LATERAL MEDULLARY SYNDROME: CLINICO-RADIOLOGICAL CORRELATION

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ABSTRACT

Lateral Medullary syndrome is not a very common disorder and remains undiagnosed at times. It is commonly caused by occlusion of the cranial segment of the vertebral artery or the posterior inferior cerebellar artery. The usual symptoms of this syndrome include vertigo, dizziness, nystagmus, ataxia, nausea and vomiting, dysphagia, hoarseness, hiccups, impaired sensation over half the face, impairment of pain and thermal sensation over the contralateral hemibody and limbs and the ipsilateral face, and Horner's syndrome. Four cases of lateral medullary syndrome are reported who were diagnosed on clinical basis and later on confirmed by MRI. Our objective is to correlate clinical and radiologic findings in patients with lateral medullary syndrome.

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INTRODUCTION

Lateral medullary syndrome (LMS), first described in 1808 by Gaspard vieussux[1], is a well recognized clinical condition due to involvement of vertebral/posterior inferior cerebellar artery. It can now easily be recognized with the advent of MRI. Clinico - radiological correlation of LMS is, however, not well established.

We report a case series of four patients with LMS and try to correlate clinico - radiological findings.

CASE SERIES

First case was a 73 year old male, known diabetic and hypertensive, presented with complaints of headache, vertigo, ataxia, slight decrease sensations in bilateral extremities. MRI on first day of admission revealed tiny high T2 and FLAIR signal abnormalities in left posterolateral medulla in caudal region suspicious for acute infarcts. Repeat MRI on second day revealed that these signal abnormalities became more intensified in comparison to the previous scan (Figure 1).

Second case was a 52 year old male, known hypertensive, presented with complaints of headache, blurring of vision, altered sensation in left face and right side body for 3 days. CT brain immediately done was normal. MRI revealed high T2 signal abnormality in left half of medulla. MR angiogram revealed thrombosed left distal vertebral artery (Figure 2).

Third case was a 32 year old female with complaints of headache, vertigo, dysphagia and altered sensation in left arm. There was no significant past history. MRI revealed low T1 and high T2/FLAIR signal abnormality in dorsolateral side of right half of rostral medulla suggestive of infarct whic was evident an both axial and coranal FLAIR images (Figure 3a and b).

Fourth case was a 52 year old male with complaints of headache, vertigo, dysarthria, balance problems and left leg weakness. MRI revealed hyperintense T2 signal in dorsolateral part of right half of medulla. MRA revealed non-visualization of right vertebral artery.

DISCUSSION

The lateral medullary syndrome (Wallenberg's syndrome) 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).

The syndrome is characterized by sensory deficits affecting the trunk and extremities on the

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Figure 1: FLAIR axial image of posterior fossa revealed tiny high FLAIR signal abnormality in left posterolateral medulla in caudal segment

