Case Report

Shanshan Ma, Xiuping Zhang, Liu Zhao, Fei Peng, Yanhui Xing, Ruiping Wang and Xiaohong Chen*

A rare case of Opalski syndrome with a suspected multiple sclerosis etiology

https://doi.org/10.1515/med-2025-1362
Received August 11, 2025; accepted December 1, 2025;
published online December 17, 2025

Abstract

Objectives: Opalski syndrome is a rare lateral medullary variant that combines classic Wallenberg signs with ipsilateral hemiparesis, resulting from lesions affecting the corticospinal fibers below the pyramidal decussation. Stroke is the predominant cause of Opalski syndrome, and to date, no cases have been reported in association with multiple sclerosis (MS).

Case presentation: A 56-year-old man with hypertension developed dizziness, right-sided weakness, and gait instability. Examination revealed right-sided Horner's syndrome, hemiparesis, facial numbness, truncal ataxia, crossed sensory loss, and a positive Babinski sign. MRI demonstrated an acute infarction in the right dorsolateral medulla, along with multiple T1 and FLAIR hyperintensities in the periventricular white matter, basal ganglia, corpus callosum, and the C5–C6 spinal cord. Magnetic resonance angiography (MRA) findings were unremarkable. The patient declined lumbar puncture. A diagnosis of Opalski syndrome was established, and multiple sclerosis (MS) was proposed as a potential underlying etiology. Management included dual antiplatelet therapy, statin, vitamins, and physiotherapy, leading to gradual improvement with mild residual deficits.

Conclusions: This may represent the first case of Opalski syndrome linked to suspected MS, highlighting the importance of considering non-vascular etiologies in atypical lateral medullary presentations.

Keywords: Opalski syndrome; lateral medullary syndrome; multiple sclerosis; non-vascular etiology; case report

Background

Opalski syndrome, a rare clinical entity that was initially described in 1946 [1], represents a unique variant of lateral medullary syndrome (LMS), which is also known as Wallenberg syndrome. This syndrome is characterized by the presence of classic LMS symptoms in conjunction with ipsilateral hemiparesis [2, 3]. The underlying pathology of Opalski syndrome involves a lesion localized to the lateral medulla oblongata, a crucial region of the brainstem responsible for a wide range of vital functions and neurological pathways. While the exact anatomical location of the lesion can vary, it mainly influences the corticospinal fibers below the pyramidal decussation [4].

Clinically, Opalski syndrome manifests with a distinct constellation of neurological deficits. The most notable feature is ipsilateral hemiparesis, distinguishing it from the more common crossed neurological findings observed in classic LMS [4]. This hemiparesis is mainly accompanied by several hallmark symptoms of LMS, including vertigo, nausea, vomiting, dysphagia, nystagmus, and ataxia [5]. Sensory disturbances are also frequently observed, characterized by ipsilateral facial sensory loss (affecting pain and temperature sensation) and contralateral body hemianesthesia below the neck [4, 6]. Ipsilateral Horner's syndrome, characterized by a triad of miosis, ptosis, and anhidrosis, may also be present [7]. The specific combination and severity of these symptoms vary among individuals, which complicates the diagnostic process for this condition.

The most common cause of LMS is stroke, typically resulting from occlusion of the vertebral artery or its branches, particularly the posterior inferior cerebellar artery (PICA) [7, 8]. The underlying mechanisms of vascular compromise include atherosclerosis, embolism, or arterial dissection [9, 10]. Although less common, non-vascular

***Corresponding author: Xiaohong Chen**, Department of Neurology, Liaoning Province Jinqiu hospital, No.317 Xiaonan Street, Shenhe District, Shenyang, 110016, China, E-mail: Chenxiaohong0811@126.com. https://orcid.org/0009-0009-0906-0134
Shanshan Ma, Fei Peng, Yanhui Xing and Ruiping Wang, Department of Neurology, Liaoning Province Jinqiu Hospital, Shenyang, China. https://orcid.org/0009-0007-6482-1309 (S. Ma). https://orcid.org/0009-0003-2455-3590 (F. Peng)
Xiuping Zhang, Department of Neurology, Second Hospital of Shanxi Medical University, Taiyuan, China. https://orcid.org/0009-0006-6924-8540
Liu Zhao, Department of Radiology, Liaoning Province Jinqiu Hospital, Shenyang, China. https://orcid.org/0009-0008-2856-666X

conditions, such as multiple sclerosis (MS), tumors, infections, and traumatic injuries may also cause LMS [11–14]. While Wallenberg syndrome caused by MS mimicking a stroke has been documented [11], no similar case of Opalski syndrome has been reported. In this study, a case of Opalski syndrome involving the upper cervical spinal cord was reported. The magnetic resonance imaging (MRI) features of the central nervous system and the underlying etiology were analyzed to enhance the diagnosis and treatment of this condition.

