Journal Homepage: -www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/21194
DOI URL: http://dx.doi.org/10.21474/IJAR01/21194

RESEARCH ARTICLE

OPALSKI SYNDROME: A RARE VARIANT OF WALLENBERG SYNDROME

Ouardaoui Adnan, Touchanti Ouafae, El Qsimi Imad Eddine, Akkaoui Fatima, Ghomari Khayat Omar, Elhammichi Amine, Azenag Amina, Jilla Mariam and Belfkih Rachid

Manuscript Info

Manuscript History
Received: 16 April 2025
Final Accepted: 19 May 2025
Published: June 2025

Key words:-
Opalski Syndrome, Wallenberg Syndrome, Ipsilateral Hemiparesis, Vertebrobasilarinfarct

Abstract

Opalski syndrome, a rare variant of lateral medullary syndrome (Wallenberg syndrome), is characterized by the extension of ischemic injury into the corticospinal fibers below the pyramidal decussation, resulting in ipsilateral hemiparesis. This condition typically arises from vertebral artery occlusion, atherosclerosis, or other vascular etiologies. It presents with a combination of crossed sensory deficits, cerebellar dysfunction, and cranial nerve abnormalities. Advanced imaging techniques, particularly diffusion-weighted MRI, play a critical role in its diagnosis by identifying characteristic medullary and spinal cord infarctions. Management typically involves antiplatelet therapy and risk factor modification to prevent recurrent ischemic events. Increased awareness of Opalski syndrome's clinical and radiological profile is essential for accurate diagnosis and optimal patient outcomes. Herein, we describe a case of a patient presenting with a lateral medullary infarction complicated by ipsilateral hemiparesis, leading to a definitive diagnosis of Opalski syndrome.

Introduction:-

Lateral medullary syndrome (Wallenberg syndrome) is a well-established neurological disorder attributed to ischemia in the vertebrobasilar vascular system. Although commonly associated with crossed sensory deficits and cranial nerve dysfunction, its association with ipsilateral hemiparesis, termed Opalski syndrome, is rare[1]. This condition occurs when ischemia extends to involve the corticospinal fibers below the pyramidal decussation. We present a case of a patient admitted with a lateral medullary infarction and ipsilateral hemiparesis, subsequently diagnosed with Opalski syndrome.

Case Presentation:

A 51-year-old woman presented to the emergency room with sudden onset of vertigo, unsteady gait, and left-sided ptosis. She had a medical history of arterial hypertension and type 2 diabetes mellitus managed with oral hypoglycemics. Neurological examination revealed left facial hypoesthesia, left-sided cerebellar ataxia, and right-sided hemiparesis (muscle strength graded 4/5), with a National Institutes of Health Stroke Scale (NIHSS) score.of 4.Additionally, hyperreflexia and a positive Babinski sign were observed on the right side. No significant abnormalities were noted in vital signs or sensory examination.

Magnetic resonance imaging (MRI) of the brain demonstrated an infarct in the left lateral medulla extending to the cervical spinal cord, consistent with Opalski syndrome, visualized on diffusion-weighted MRI and FLAIR

Corresponding Author:-Ouardaoui Adnan

sequences. Electrocardiography, echocardiography, and Doppler ultrasonography of the carotid and vertebrobasilar arteries were unremarkable. Blood tests revealed elevated low-density lipoprotein (LDL) cholesterol (1.5 g/L), suggesting underlying atherosclerosis.

Management involved conservative treatment with aspirin, clopidogrel, and atorvastatin. Physical therapy was initiated to address motor deficits. The patient demonstrated significant clinical improvement at discharge and achieved a NIHSS score of 0 at a 14-day follow-up.

Discussion:-

Opalski syndrome represents a unique and rare variant of lateral medullary syndrome (LMS) with distinct clinical and anatomical features. First described by Adam Opalski in 1946[2], this condition is characterized by the extension of ischemic damage from the lateral medulla to the corticospinal tract below the pyramidal decussation, resulting in ipsilateral hemiparesis. While the classical features of LMS, such as contralateral sensory deficits and ipsilateral cranial nerve dysfunction, are often present, the additional motor deficits differentiate Opalski syndrome from other brainstem syndromes[3].

The pathophysiology of Opalski syndrome typically involves occlusion of the vertebral artery or its branches, including the posterior inferior cerebellar artery (PICA) and the anterior spinal artery. Atherosclerosis is the most frequently implicated etiology, but other causes such as vertebral artery dissection, cardioembolic events, and small vessel disease have also been documented. In a review by Sacco et al., lateral medullary infarctions were frequently associated with large-artery atherosclerosis, emphasizing the need for aggressive vascular risk factor management in these patients[4].

