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Opalski syndrome: A rare presentation of Wallenberg syndrome

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Abstract
Lateral Medullary Syndrome (LMS) is a well studied vascular syndrome of the posterior circulation territory. This syndrome is localised with its typical presentation including vertigo, nystagmus, hoarseness, dysphagia, ipsilateral cerebellar signs and Horner's syndrome with classical crossed sensory deficits, specifically loss of pain and temperature sensation affecting trunk and extremities contralateral to the infarct along with ipsilateral facial numbness. We present a case of Opalski syndrome which is a rare variant of Wallenberg syndrome, where lateral medullary syndrome is associated with ipsilateral hemiparesis and positive Babinski sign. This case report showcases one of the varied presentation of ipsilateral weakness in Lateral Medullary Syndrome or Wallenberg syndrome.

Keywords: Lateral Medullary syndrome, hemiparesis

Introduction
We present the case of 70 year old male who has history of coronary artery disease and systemic hypertension now presented with vomiting, vertigo and numbness over right side of his face. He also complained of weakness of right upper and lower limb with swaying towards right side on attempted walking. He gives history of regurgitation on swallowing feeds. He was a chronic smoker with smoking index 300 and has quit smoking for past one year. No history of diabetes mellitus, dyslipidaemia, thyroid disorders. No history of alcohol addiction or high risk behaviour. He was on antiplatelets and antihyprtensives for past one year and has no drug default.

On general examination patient had BP-160/100 mm Hg in right upper limb in supine position with other vital parameters within normal limits. His cardiovascular, respiratory and abdominal examination were unremarkable. On detailed neurological examination higher mental functions were normal. Cranial nerve examination revealed right sided ptosis, miosis and nystagmus with dysaesthesia over right half of the face. Motor system examination showed right side hemiparesis with extensor plantar reflex. Sensory system examination revealed decreased perception of pain and temperature over the left half upper limb. He also had truncal ataxia and positive cerebellar signs on right side.

Routine blood profile was within normal limits. ECG showed T inversions in leads V4-V6 with normal trop I results and ECHO with good LV function Blood sugars, lipid profile, thyroid profile, liver function, renal function, chest radiograph were within normal limits. Screening test for Corona virus with Rapid antigen, HIV and syphilis were all negative.

Magnetic resonance imaging (MRI) brain revealed an infarct in the dorsolateral aspect of the right side of medulla (Fig 1, Fig 2) consistent with lateral medullary syndrome and infarct in right cerebellar hemisphere with small vessel ischemic changes-FAZEKA -2. Magnetic resonance angiogram (Fig 3) including neck vessels were normal with normal venogram.

