



A Rare Variant of Lateral Medullary Syndrome with Ipsilateral Lower Motor Neuron Facial Palsy

Sriramakrishnan V¹, Manwen konyak¹, Anto Ignat Stany¹

¹ Department of Neurology, Tirunelveli Medical College & Hospital, Tirunelveli

Submitted: 25-01-2021

Revised: 05-02-2021

Accepted: 08-02-2021

ABSTRACT: Lateral medullary syndrome or Wallenberg syndrome is an interesting clinical entity with varied presentations. Its clinical features include ipsilateral Horner's syndrome, ataxia, pain, decreased sensation over face, palate, pharynx, vocal cord palsy, dysphagia, dysarthria and contralateral decreased sensation of pain and temperature over trunk and extremities. However, variable presentation of this syndrome occurs, as seen in this case presentation and various literature reviews. Facial nerve involvement can occur in variant of lateral medullary syndrome with pontomedullary junction lesions. Its involvement in pure lateral medullary lesion is very unusual. We report here a case of 30-year-old female with classical features of lateral medullary syndrome with ipsilateral lower motor neuron facial palsy. Magnetic resonance imaging brain showed right dorsolateral medullary infarct. The facial nerve involvement can be explained by possible involvement of the facial nerve at the pontomedullary exit from occlusion of the aberrant vertebral artery which supply the exiting nerve.

Keywords : Lateral medullary syndrome, LMN facial palsy, aberrant vertebral artery, lower motor neuron

I. INTRODUCTION

The lateral medullary syndrome was first described in 1808 by Gaspard Vieussux. First descriptions by Wallenberg was in 1895 (clinical) and 1901 (autopsy findings)¹. It is most often secondary to intracranial vertebral artery (67%) or posterior inferior cerebellar artery (10%) occlusion². Spontaneous dissections of the vertebral arteries are a common causes. The syndrome has also been described with cocaine abuse³, medullary neoplasms (usually metastases), abscess, and demyelinating disease⁴, neck trauma, radionecrosis and bullet injury to vertebral artery. The patterns included ipsilateral trigeminal ± contralateral limb/body pattern in 26%. Trigeminal involvement without limb/body involvement in 10%³. The triad of Horner syndrome, ipsilateral ataxia, and contralateral hypalgesia clinically identifies

patients with lateral medullary infarction⁵. Along with these classical features, it presents with many rare manifestations. Its occurrence is unusual and especially with LMN facial palsy. Although central facial palsy has been reported in few literatures, here we report a very unusual association of lateral medullary syndrome with LMN facial palsy. Clinical features with the possible structures affected are given in table 1.

II. CASE REPORT

A 30 year old female diabetic presented with a c/o sudden onset giddiness, vomiting, difficulty in swallowing (liquids > solids), swaying while walking, numbness on left half of the body, deviation of angle of mouth to right side and tinnitus. On examination, general examinations were unremarkable and her vital parameters were normal. Her higher mental functions were normal. There was dysarthria with nasal twang and difficulty to pronounce gutturals, decreased pain and temperature over left half of face, absent direct corneal reflex, deviation of angle of mouth to right side, loss of nasolabial fold on right side, with absent forehead wrinkles and associated with bell's phenomenon { Figure 1 }. On phonation, uvula deviated to the right, drooping of left soft palate, absent gag reflex. Motor examinations & reflexes were normal. Sensory examination revealed decreased pain and temperature on right face and contralateral half of the body. Cerebellar dysfunction including horizontal nystagmus, impaired finger-nose finger test & impaired tandem walking were noted. Routine blood and other investigations were normal {table 2}. CT Brain showed no abnormality. MRI Brain showed hyperintensity in the right upper dorsolateral medulla suggestive of acute infarct { Figures 2,3 }. Antiplatelets, Anticoagulation and other supportive treatments were given and her dysphagia was improving and patient was discharged after one week with an advice for physiotherapy.



III. DISCUSSION

The presence of facial palsy is very unusual in otherwise typical case of lateral medullary syndrome. Central facial palsy may be explained in lateral medulla syndrome by damage to hypothetical looping supranuclear CBT fibers, hypothesized to descend down in contralateral ventromedial medulla, decussate at the level of upper medulla and ascend dorsolaterally to reach facial nucleus^{7,8}. Ipsilateral LMN facial palsy usually results from lesions involving the inferolateral part of the pons^{9,10}. However, in our case ipsilateral LMN facial palsy was present without involvement of the caudal pons. Probably it resulted from an occlusion of the aberrant arterial branch arising from upper vertebral artery running superiorly and laterally to the region of exit of cranial nerves VII and VIII from the pons⁹. It may also be due to small embolic infarct as evidenced clinically by maximal deficits at the onset and which could not be detected on imaging.

IV. CONCLUSION

Facial nerve involvement in lateral medullary syndrome, although rare, has been reported in various studies. Most commonly it is the central type of facial palsy but LMN facial palsy may also be present as in our case which can be explained by the involvement of aberrant branch of vertebral artery that supplies the exiting VII cranial nerve at the ponto-medullary junction or due to small embolic infarct which could not be detected on imaging.

Conflict of Interest

Nil

Disclosures

The authors have no disclosures.