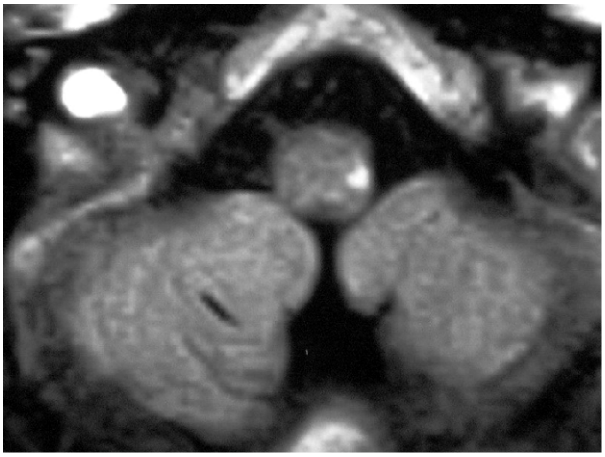


Fig 2: MRA revealed thrombosed left distal vertebral artery

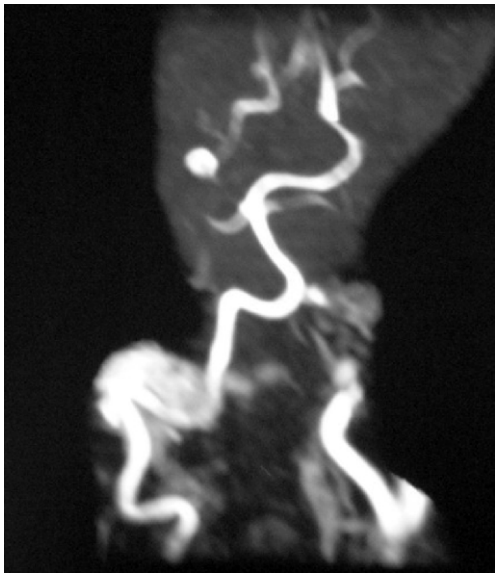


Figure 3a: High FLAIR signal seen in right half of medulla on axial images

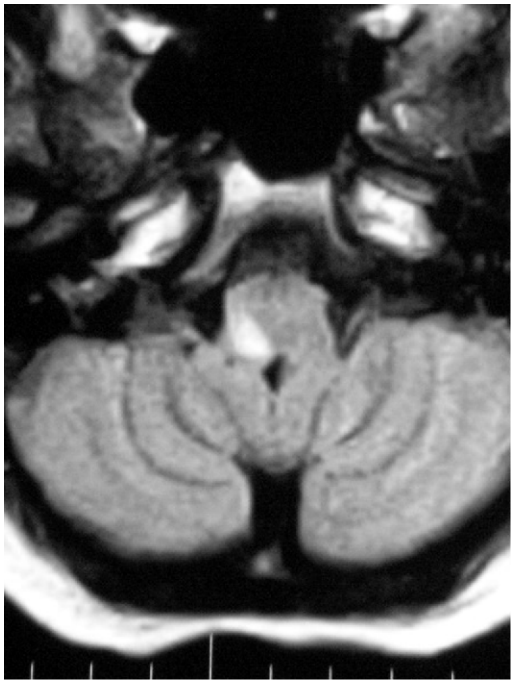


Figure 3b: High FLAIR signal seen on Lateral side of right half of medulla on coronal images in rostral segment

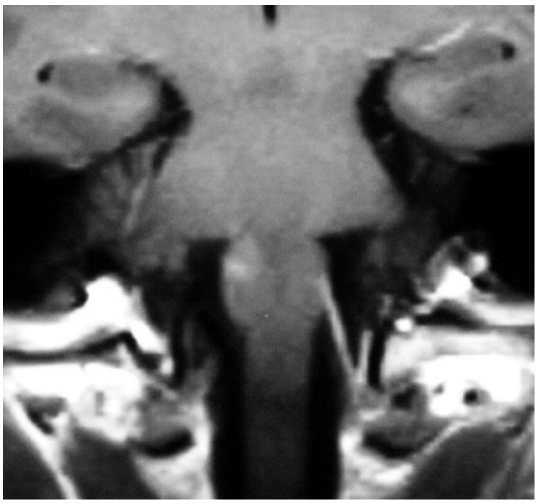


Figure 4a: Rostral Medulla

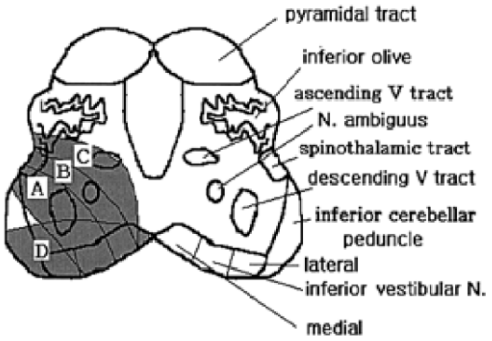
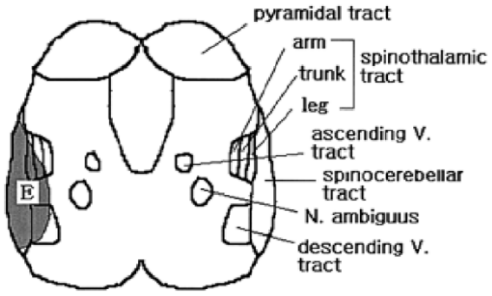


