CASE REPORT
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VARIANT OF LATERAL MEDULLARY SYNDROME

Neurology

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ABSTRACT

Lateral Medullary Syndrome (LMS) is a vascular syndrome affecting the posterior circulation territory. Its localized nature stems from its distinctive presentation, unique blood supply territory, and relatively small area of involvement. We present a case of Wallenberg's syndrome, which deviated from the classic syndrome by lacking Horner's syndrome and exhibiting contralateral facial sensory loss. This case underscores the diverse presentations that can arise from differential involvement of the lateral medulla.

KEYWORDS

Hemiparesis, Lateral Medullary Syndrome, Wallenberg' s, posterior circulation

INTRODUCTION

Approximately 20% of ischemic strokes affect the posterior circulation. Lateral medullary syndrome is the most prevalent type of crossed brain stem syndrome.[1] Wallenberg syndrome was initially described in 1808 by Gaspard Vieusseux. Adolf Wallenberg's clinical descriptions of the syndrome were published in 1895, while autopsy findings were published in 1901, providing a more comprehensive understanding of the condition.[2,3]This syndrome is caused by the infarction of the lateral medulla oblongata due to the occlusion of the vertebral artery or posterior inferior cerebellar artery. Stroke in this region of the brain stem often leads to a range of impairments, including motor, sensory, cognitive, perceptual, speech, and language deficits.

The lateral medulla comprises several structures, including the inferior cerebellar peduncle, vestibular nuclei, trigeminal nucleus and tract, spinothalamic tract, descending sympathetic fibers, nucleus ambiguous, and nucleus solitarius.The classical presentation of lateral medulla syndrome involves crossed sensory deficits, particularly the loss of pain and temperature sensation affecting the trunk and extremities contralateral to the infarct, accompanied by ipsilateral facial numbness. Other features of this syndrome include vertigo, nystagmus, hoarseness, dysphagia, ipsilateral cerebellar signs, and Horner's syndrome.[4]

In most cases, the diagnosis of LMS is made on the basis of classical clinical features. Some authors even recommend delaying the MRI scan when the presentation is classical. However, a wide variation has been recognized in the clinical presentation, which may not be observed most of the time. Therefore, efforts have been made to classify LMS on the basis of the pattern of sensory involvement, anatomical localization, and radiological pattern of involvement.

Case Report

A 44-year-old male with a history of hypertension and coronary artery disease (CAD) presented to the emergency department with the primary complaints of a reeling sensation, headache, slurred speech, and difficulty swallowing. These symptoms began 8 hours ago and have been accompanied by five episodes of vomiting in the past 6 hours. Additionally, the patient reported left ear pain and discomfort in the left facial region.

On examination, the patient's Glasgow Coma Scale (GCS) was 15/15, indicating a normal level of consciousness. Blood pressure was 160/100, and other vital signs were within normal ranges. The motor examination revealed no abnormalities, while the sensory examination showed decreased sensations in the left upper limb, face, and lower limb. Additionally, there was decrease in sensations over the right face. Reflexes were within the normal range.

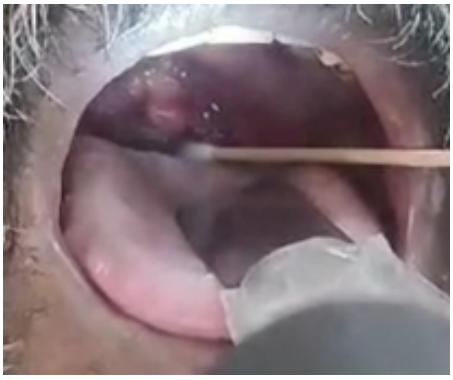

Fig 1: Testing Gag reflex: revealed decreased palatal movement on right side

The patient exhibited ataxia, finger-nose dysmetria, finger-finger dysmetria, horizontal nystagmus, and decreased palatal movement on the right side. During the ABC test, the patient's hearing was less than that of the examiner.

An MRI was subsequently performed and diagnosed as Wallenberg syndrome.

