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CLINICAL PROFILE OF LATERAL MEDULLARY SYNDROME IN A TERTIARY CARE HOSPITAL IN SOUTH TAMILNADU

Neurology

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ABSTRACT

Objectives: To describe the clinical profile of Lateral Medullary Syndrome admitted in Department of Neurology in a tertiary care hospital in South TamilNadu. **Methods:** Patients who satisfied clinical criteria for Lateral Medullary syndrome were recruited consecutively until sample size of 30 was reached over a period of 12 months. They were examined clinically; vascular risk factors assessed, relevant investigations including FBS, PPBS, MRI Brain with MRA were performed. **Results:** Among the LMS patients 90% were males. All of them had infarcts. Diabetes and Dyslipidemia were the most common systemic risk factors. Limb Ataxia was universal. 40% of our patients had ipsilateral facial palsy. Ipsilateral hemiparesis was seen in 50%. The most common sensory pattern observed was Stopford type IV. MRA was normal in 60% of patients. **Conclusion:** LMS presents as a fragmented syndrome. It is more common in males and seen frequently after 50 years. Diabetes and Dyslipidemia are the systemic vascular risk factors frequently associated with LMS. LMS is almost always due to infarct. Small vessel disease is more often the cause. Vertebral artery is the frequently involved large vessel. Ipsilateral facial palsy and ipsilateral hemiparesis was a frequent observation among LMS patients in our population. Stopford type IV sensory pattern was more common than classical Stopford type I in our population. Rare causes of LMS include dolichoectasia, arterial dissection and vascular malformation.

KEYWORDS

lateral medullary syndrome, Wallenberg syndrome, posterior circulation stroke.

INTRODUCTION:

The vertebral arteries (VA), chief arteries supplying the medulla are branches of Ist part of the subclavian artery. They ascend the neck passing through the transverse foramina of the upper six cervical vertebrae, enter the skull through foramen magnum, pierce the meninges to enter the subarachnoid space. At the lower border of pons, they join to form the basilar artery. Each VA supplies the lower three fourths of the pyramid, medial lemniscus, nearly all of the retro-olivary region, the inferior cerebellar peduncle and the postero inferior part of cerebellar hemispheres. The VAs are occluded by atherothrombosis in the intracranial part in 8 out of 10 cases of LMS. Embolism to PICA is less common.

The complete LMS comprises of symptoms derived from (a) vestibular nucleus- vertigo/giddiness, nausea, vomiting, and oscillopsia. (b) spinothalamic tract –contralateral, less often ipsilateral impairment of pain and temperature, (c) descending sympathetic tract-ipsilateral Horner syndrome,(d) issuing fibers of IX,X cranial nerves-hoarseness, dysphagia, hiccough, ipsilateral palatal and vocal cord paralysis, diminished gag reflex,(e) restiform body, inferior cerebellum- ipsilateral limb ataxia, lateropulsion (f) descending tract and nucleus of V nerve-impaired pain and temperature over ipsilateral face ,rarely, (g) utricular nucleus- vertical diplopia, rotation of vertical meridian (h)Nucleus Tractus Solitarius- loss of taste (i) Cuneate and Gracile nuclei- numbness of ipsilateral limbs. Vertigo is the most common stand alone feature. Occlusion of the intracranial vertebral artery can lead to a total unilateral medullary (Babinski-Nageotte) syndrome or a combination of medial and lateral medullary syndromes. Most of the times, the clinical presentation is partial. LMS is almost always caused by infarction; few cases are due to hemorrhage, tumour, demyelination, abscess, aneurysm, AVM.

II. METHODS:

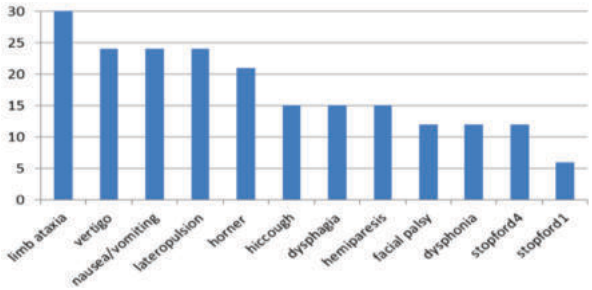
This cross-sectional, descriptive study was conducted in Department of Neurology, Govt Tirunelveli medical college Hospital from January 2022 to Dec 2022 after obtaining approval from the Institutional Ethics Committee. A total of 30 patients were included by consecutive sampling. Both female and male patients, above the age of 18 years, whose clinical presentation was consistent with Lateral Medullary Syndrome, were included, after obtaining informed written consent. All included patients underwent a thorough clinical general and neurologic examination, vital parameters evaluated, relevant blood investigations were done. All of them underwent 1.5T Siemens Magnetom MRI Brain imaging with MRA. The imaging sequences included T1W, T2W, FLAIR, DWI, ADC, and GRE.

