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AN ATYPICAL PRESENTATION OF BABINSKI-NAGEOTTE SYNDROME

Neurology

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ABSTRACT

Babinski-Nageotte syndrome, a rare variant of lateral medullary syndrome, is characterized by contralateral corticospinal tract involvement. We present the case of a 42-year-old male who demonstrated classic features of left lateral medullary syndrome, including ipsilateral cerebellar signs, involvement of the 8th, 9th, and 10th cranial nerves, and crossed hemianesthesia. However, the presence of atypical finding of ipsilateral upper motor neuron facial palsy along with contralateral pyramidal tract involvement, supports the diagnosis of Babinski-Nageotte syndrome.

KEYWORDS

Babinski-Nageotte syndrome, Lateral medullary syndrome, Wallenberg's Syndrome

INTRODUCTION

Lateral Medullary Syndrome (Wallenberg's Syndrome) is a neurological disorder caused by ischemia or infarction of the lateral medulla oblongata, resulting from the occlusion of the vertebral artery or the posterior inferior cerebellar artery (PICA)[1]. These arteries supply vital regions, including parts of the medulla, cerebellum, and choroid plexus. The syndrome's damage affects the inferior cerebellar peduncle, dorsolateral medulla, spinal trigeminal nucleus, vagus and glossopharyngeal nerve nuclei, descending sympathetic fibers, spinothalamic tract, and vestibular nuclei. The symptomatology involves contralateral loss of pain and temperature sensation in the trunk and limbs, ipsilateral loss of sensations on the face, ipsilateral nystagmus, Horner syndrome, dysphagia, dysarthria, gait ataxia, absent gag reflex, hoarseness[2]. Lateral Medullary Syndrome has multiple variants, requiring careful examination and neuroanatomical correlation. Clinical presentations of brainstem infarcts can vary, and while Wallenberg's Syndrome typically presents with classical features, it may also include atypical manifestations.

Case Presentation

A 42-year-old male, a chronic smoker and alcoholic, presented with a sudden onset of giddiness, vertigo, difficulty swallowing, nasal regurgitation, and slurred speech. By the next morning, he noticed left-sided facial weakness, a tingling sensation on the left side of his face, and was swaying on left while walking. After a few hours, He experienced weakness in his right upper and lower limbs with a subtle reduction in sensation on the right side of his body. His vital signs revealed a blood pressure of 160/88 mmHg, a heart rate of 90 bpm, and a respiratory rate of 14 breaths per minute.

A detailed neurological examination showed intact higher mental functions. Cranial nerve assessment revealed normal pupillary reaction with no extra-ocular muscle involvement but partial ptosis on the left side. There was loss of pain and temperature sensation on the left side of his face, pointing to trigeminal nerve involvement. He had left-sided upper motor neuron facial weakness, evident by the loss of naso-labial folds with preserved forehead wrinkling. A decreased gag reflex and palatal weakness on the left side suggested involvement of the glossopharyngeal and vagus nerves. There was an abnormal vestibule-ocular reflex on left which was attributed to the involvement of vestibular nuclei. His muscle bulk and tone were normal, power of right upper and lower limb was reduced to 4/5 with extensor plantar on right side. Additionally, there was a slight decrease in pain sensation on the right side of his trunk and limbs, while other sensations remained intact. His gait was broad-based with a tendency to sway to the left, although other cerebellar signs were absent. There was no bowel or bladder involvement. The above clinical evaluation corresponded to lateral medullary syndrome. MRI was suggestive of acute infarct of size 7.4 x 4.0mm in the left medulla on diffusion scan (Fig. 1). MR Angiography of the brain revealed atherosclerotic irregularity in the left vertebral artery with 60% short segment narrowing in distal left vertebral artery.

The patient was initiated on antiplatelet therapy with aspirin and a high-dose statin. Nutritional support was provided via nasogastric tube feeding. Over the following 2 months, he showed gradual recovery, with only mild residual facial weakness persisting.