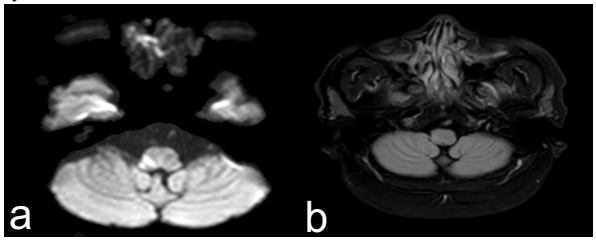

Figure 2: Acute infarct involving right lateral medulla- Area of restricted diffusion in the right lateral medulla measuring approximately 9 x 5 mm, demonstrating hyperintensity on DWI (fig a) and subtle hyperintensity on T2 WI (fig b).

DISCUSSION

Half of the patients considering a diagnosis of Wallenberg syndrome did not meet all the original symptoms described by Wallenberg, and a significant portion had additional clinical features of brain stem

dysfunction. Modern neuroimaging techniques have made it easier to identify rare variants of LMS. Considering the extent of the lesion in the rostral-caudal and lateral-medial directions, six variants have been described based on the sensory pattern of presentation.

Various Patterns of LMS: (Kim JS et al.,MacGowan DJ et al., 1997):[5,6]

1. **Hypalgesia of ipsilateral face and contralateral body -** Lateral spinothalamic tract (c/l body sensory deficit), Descending trigeminal spinal tract and nucleus (Ipsilateral face sensory deficit)
2. **Bilateral facial hypalgesia and contralateral body hypalgesia -** Crossed trigeminothalamic pathway
3. **Pain and temperature sense loss in contralateral face and body -** Trigemino thalamic and lateral spinothalamic tracts
4. Hypalgesia of contralateral body (face spared)
5. **Hypalgesia of ipsilateral face -** Trigeminothalamic tract (body spared)
6. None

More subtle variants of Wallenberg syndrome also consider the implication of proprioceptive and vibration impairment, as well as the pattern of trigeminal territory involvement (i.e., segmental vs. onion skin patterns or incomplete involvement of the three trigeminal branches). A further variant of Wallenberg syndrome that includes ipsilateral hemiplegia (caused by caudal extension affecting the corticospinal fibers after the pyramidal decussation) has been called Opalski syndrome.

Zhang Et Al., Described Five Patterns Of Sensory Impairment:[7]

Type 1 : Ipsilateral face and contralateral trunk and limbs
Type 2 : Ipsilateral face and contralateral face, trunk, limbs
Type 3 : Contralateral face and body
Type 4 : Ipsilateral face and contralateral trunk and leg
Type 5 : Contralateral face, arm and upper trunk

Our patient's presentation deviated from the typical description of Wallenberg's syndrome, particularly in the presence of pain and decreased sensation on the contralateral face, along with the contralateral body, and involvement of the ipsilateral face. Therefore, our patient would be classified as having Type 2 sensory involvement. In clinical and radiologic correlation studies conducted by Kim et al., only 25% of patients exhibited Type 2 involvement, while 26% presented with the classic pattern (Kim JS et al.,2003).[8]

This variation observed may be attributed to the different patterns of involvement of the medulla, classified horizontally as ventral, dorsal, and lateral, and vertically as caudal and rostral (Kim JS et al.,2003)[8] More caudal and dorsolateral lesions tend to present classically (Vrettos A et al.,2013)[9], while more rostral lesions, which tend to be more ventral, present with contralateral trigeminal involvement. The spontaneous facial pain experienced by our patient can be attributed to involvement of a phylogenetically older pain pathway that receives fibers from both the spinothalamic tract and the trigeminal nerve(Currier RD et al., 1971)[10].

CONCLUSION

Lateral medullary syndrome can present in diverse ways, with varying presentations. The absence of a classic pattern shouldn't deter one from diagnosing Wallenberg syndrome. The correlation between clinical presentation and radiological findings can help understand how vascular lesion localization can lead to changes in presentation. Therefore, consider the variations in sensory involvement of Wallenberg syndrome before ruling in or out the diagnosis of Wallenberg syndrome.

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