Information was collected by the same physician and tabulated in the study proforma. Data were summarized using frequency counts and descriptive statistics.

III. RESULTS:

Among the cases included in the study, there were 27 men and 3women. The patients' ages ranged from 42 to 65 years, average being 55.9 years. The most common systemic conditions assessed in this study were Diabetes Mellitus, Systemic Hypertension and Dyslipidemia. Nine of our cases had all the three vascular systemic risk factors, three had only hypertension, six had only Dyslipidemia, nine had T2DM and Dyslipidemia, three of them did not have any. Among the 27 men participants, eighteen of them were chronic smokers and alcoholics.

The presenting complaint was acute onset of giddiness/ vertigo in 24 of the 30 participants. Nausea +/- vomiting was present in 24 patients. Limb ataxia was observed in all the participants. Lateropulsion was seen in 24 patients. Hoarseness of voice was observed in 12 patients. Dysphagia/palatopharyngeal weakness/ depressed gag reflex/ nasal regurgitation of fluids were observed in 15 patients. Hiccough was observed in 15 patients. Horner syndrome was observed in 21patients. Ipsilateral motor weakness was seen in 15 patients. Ipsilateral facial weakness was seen in 12 patients. None of them had abnormal breathing pattern, dissociative anaesthesia.Three patients had loss of all sensory modalities (spinothalamic+ lemniscal). Twenty one patients had reduced pain and temperature over ipsilateral hemiface; sensory abnormalities restricted only to the face and not involving the trunk were seen in 6 patients. Normal facial pain and temperature sensations were seen in 9 cases. Abnormal pain and temperature sensation of trunk and limbs were seen in 21patients; ipsilateral in 12, contralateral in 6, pure trunk involvement with facial sparing was seen in 3. One patient complained of severe headache and one complained of nuchal pain.



Bar chart 1 showing frequency of different symptoms in LMS

MRI showed infarct in dorso lateral medulla in 21 cases. MRA was normal in 18 cases; abnormal in 12 cases. One of them had VA dissection, one had dolicoectasia of right VA, and one had vascular malformation compressing lateral medulla.

In our study, men were affected predominantly and LMS commonly affected people in the older age group. T2DM was the most common systemic vascular risk factor associated followed by Dyslipidemia. Ipsilateral limb ataxia was universal. Acute vertigo, nausea-vomiting, lateropulsion, partial Horner syndrome, palatopharyngeal weakness, hiccough, ipsilateral motor weakness (Opalski variant), dysphonia, facial weakness was observed in decreasing order of frequency. None of them had contralateral weakness. Stopford type 4 sensory pattern was more frequent followed by type 1. Patient with nuchal pain at onset of stroke had VA dissection.

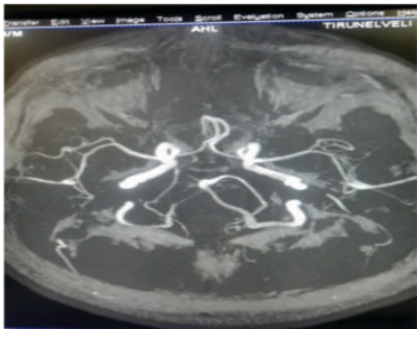


Image 1. MRA showing occlusion of right vertebral artery

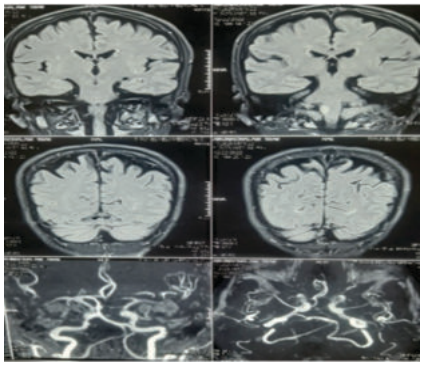


Image 2. MRI showing infarct of right lateral medulla with MRA showing dolichoectasia

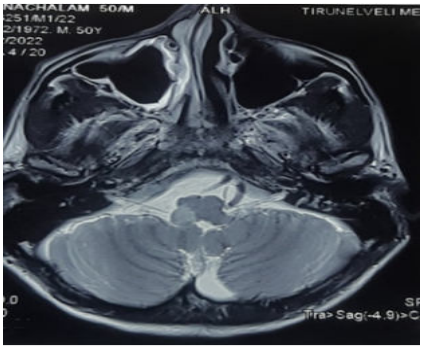


Image 3. MRI showing infarct of right lateral medulla

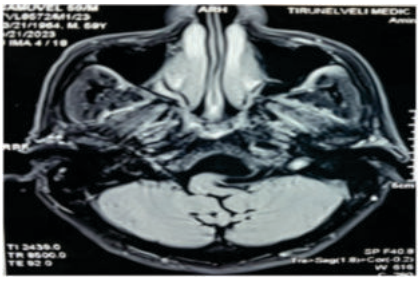


Image 4. MRI showing vascular malformation compressing left lateral medulla

IV.DISCUSSION:

Wallenberg described the lateral medullary syndrome, the most common form of brainstem stroke. Partial resolution and survival is the rule. Dysphagia improves. Patient may have residual dysphonia, ataxia and sensory deficits. Aspiration is a major threat- dysphonia, palatal weakness and facial sensory deficits predict an increased risk.

