Case Report Open Access

Opalski Syndrome

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Abstract

Lateral Medullary Syndrome (LMS) is a well-documented vascular syndrome of the posterior circulation territory. This syndrome is easily localized because of characteristic presentation, unique territory of blood supply and very small area of involvement. We present a case of Wallenberg's syndrome which did not have all the classical components of the syndrome, like Horner's syndrome. It was diagnosed as Opalski syndrome, which is a rare variant of Wallenberg syndrome, where lateral medullary syndrome is associated with ipsilateral hemiparesis. This case report highlights how differential involvement of the lateral part of medulla can result in varied presentation.

Background:

Lateral medullary infarction (Wallenberg syndrome) is a relatively common vertebrobasilar vascular syndrome.
However, ipsilateral hemiparesis as part of lateral medullary infarction is rare, and is known as Opalski's syndrome. Some pathologic and neuroradiologic reports have shown that the lesion is located lower than in Wallenberg syndrome, and the ipsilateral hemiparesis seen in this syndrome is attributed to the involvement of corticospinal fibers caudal to the pyramidal decussation.[1] However, Opalski's syndrome with cerebellar lesion is rare.
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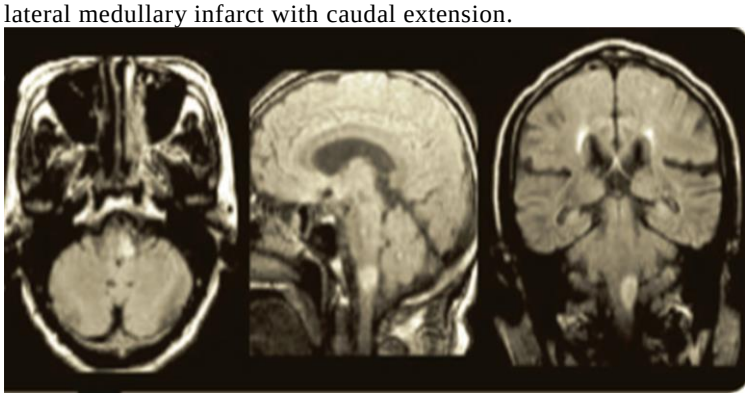
The case description:

A 61-year-old lady presented to the outpatient clinic complaining of difficult swallowing food and walking along with mild weakness in her left hand.
She was watching television when she started to have dizziness. When she went to her room to lay down she developed severe vertigo and her daughter noticed slurring in her speech. She attributed her symptoms to sleepiness and slept. Next morning, she started to notice choking when she drank water and difficulty in standing and walking, along with mild weakness in her left hand. There was no associated facial weakness, difficulty in chewing or moving the food bolus in the mouth or any evidence of tongue weakness. She couldn't account on discrepancy in sensation between the two sides. There was no urinary incontinence, seizures, visual difficulty, diplopia or ptosis. There was no evidence of headache or vomiting and there had been no fever during the period of illness.

She had a past history of transient visual loss two months back which was bilateral and recovered within a few mins. She was right handed, illiterate and known diabetic on insulin mixtard 35 units in the morning and 20 units in the evening but her blood glucose was rarely controlled, and she was also hypertensive on Lisinopril and was controlled on it. She was diagnosed 2 months ago with depression and was prescribed citalopram 10 mg without noticed improvement. There was family history of diabetes and hypertension in both her parents and her father had died of a myocardial infarct when he was 50 years old.
When we examined the patient, she had a pulse rate of 96/min and was afebrile. She had a blood pressure of 150/90 mm Hg in supine position and a respiratory rate of 14/min. She was conscious and oriented. She had dysarthria with a nasal tone and hoarse voice. Her cranial nerve examination revealed a left sided Horner's with miosis and absent sweating on left side face. She had a gaze evoked nystagmus with fast phase to left side. The uvula was deviated to right side and there was absent gag on left side suggestive of IX and X nerve palsy of left side.
The motor system examination revealed normal bulk of all muscles. The tone was reduced in the left side with

mild hypotonia in left lower limb. There was weakness of left half of the body and power was 4/5 in left upper limb and 2/5 in left upper limb. There was grip, as well as foot weakness on the left side. The reflexes were brisk on the left side. The planter response was extensor on left and flexor on right. He had sensory loss over right half of body which included loss of pain and temperature sensation with loss of pin prick by 80% and left sided numbness and impaired sensation over face. There was in coordination in the left upper and lower limbs which was more than could be attributed to weakness suggestive of cerebellar dysfunction. There were no involuntary movements and neck was soft.

The CT of the patient was normal. The MRI revealed an acute left lateral medullary infarct with caudal extension.



Her carotid artery Doppler revealed non occlusive atherosclerosis and her echo revealed hypertensive hear disease with dilated left atrium (55 mm) and an atrial thrombus.

We had this 61-year-old patient with a past history of type 2 diabetes mellitus, hypertension, depression and TIA with sudden onset vertigo, dysarthria and dysphagia and left sided Horner's syndrome and hemisensory loss for pain and temperature on right half of body and sensory loss over left half of face which was characteristic of lateral medullary syndrome. However, she also had a left side weakness with hyperreflexia and extensor planter response because of which the diagnosis of Opalski syndrome was made

Discussion:

In the original description by Opalski in 1949, two patients with lateral medullary infarcts of undetermined etiology were described with mild hemiparesis and same sided hyperreflexia and Babinski's sign, along with features of the lateral medullary syndrome. [1]

The cause of weakness in lateral medullary infarcts is controversial. In his original description, Opalski attributed the weakness to the extension of the ischemia from the lateral medulla to the upper cervical cord involving corticospinal fibers caudal to pyramidal decussation. He also considered that the ischemia was due to additional implication of the posterior spinal artery. The other possible explanations have been given by the work of Liu and his colleagues who considered that the motor deficit may be as a result of the compromised medullary penetrating arteries which arise from the distal vertebral artery or the anterior spinal artery and supply the pyramidal fibers below the decussation [2]. According to Dhamoon and associates, the pyramidal fibre involvement may be due to regional perfusion failure of the border zone area in the spinal cord which lies between the anterior and posterior spinal arteries which and may be involved as the result of a hemodynamic alteration due to vertebral artery stenosis or occlusion.[3]

Opalski's syndrome is not merely a stroke syndrome which challenges the preformed notions about the presentation of lateral medullary infarcts, it also enhances the understanding of vascular lesion localization and how involvement of surrounding structures may lead to changed presentations.

References

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