Figure 4b: Caudal Medulla



opposite side of the infarction and sensory deficits affecting the face and cranial nerves on the same side with the infarct. Specifically, there is loss of pain and temperature sensation on the contralateral side of the body and ipsilateral side of the face. The crossed finding is diagnostic for the syndrome. Other clinical symptoms and signs are swallowing difficulties (dysphagia)[2], slurred speech, ataxia, facial pain, vertigo, nystagmus, Horner's syndrome, diplopia and possibly palatal myoclonus. The affected persons in this case series had dysphagia resulting from involvement of the nucleus ambigus as well as dysarthria.

Symptoms of leteral medullary syndrome depends upon the size and location of the areas of brainstem damaged by the stroke (Figure 4a and b). Damage to the spinal trigeminal nucleus causes absence of pain on the ipsilateral side of the face as well as absent corneal reflex.

If spinothalamic tract is damaged, it results in loss of pain and temperature sensation to the opposite side of the body. Damage to the cerebellum or the inferior cerebellar peduncle causes ataxia. Damage to the hypothalamo-spinal fibers disrupts sympathetic nervous system giving rise to Horner's syndrome. Nystagmus and vertigo result from involvement of vestibular nuclei. Onset is usually acute with severe vertigo. Vertigo was also seen in majority of our cases too.

Lateral medullary syndrome is an uncommon stroke which can be diagnosed clinically and confirmed by Head imaging (CT/MRI of the brain) as was done in our case series[3-5]. Among the symptoms and signs, dysphagia is troublesome as in case three and has been reported in 51% to 94%[3]. The outlook for someone with the syndrome depends upon the size and location of the area of the brainstem damaged by the stroke. Some individuals may see a decrease in their symptoms within weeks or months following treatment. Others may be left with significant neurological disabilities for years after the initial symptoms appeared.

In our study, there was predominance in men presenting at 59 years of average age (range 32-73). Hypertension was the main risk factor (50%). There were different clinical findings depending on the site of the lesion, whether rostral, caudal, middle etc. Headache and vertigo were the most frequent symptoms present in all the patients. Vertigo was most likely due to involvement of vestibular nuclei. Two patients had balance problems which were likely due to involvement of inferior cerebellar peduncles as there was no cerebellar lesion. Sensory weakness in ipsilateral face was present in 25% of patients due to involvement of spinal trigeminal nucleus. There was sensory weakness in contralateral body half in 75% of patients which was due to involvement of spinothalamic tract. One patient had dysphagia (25%). There was no horner syndrome in any of the patient. Patients with involvement of rostral medulla had either dysarthria or dysphagia. They all had sensory weakness on contralateral side of body. Patients with involvement of caudal medulla had no dysarthria or dysphagia. There was acute onset of symptoms in all patients. Lateral medullary infarction alone was present in all of four cases with none of the lesion extending beyond the lateral medulla. Vertebral artery disease was confirmed by vascular imaging studies in 75% of patients as it is the most common culprit artery for LMS. Cerebellar lesions were absent in all cases.

Our case series illustrates that the clinical and topographic spectra of LMS are diverse, and MRI analysis in rostrocaudal and dorsoventral aspects allows us, although not unequivocally, to make clinical-MRI correlations.

CONCLUSION

All symptoms of typical lateral medullary syndrome are not necessarily present in each patient. They are largely dictated by the site and extent of the area of involvement.

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CONTRIBUTORS

Both the authors contributed significantly to the collection of cases and writing of the menuscript that resulted in the submitted manuscript.