Unusual manifestations of LMS include include ipsilateral UMN facial palsy due to involvement of Dejerine's aberrant pyramidal tract and does not imply extension of lesion beyond lateral medulla. Hypoalgesia may involve only ipsilateral face or contralateral body[1]. Classical crossed pattern occurs only in minority.

Varied patterns of the sensory abnormality have been described in LMS which is due to the different patterns of medullary involvement that can be either horizontal (ventral, dorsal, and lateral) or vertical (rostral and caudal).[2]

The more rostral and more ventral the lesion, the incidence of contralateral trigeminal nerve involvement simultaneously increases.[3] Sensory loss can also involve touch sensations.

1.Loss of pain and temperature sensation on the ipsilateral face and contralateral body (classical crossed pattern)	Ipsilateral descending spinal tract and nucleus of the trigeminal nerve, as well as the crossed lateral spinothalamic tract.	far lateral medullary lesion	Stopford Type I [4]
2.Only contralateral face, arm, and trunk	The ipsilateral side of the face is spared due to preservation of the descending spinal tract and nucleus of the trigeminal nerve	mediolateral medulla	Stopford type II
3.Bilateral facial hypoesthesia	the crossed trigeminothalamic tract carrying pain and temperature sensation from the contralateral side of the face may be involved	Medial extension	Stopford Type III
4.Sensory loss of contralateral face–arm–trunk–leg with sparing of ipsilateral face [5]		lateral medullary infarction	Stopford Type IV

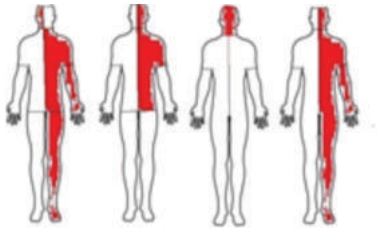


Fig 2. Stopford sensory patterns type I-IV

Zhang *et al*. described five patterns of sensory abnormalities.
Type I: Ipsilateral face and contralateral trunk and limbs; Type II: Ipsilateral face and contralateral face, trunk, and limbs; Type III: Contralateral face and body; Type IV: Ipsilateral face and contralateral trunk and leg; and, Type V: Contralateral face, arm, and upper trunk.[6]

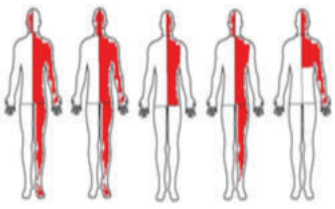


Fig 3. Zhang sensory patterns type I-V

The absence of Horner's syndrome, palatopharyngeal palsy can be explained by the dorso-lateral involvement of the medulla. Dorsolateral lesions tend to be more superficial and hence do not

involve Nucleus Ambiguus, which is located more deeply. Contralateral hemiparesis occurs due to inferior extension of the lesion, to the medullary pyramid prior to decussation. Ipsilateral hemiparesis occurs due to inferior extension to lateral funiculus of rostral spinal cord (Opalski submedullary syndrome). Rarely inferior extension to Gracile & Cuneate nuclei can cause impaired sensation of ipsilateral arm and leg. [7] Hiccups occur due to involvement of dorsolateral aspect of the middle medulla. Involvement of nucleus of solitary tract (NTS), nucleus ambiguus (NA), related reticular formation, Vth cranial nerve nuclei, and their connections with the middle part of the medulla are essential for hiccups to occur.

Other rarer manifestations include proximal arm ataxia, inability to sneeze, paroxysmal sneezing, and Ondine's curse, weakness of sternocleidomastoid, loss of taste, facial trophic ulcers, and persistent facial pain.

CONCLUSION:

LMS presents as a fragmented syndrome. It is more common in males and seen frequently after 50 years. Diabetes and Dyslipidemia are the systemic vascular risk factors frequently associated with LMS. LMS is almost always due to infarct. Small vessel disease is more often the cause. Vertebral artery is the frequently involved large vessel. Ipsilateral facial palsy and ipsilateral hemiparesis was a frequent observation among LMS patients in our population. Stopford type IV sensory pattern was more common than classical Stopford type I in our population. Rare causes of LMS include dolichoectasia, arterial dissection and vascular malformation.

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Conflict of interest: Nil

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