

Opalski's syndrome: A rare variant of lateral medullary syndrome

Sir,

Lateral medullary (or Wallenberg's) syndrome is characterized by vertigo, diplopia, dysarthria, Horner's syndrome, numbness (ipsilateral face and contralateral limb) and traditionally it is not associated with any limb weakness. Localization in this syndrome is easy because of characteristic presentation, exclusive blood supply and very small area of involvement. However in Opalski syndrome^[1] and Babinski-Nageotte syndrome,^[2] lateral medullary syndrome is associated with hemiplegia. In Opalski syndrome hemiplegia is ipsilateral due to the extension of the infarct caudally to involve the corticospinal fibers after the pyramidal decussation.^[1] In Babinski-Nageotte syndrome^[2] there is contralateral hemiparesis because pyramidal tract is affected before decussation.

We report a case of a 62-year-old man who presented to us with a history of sudden onset vertigo, difficulty in speaking and swallowing with left sided weakness and marked gait ataxia.

The patient was sitting around the bonfire to beat the extreme cold when he started to have dizziness and

urged his wife to take him inside the house. When he came in and lay down he developed severe vertigo and his relatives noticed difficulty in understanding his speech with hoarseness of voice. When offered water he had choking sensation with nasal regurgitation of the water. He later felt weakness on left half of body and could not stand without support. There was no associated facial weakness, difficulty in chewing or moving the food bolus in the mouth or any evidence of tongue weakness. There was no unconsciousness but there was alteration of sensorium transiently at the onset of symptoms. 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.

He had a past history of transient visual loss two months back which was bilateral and recovered within a few mins. He had a CT scan done at that time which was normal. He had been a chronic smoker and smoked around 24 bidis a day for more than 30 years. He was right handed, educated till class VIII and had no history suggestive of Diabetes mellitus, hypertension or heart disease. There was no history of stroke or risk factors in any family members. Prior to admission at our center he had received antibiotics empirically as he had some aspiration during trials of feeding.

When we examined the patient, he had a pulse rate of 96/min and was afebrile. He had a blood pressure of 150/90 mm Hg in supine position and a respiratory rate of 14/min. He was conscious and oriented. He had dysarthria with a nasal twang and hoarse voice. His cranial nerve examination revealed a left sided Horner's with miosis and absent sweating on left side face. His visual acuity fields and fundus were normal. He had a gaze evoked nystagmus with fast phase to left side and a torsional component more pronounced on looking to left side. The

uula 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 more marked 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 lower 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 MRI of the patient [Figure 1a-c] was done which revealed an acute left lateral medullary infarct with caudal extension. His Carotid and Vertebral artery Doppler revealed non occlusive fibro-fatty plaques and his trans-oesophageal echocardiography was normal.

He developed respiratory distress on the second day of admission and required mechanical ventilation. He developed Pneumonia and the cultures suggested Klebsiella and citrobacter infection. He later developed septicemia and multi-organ dysfunction and died after 12 days of admission in spite of intensive care and antibiotic treatments.

We had this 62-year-old patient with a past history of stroke 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

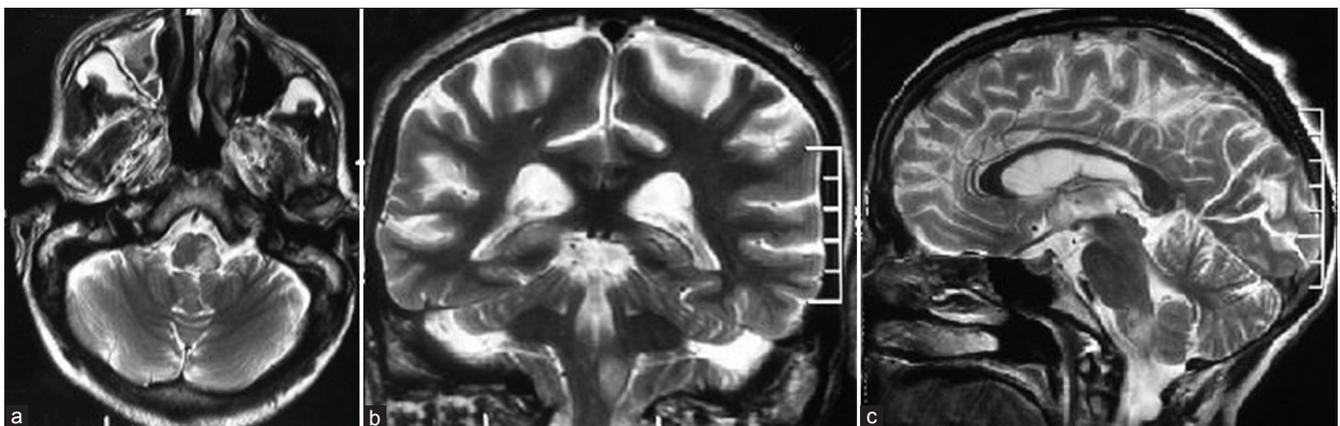


Figure 1: Magnetic resonance imaging (MRI) T2WI axial, (a), coronal (b) and sagittal (c) sections showing acute left lateral medullary infarct with caudal extension

of body and sensory loss over left half of face which was characteristic of lateral medullary syndrome. However, he also had a left side weakness with hyperreflexia and extensor planter response because of which the diagnosis of Opalski syndrome was made.

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] In 1954 Cywinski^[3] and colleagues described two more patients of the similar type due to atherothrombosis and they had selective facial sensory loss of V1 and V2 in one patient. Around 30 years later, Dhamoon and co-workers reported a case with severe weakness ipsilateral to the lateral medullary syndrome following vertebral artery occlusion.^[4] Other descriptions have been provided by Hommel,^[5] Montaner,^[6] Kimura,^[7] Sanahuja,^[8] and respective co-workers.

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 Liu *et al.*,^[9] 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. This may also explain the differential weakness manifesting as monoparesis similar to our case that had more marked lower limb weakness. According Dhamoon *et al.*, 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.^[4]

A large study of lateral medullary syndromes including 130 patients by Kim JS^[10] identified seven patients who had mild ipsilesional hemiparesis without reflex abnormalities and Babinski's sign. Kim questioned whether the weakness was attributed to a pyramidal lesion, or represented a spinocerebellar hypotonic hemisyndrome. However, Hermann *et al.*,^[11] have correctly pointed out that hyperreflexia and Babinski's sign have rarely been described since Opalski's description except once.^[8] They have suggested that eponym Opalski's syndrome should be reserved for patients presenting with

ipsilesional hemiparesis, hyperreflexia and Babinski's sign, and contralesional hypothermalgesia.

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. It also emphasizes that in presence of cerebellar involvement when there is doubt about the co-existing pyramidal involvement, the presence of Babinski's sign and hyperreflexia help to ascertain the corticospinal involvement and differentiate it from cerebellar hypotonic hemisyndrome.

Sanjay Pandey, Amit Batla

Department of Neurology, RN. 507, GB Pant Hospital, Delhi, India

Address for correspondence:

Dr. Sanjay Pandey,
Department of Neurology, RN. 507, GB Pant Hospital, Delhi, India.
E-mail: sanjaysgpgi2002@yahoo.co.in

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