REFERENCES

- [1]. Wallenberg A. Acute bulbar palsy. Posterior inferior cerebellar artery embolism. Arch Psychiatry 1895;27:504-40.
- [2]. Kim JS. Pure lateral medullary infarction: Clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126:1864-72.
- [3]. Mody CK, Miller BL, McIntyre HB, Cobb SK, Goldberg MA. Neurologic complications of cocaine abuse. Neurology 1988;38:1189-93.
- [4]. Smith DB, Demasters BK. Demyelinating disease presenting as Wallenberg's syndrome. Report of a patient. Stroke 1981;12:877-8.
- [5]. Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome. Clinical-magnetic resonance imaging correlations. Arch Neurol 1993;50:609-14.
- [6]. Norrving B, Cronqvist S. Lateral medullary infarction: Prognosis in an unselected series. Neurology 1991;41:244-8.
- [7]. Terao S, Takatsu S, Izumi M, Takagi J, Mitsuma T, Takahashi A, et al. Central facial weakness due to medial medullary infarction: The course of facial corticobulbar fibres. J Neurol Neurosurg Psychiatry 1997;63:391-3.
- [8]. Urban PP, Wicht S, Vucorevic G, Fitzek S, Marx J, Thömke F, et al. The course of corticofacial projections in the human brainstem. Brain 2001;124:1866-76.
- [9]. Paul W, Brazis, Joseph C, Masdeu, Jose Biller localization in clinical neurology
- [10]. Venugopal K, Kushal D P, Shyamala G, Mohammed M Z, Naik S, Santosh Kumar D P. A stochastic variant of Wallenberg syndrome with ipsilateral central facial palsy



Figure 1

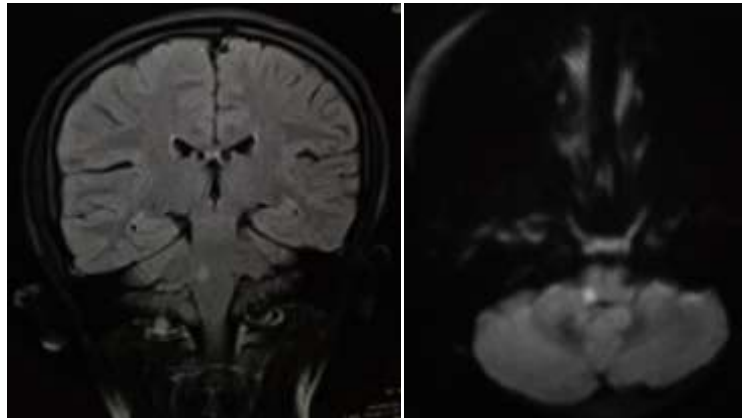


Figure 2,3

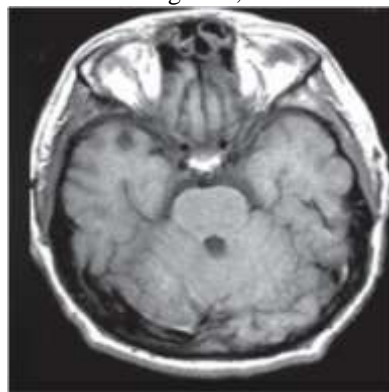


Figure 4

Table 1: Clinical features with the involved structures

Symptoms	Structures affected
1.Pain, numbness and ipsilateral impairment of sensation over face	Descending tract and nucleus of trigeminal nerve
2.Pain and temperature loss over opposite side of the body	Lateral spinothalamic tract
3.Dysphagia, dysarthria, hoarseness of voice Ipsilateral palate, pharynx, vocal cord palsy absent gag reflex	Nucleus ambiguus (9th and 10th cranial nerve)
4.Nausea, vomiting, vertigo, diplopia nystagmus, oscillopsia	Vestibular nucleus
5.Ipsilateral cerebellar signs and symptoms	Inferior cerebellar peduncle
6.Ataxia, falling to one side	Restiform body, cerebellum, spinocerebellar pathway
7. Loss of taste	Nucleus and tractus solitarius
8. Ipsilateral Horner's syndrome	Sympathetic tract involvement
9.Numbness of ipsilateral arm, leg	Gracilis and cuneatus nucleus
10. Hiccups	Dorsolateral region of middle medulla



Table 2: Laboratory and other investigations of the patient

<u>Parameters</u>	<u>values</u>
Total count	8700 cells/cumm
Neutrophils	68%
Eosinophils	05%
Lymphocytes	14%
Platelets	167,000
RBC counts	3.2 lakhs/cumm
ESR	20
Sodium	140 meq/l
Potassium	3.7 meq/l
Chloride	94 meq/l
RBS	128 mg/dl
Blood urea	42 mg/dl
Serum creatinine	0.4 mg/dl
LFT	Normal
LDL	118 mg/dl
HDL	42 mg/dl
TG	110 mg/dl
ECG	Normal
Fundus examination	Normal
MRI brain plain	Infarction in right upper dorsolateral medulla
MRA and MRV	Normal

Legends :

Figure 1 : Ipsilateral LMN facial palsy

Figure 2,3 : Coronal and Axial MRI brain showing right upper dorsolateral medullary infarct

Figure 4 : Axial MRI brain showing normal pons