroke. Varied presentations of LMS such as distortion of speech, orientation and vision is cause for under diagnose of the disease. **Objective:** In our case, we present a 50 year old hypertensive man presenting with loss of consciousness and difficulty in walking. On neurological evaluation he had swaying episodes towards left, blurring of vision with nystagmus, regurgitation of feeds and dysphagia. With this clinical and radiological examination he was diagnosed as a case of Lateral Medullary Syndrome. **Conclusion:** The patient's condition improved with symptomatic treatment and was nearly asymptomatic at the time of discharge.

key words: Lateral Medullary Syndrome, dysphagia, nystagmus, symptomatic therapy.

INTRODUCTION

The lateral medullary syndrome was first described in 1808 by Gaspard Vieussens. First descriptions by Wallenberg were in 1895 (clinical) and 1901 (autopsy findings). It is most often caused by occlusion of the intracranial segment of the vertebral artery (VA), less commonly; it is caused by occlusion of the posterior inferior cerebellar artery (PICA) [1].

The area of the brain stem involved in LMS is the posterolateral part of the medulla oblongata, which is the portion receiving

arterial blood supply from the posterior inferior cerebellar artery (PICA). 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 haemorrhage involving PICA would impede the blood supply to that part of the medulla, causing dysfunction of several nuclei.[2]

Features common with lateral medullary syndrome [3]

SIGNS

Ipsilateral loss of pain and thermal sense over half of face
Ataxia, falling to side of lesion
Ipsilateral Horner's syndrome (constricted pupil, ptosis, decreased sweating)
Dysphagia, dysarthria, ipsilateral paralysis of palate and vocal cord, diminished ipsilateral gag reflex
Contra lateral impaired pain and thermal sense below the neck (UE and LE)
Vertigo/dizziness

Nausea/vomiting, nystagmus, diplopia

STRUCTURES AFFECTED

Spinothalamic tract and descending nucleus and tract of cranial nerve V
Inferior cerebellar peduncle, spinocerebellar tract
Descending sympathetic tract

Efferent fibres of cranial nerves IX and X, and nucleus ambiguus (NA)

Spinothalamic tract and nucleus of cranial nerve V carrying pain and temperature sense to the opposite side of the body
Vestibular nuclei and vestibulocerebellar pathway in the inferior cerebellar peduncle
Vestibular nuclei

Clinical symptoms include swallowing difficulty, or dysphagia, slurred speech, ataxia, facial pain, vertigo, nystagmus, Horner's syndrome, diplopia and possibly palatal myoclonus. [4]

Patient signs and symptoms are divided into very common (90%), moderately common (50 to 70%) and less common (< 40%). The most common signs and symptoms are sensory symptoms/signs, gait, ataxia, dizziness and Horner sign. There are sensory signs affecting trunk and extremities opposite site of lesion and face and cranial nerves on the same site of lesion and face and cranial nerves on the same site of lesion. The syndrome is characterized with loss of pain and temperature sensation on the contra lateral side of body and ipsilateral side of face.⁵

CASE REPORT

A male patient 50 years old, hypertensive presented to General Medicine Department with complaints of loss of consciousness that lasted for about 5 min followed by altered sensorium and difficulty in walking since one day. The patient had swaying episodes to left and is unable to stand. The patient had history of headache, blurred vision, dysphagia and regurgitation of feeds. On examination the patient was conscious and coherent with Pulse Rate 80/min and Blood Pressure 160/110 mm of Hg and

neurological examination revealed pupils-right 3mm and left 2mm reacting to light, plantar showed right-extensor and left-withdrawal, left facial palsy, nystagmus, seventh, ninth and tenth cranial nerve palsy and absence of gag reflex. The 2D ECHO revealed Ischemic Cardio Myopathy with mild to moderate left ventricular dysfunction. Laboratory investigations had shown elevated serum creatinine – 2.7 mg/dl and CBC was normal. The patient was managed with osmotic diuretic Mannitol with stat dose of 300ml and maintenance dose of 100ml thrice a day for 5 days and continued with Oral Glycerol until discharge. A low molecular weight heparin Enoxaparin 40mg was given subcutaneously twice a day for 5 days. Anti-platelet and Anti-coagulant agents such as Clopidogrel 75mg OD and Aspirin 150mg OD were also given. Third generation Cephalosporin Ceftriaxone 1gm was given intravenously for a period of 5 days. An Anti-hypertensive agent Enalapril 5mg was given orally once daily. A Proton pump inhibitor Pantoprazole 40mg was given to reduce gastric irritation. A Cardiac glycoside Digoxin 0.125mg was given for 5 days.

DISCUSSION

Wallenberg syndrome is an uncommon stroke. It is caused by a stroke in one of the two arteries of the brainstem. The stroke

associated with WS usually causes damage in the lateral medulla of the brainstem. It may also cause damage to the cerebellum. The case can be diagnosed clinically or can be confirmed with CT or MRI scan.[1]

The clinical presentation of this type of stroke allows some variation between different patients. Similar to any other stroke, WS can be caused by thrombosis, embolism, or hemorrhage. 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.[2]

Among other signs and symptoms, dysphagia has been reported in 51% to 94% of the patients with WS. It has been widely accepted that in most cases the dysphagia in WS is initially severe enough to require no oral feeding but often improves rapidly, and the patient can return to oral feeding within 1 to 2 months after the stroke.⁴ In this case patient had dysphagia which is caused by the paralysis of palate and vocal cord (the ninth and tenth cranial nerves). Here the patient had experienced ipsilateral ataxia and falling towards the side of lesion, which is caused by infraction of the inferior cerebellar peduncle. Feeling of falling towards the side of lesion is due to the disease of the spinocerebellar tract. [6]

Here, in this case the patient was managed with symptomatic treatment and was given an osmotic diuretic Mannitol to decrease intracranial pressure. Aspirin is the preferred choice in this case as it reduces the risk of early recurrent ischemic stroke and Antiplatelets agents like clopidogrel was also given. The choice of aspirin combined with clopidogrel is a usual practice in the hospital and such a pattern lead to optimization of medical needs confined to the patient.⁶ There is large collection of LMIs documented by MRI. In some studies, based on MRI findings LMI lesions were classified into rostral lesions and caudal lesions.⁷ Neurologic and functional recovery is dependent on the initial stroke severity.⁸

The syndrome has been noted in association with mechanical trauma to the vertebral artery in the neck, vertebral arteritis, metastatic neoplasm, hematoma, aneurysm of the vertebral artery, herpetic brain stem encephalitis and arteriovenous malformations.⁹ All patients with suspected acute stroke should receive urgent neuroimaging to exclude alternative diagnosis and to screen for contraindications to stroke therapies such as intracerebral hemorrhage, focal compression or herniation. MRI remains the gold standard test for the diagnosis of acute stroke, with an overall sensitivity of 83% and specificity 96%. Patients presenting within 4.5 hrs of symptom onset should be considered for treatment with intravenous tissue plasminogen activator, in consultation with specialty services managing acute stroke care.[10]

CONCLUSION

Lateral medullary syndrome can have a unpredictable presentation of signs and symptoms dependent on the brainstem nuclei and spinal tracts involved, the patient should remain NPO until the swallowing is fully evaluated to prevent major aspiration and intubation. Diagnosis of this syndrome requires clinical signs and symptoms as well as radiological studies MRI of the head and neck to identify the lesion. The central causes of unilateral vocal cord palsy are becoming increasingly common, and should be required when peripheral causes have been ruled out, so that anti-stroke therapy would be initiated as early as possible.

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