Imaging studies, particularly MRI with diffusion-weighted imaging (DWI), are pivotal in identifying the characteristic infarction patterns in Opalski syndrome. DWI highlights acute ischemic changes, while FLAIR imaging can provide complementary information regarding the extent of medullary and spinal involvement. In some cases, angiographic studies may reveal underlying vertebral artery dissection or other vascular abnormalities[4,5].

Clinically, Opalski syndrome is distinguished by ipsilateral hemiparesis, a feature absent in classical LMS[6]. Additional findings may include ipsilateral ataxia, cranial nerve palsies, and cerebellar dysfunction. The presence of a Babinski sign or hyperreflexia on the ipsilateral side further supports the diagnosis by indicating corticospinal tract involvement. Zhang et al. have highlighted the variability in sensory deficits associated with lateral medullary infarction, ranging from classic crossed sensory loss to more localized patterns[3,7]. In our case, the ipsilateral hemiparesis was a defining feature that guided the diagnostic process.

Differential diagnoses for Opalski syndrome include other brainstem syndromes such as Babinski-Nageotte syndrome and Cestan-Chenais syndrome. While Babinski-Nageotte syndrome shares some overlapping features, such as hemiparesis and crossed sensory deficits, it differs in the anatomical localization of the lesion. Cestan-Chenais syndrome primarily involves the medulla and cervical spinal cord but is often associated with additional corticobulbar symptoms[4].

Management of Opalski syndrome revolves around addressing the underlying vascular etiology and preventing recurrent ischemic events. Antiplatelet agents, such as aspirin and clopidogrel, remain the cornerstone of treatment. Statin therapy is indicated for patients with atherosclerosis or dyslipidemia. In cases where vertebral artery dissection is identified, anticoagulation may be considered, although its role remains debated in the context of posterior circulation stroke. Rehabilitation, including physical and occupational therapy, plays a crucial role in improving functional outcomes[3,8].

The prognosis for patients with Opalski syndrome varies depending on the extent of the infarction and the timeliness of intervention. Early recognition and management are associated with better functional recovery, as evidenced by the significant improvement observed in our patient. Long-term follow-up is essential to monitor for recurrent strokes and to optimize secondary prevention strategies[3,5].

In conclusion, Opalski syndrome is a rare but clinically significant variant of LMS that requires a high index of suspicion for diagnosis. Advances in neuroimaging have greatly facilitated the identification of its characteristic features, enabling timely intervention. Increased awareness among clinicians can lead to improved outcomes for patients affected by this unique neurological condition.

In our case, the absence of thrombolytic therapy was due to delayed presentation beyond the therapeutic window. Antiplatelet therapy and lipid management, however, proved effective in mitigating further neurological deficits. The patient's favorable outcome underscores the importance of early recognition and targeted management.

Conclusion:-

This case highlights the clinical and radiological hallmarks of Opalski syndrome, a rare manifestation of lateral medullary infarction. Comprehensive neurological evaluation and timely imaging are essential for accurate diagnosis and management. Increased awareness of this syndrome can aid clinicians in identifying and treating similar cases effectively.

References:-

1. Montaner J, Alvarez-Sabín J. Opalski's syndrome. J NeurolNeurosurg Psychiatry. 1999;67(5):688-689. doi:10.1136/jnnp.67.5.688
2. Opalski A. A new sub-bulbar syndrome: Partial syndrome of the posterior vertebro-spinal artery. Paris Med. 1946:214–20.
3. Pandey S, Batla A. Opalski's syndrome: A rare variant of lateral medullary syndrome. J Neurosci Rural Pract. 2013;4(1):102-104. doi:10.4103/0976-3147.105642
4. Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain J Neurol. 2003;126(Pt 8):1864-1872. doi:10.1093/brain/awg169
5. Hermann DM, Jung HH, Bassetti CL. Lateral medullary infarct with alternating and dissociated sensorimotor deficits: Opalski's syndrome revisited. Eur J Neurol. 2009;16(4):e72-74. doi:10.1111/j.1468-1331.2009.02556.x
6. Liu CY, Chang FC, Hu HH, Hsu LC. Ipsilateral cruralmonoparesis in lateral medullary infarction due to vertebral artery dissection. Eur J Neurol. 2006;13(7):e8-9. doi:10.1111/j.1468-1331.2006.01290.x
7. Cywinski Z, Horski-Horonczyk S, Prusinski A. [Two cases of Opalski syndrome (a partial posterior vertebrospinal artery syndrome)]. NeurolNeurochirPsychiatr Pol. 1954;4(5):511-514.
8. Castro CEN, Ortiz JM. Síndrome de Opalski, una variante del síndrome de Wallenberg: reporte de caso y revisión de la literatura. Acta Neurológica Colomb. 2017;33(3):182-187. doi:10.22379/24224022155.