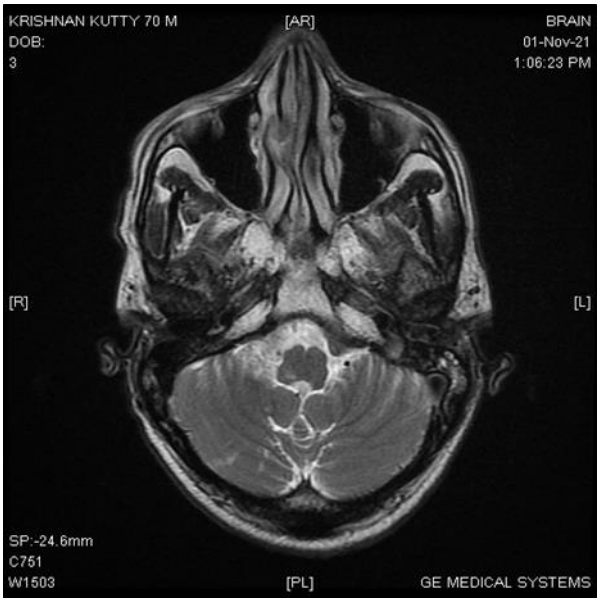

Fig 1: Infarct right medulla

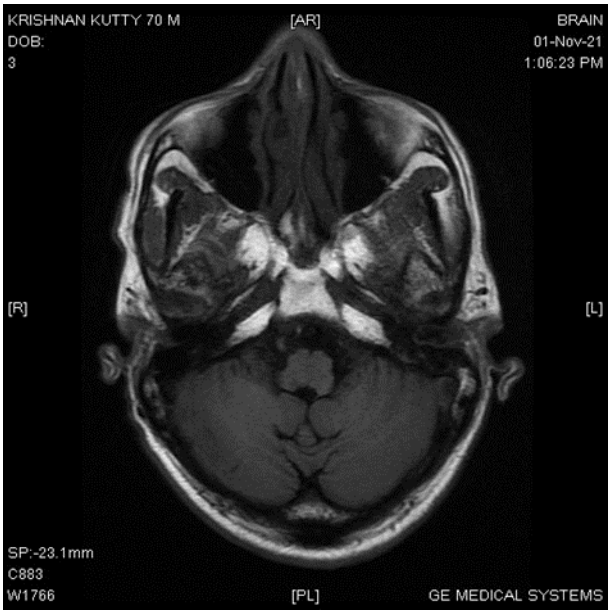

Fig 2: MRI -Infarct right medulla extension

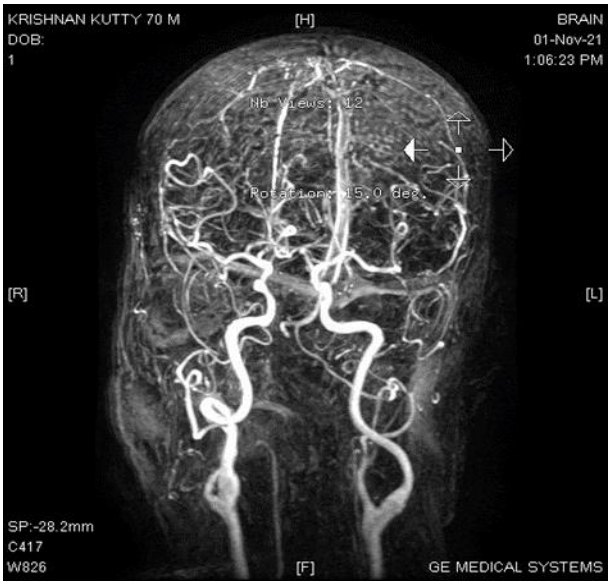

Fig 3: MRA with normal vessels

Discussion

The cause of weakness in lateral medullary infarcts is controversial. In the original article by Opalski [1] in 1949, two patients with lateral medullary infarcts of undetermined etiology were described having mild hemiparesis and ipsilateral hyperreflexia and Babinski's sign, along with features of the lateral medullary syndrome. He also considered that the ischemia was due to additional involvement of the posterior spinal artery The other possible explanations as postulated by Liu *et al*., [3] where the motor deficit may be as attributed to 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 can explain the differential weakness that is seen in some case reports. In Opalski syndrome hemiplegia is ipsilateral due to the extension of the infarct caudally to involve the corticospinal fibers after the pyramidal decussation. In Babinski-Nageotte syndrome [2] there is contralateral hemiparesis because pyramidal tract is affected before decussation. Hermann *et al*., [4] have pointed out that hyperreflexia and or Babinski's sign have rarely been described since Oplaski's description except once. So these cases are supposedly rare. Though we find few more cases like one with variable weakness [5] now, these variants are still rarely encountered in clinical practice.

Conclusion

Lateral Medullary Syndrome has varied clinical presentations depending on the extent of ischemia. Ischemia could be due to the involvement of medullary penetrating arteries and the extension of the infarct caudally involving the coticospinal tracts post decussation. These cases reiterate the need for meticulous clinical examination in all cases and to then correlate the clinical findings with radiological evidence to explain the clinical scenario rather than relying on imaging modalities alone. These cases widen the horizon of clinical medicine.

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