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Case Report

Opalski's syndrome: a rare variant of Wallenberg's syndrome

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ABSTRACT

A 54-year-old, right-handed male presented with acute-onset severe headache, vertigo, and vomiting. Initial neurologic examination illustrated dysarthria (lingual), nystagmus (horizontal), left dysmetria on finger-to-nose testing, and weakness of the left upper and lower limb. Magnetic resonance imaging showed left lateral medullary infarction (Wallenberg syndrome). The patient was discharged 3 weeks later to an inpatient treatment with neurorehabilitation facility with gradual improvement of his symptoms.

Keywords: Opalski syndrome, Lateral medullary syndrome, Wallenberg syndrome, Pyramidal tract

INTRODUCTION

Lateral medullary syndrome (LMS) is a vascular syndrome of the posterior circulation territory. This syndrome is localised with its typical presentation such us 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.[1] We present a case of Opalski syndrome which is a rare variant of Wallenberg syndrome, where LMS is associated with ipsilateral hemiparesis and positive Babinski sign.[1] This case report showcases one of the varied presentation of ipsilateral weakness in LMS or Wallenberg syndrome.

CASE REPORT

We present the case of 54-year-old male known hypertensive on medications, now presented with vomiting, vertigo and numbness over the right side of his face. He also complained of weakness of the left upper and lower limb. He also gave history of swaying towards the left side on walking. History of difficulty while swallowing to both liquids and solids. No history of diabetes mellitus, dyslipidemia, thyroid disorders. He was a chronic smoker with smoking index 300 and has quit smoking for the past one year. He is a non-alcoholic and no history of any other substance abuse.

On general examination patient had BP-160/100 mm Hg in the 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 the left half of the face, cranial nerve IX-uvula deviated to right, left palatal arch movements decreased, hoarseness of voice present, CN-X: palatal and pharyngeal reflex Absent. Motor system examination showed left side hemiparesis with extensor plantar reflex. Sensory system examination revealed decreased perception of pain and temperature over the right half upper limb. He also had truncal ataxia and positive cerebellar signs on the left side. Routine blood investigations was within normal limits. Blood sugars, lipid profile, thyroid profile, liver function, renal function, chest radiograph were within normal limits. Screening tests for Coronavirus with rapid antigen, HIV and syphilis were all negative. Magnetic resonance imaging (MRI) brain revealed left lateral medullary infarction.

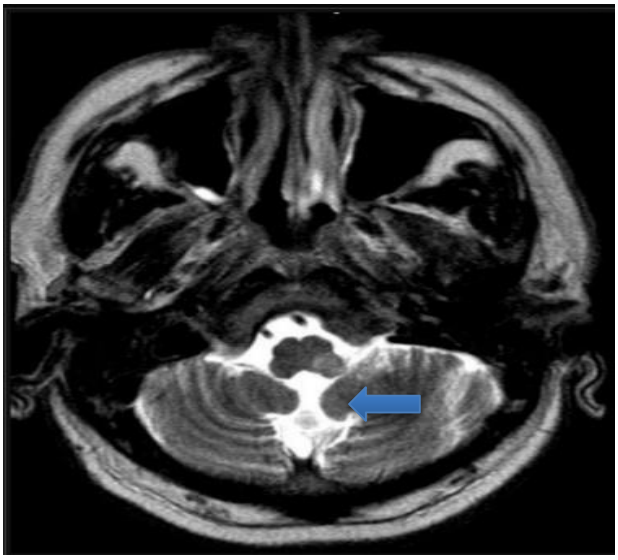

Figure 1: T2 weighted image showing hyper-intense signal in left medulla.

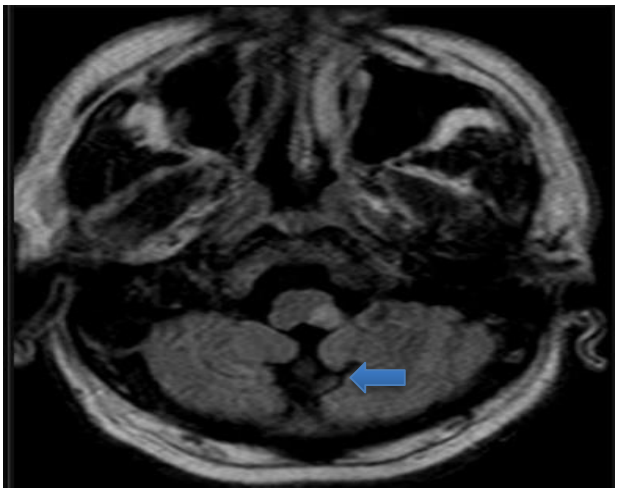

Figure 2: T2 flair image shows hyper-intense foci in left medulla.

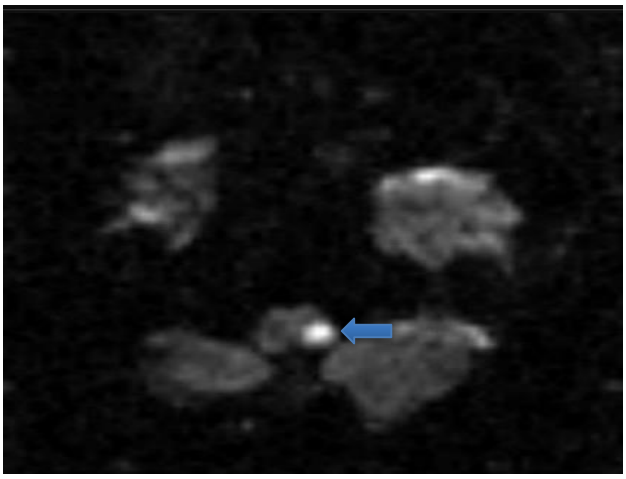

Figure 3: DWI shows diffusion restriction in left medulla.

DISCUSSION

The cause of weakness in lateral medullary infarcts is controversial. In the original article by Opalski 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 LMS.[1] 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. 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.[2] This can explain the differential weakness that is seen in some case reports.[2] In Opalski syndrome hemiplegia is ipsilateral due to the extension of the infarct caudally to involve the corticospinal fibers after the pyramidal decussation.[3] In Babinski-Nageotte syndrome (Hemi medullary syndrome) there is contralateral hemiparesis because the pyramidal tract is affected before decussation.[3] Hermann et al have pointed out that hyperreflexia and or Babinski's sign have rarely been described since Oplaski description except once.[4] So, these cases are supposedly rare. Only a few cases of Opalski's sub-bulbar syndrome noted in the literature since 1946.[5] Though we find few more cases like one with variable weakness now, these variants are still rarely encountered in clinical practice.[6] Atherosclerosis is most common cause of this syndrome.[7] Atherothrombotic occlusion of vertebral artery is said to be the most common etiology for wallenberg syndrome, also called PICA syndrome.[8]

CONCLUSION

LMS 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 corticospinal 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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