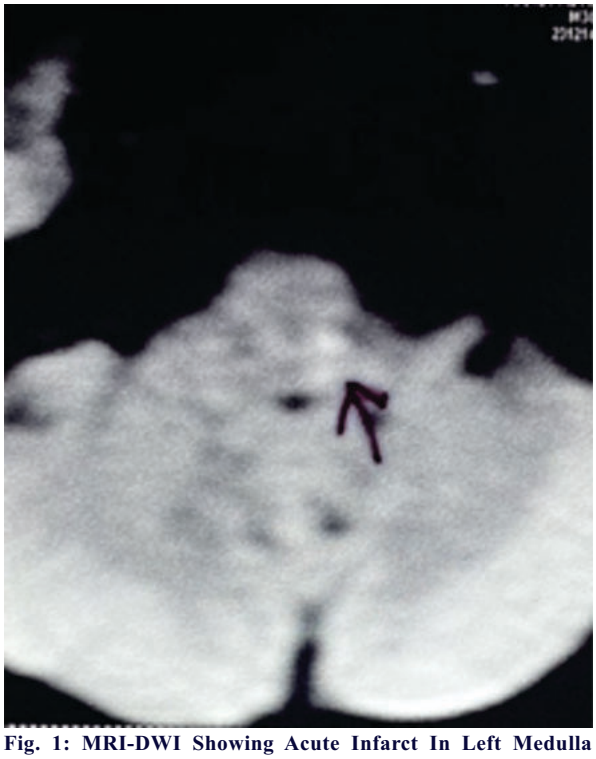

Fig. 1: MRI-DWI Showing Acute Infarct In Left Medulla Measuring 7.4 X 4.0mm In Size.

DISCUSSION

The majority of symptoms in our case align with the classic presentation of lateral medullary syndrome, including ipsilateral cerebellar signs, ipsilateral involvement of the vestibular nucleus, 9th and 10th cranial nerves, and crossed hemisensory deficits. However,

the atypical features in our case that challenged the diagnosis of a straight forward Lateral Medullary Syndrome were as follows:

- Contralateral hemiparesis
- Ipsilateral UMN facial palsy

Babinski-Nageotte Syndrome (BNS) is a variant of LMS which is characterized by contralateral hemiparesis which occurs due to caudal extension of infarct causing involvement of pyramidal tracts before decussating at the medulla[3,4]. In this case, right sided weakness with extensor plantar is suggestive of involvement of corticospinal tract.

Facial nerve involvement is rare in lateral medullary syndrome. Nonetheless, ipsilateral facial weakness can occur due to ischemia affecting the caudal portion of the 7th nerve nucleus, situated near the nucleus ambiguus. In this instance, ipsilateral UMN-type facial palsy may be explained by disruption of the hypothetical looping supranuclear corticobulbar fibers, which descend through the contralateral ventromedial medulla, decussate at the upper medulla, and then ascend in the dorsolateral medulla to reach the facial nerve nucleus (Fig. 2)[5,6].

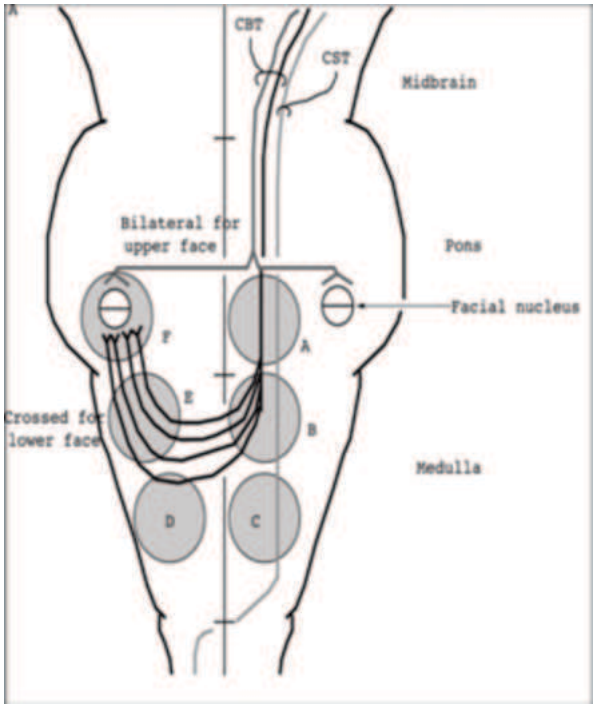

Fig.2: Diagram Illustrating The Abnormal Corticobulbar Nerve Fibers Of The Facial Nerve.[6]

It is well established that the vertebral artery is most commonly implicated in LMS[2]. This was consistent in our case as well, with MRI findings revealing vertebral artery involvement due to atherosclerosis.

The clinical scenario points to an atypical presentation of lateral medullary syndrome, characterized by involvement of the contralateral corticospinal tract and ipsilateral UMN facial palsy. These findings align with Babinski–Nageotte syndrome, suggesting neuroanatomical variations. The neuroanatomical correlation supports the diagnosis of Babinski–Nageotte syndrome with UMN-type facial palsy. Therefore, clinicians must remain alert to the possibility of atypical presentations in brainstem strokes. Hence, a detailed neuro-clinical examination is crucial for accurately localizing brainstem syndromes and recognizing their clinical variants.

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