Case presentation

A 56-year-old man was admitted with a 3-day history of sudden dizziness, right-sided weakness, and unsteady gait, predominantly leaning to the right. His symptoms began suddenly upon waking, initially mild and transient, while worsened the day before admission, when he experienced multiple episodes of dizziness, nausea, and vomiting, within 20 min after sexual intercourse. These episodes were accompanied by increasing right-sided weakness and gait instability. The patient had a 20-year history of hypertension and a suspected ischemic stroke 8 years earlier, which fully resolved with treatment. At that time, MRI revealed high signal intensity in the left basal ganglia, as well as abnormalities in the corpus callosum, centrum semiovale, and brainstem, indicative of ischemia. In the following years, the patient experienced multiple episodes of dizziness, accompanied by unsteady walking, each lasting from one to several days, which resolved spontaneously without the need for medical intervention. He had abstained from smoking and alcohol for the past 8 years.

On neurological examination, he exhibited right-sided Horner's syndrome, bilateral restricted leftward eye movement, right lower facial weakness, right-sided hemiparesis with upper motor neuron signs, truncal ataxia, and a positive Babinski sign. His NIHSS score was 4.

Laboratory findings showed mild leukocytosis with neutrophilia, lymphopenia, and elevated homocysteine levels. Thyroid function tests revealed low triiodothyronine (T3) and elevated anti-thyroid antibody levels.

Brain MRI revealed an abnormally high signal corresponding to an ischemic infarct in the right dorsolateral medulla, consistent with a diagnosis of lateral medullary syndrome (Figure 1). Magnetic resonance angiography (MRA) of the head and neck displayed no significant stenosis or occlusion (Figure 2a and b). Additionally, multiple T1 and FLAIR hyperintensities were identified in the bilateral basal ganglia, periventricular white matter, centrum semiovale, and corpus callosum, as well as abnormalities at the C5-C6 levels of the cervical spinal cord (Figure 2c and d). A diagnosis of Opalski syndrome was made based on the clinical presentation and imaging findings. Conventional MRI (Figure 3) revealed patchy areas of prolonged T1WI and FLAIR signals in the white matter surrounding the lateral ventricles, corpus callosum, and cortex, with "black hole" changes in some lesions. FLAIR sequences demonstrated lesion expansion and irregular ventricular walls (Figure 4a and b). Contrast-enhanced MRI revealed an open ring sign in the medullary lesions, with no significant spinal cord enhancement (Figure 4c), raising the suspicion of a demyelinating disease, such as multiple sclerosis (MS). Although the lumbar puncture could provide additional diagnostic information, the patient and his family declined the procedure.

After ruling out tumors, infections, and inflammatory diseases, conservative treatment of ischemic stroke was initiated, including aspirin, clopidogrel, atorvastatin, and physiotherapy. Intravenous thrombolysis was not administered due to the time elapsed since symptom onset. During the 10-day hospital stay, the patient's right-sided weakness, dizziness, and gait instability gradually improved, along with the right-sided Horner's syndrome, which showed a reduction in facial anhidrosis.

Ethical approval and consent for publication

Individual case reports do not require formal Institutional Review Board approval. Written informed consent for publication of the clinical data and images was obtained from the patient.

Discussion

This case report presented a 56-year-old male patient with Opalski syndrome, a rare variant of LMS. This case is particularly unique as the patient also presented with features suggestive of MS, such as multiple T1 and FLAIR hyperintensities in the bilateral basal ganglia, periventricular white matter, centrum semiovale, and corpus callosum, as well as abnormal signals in the cervical spinal cord at the C5-C6 levels. To our knowledge, Opalski syndrome associated with possible MS has not been previously reported in the medical literature, suggesting that this may be the first documented case.

Opalski syndrome is typically characterized by classic LMS symptoms, alongside ipsilateral hemiparesis [4]. In this case, the patient presented with right-sided Horner's syndrome, bilateral restricted leftward eye movement, right

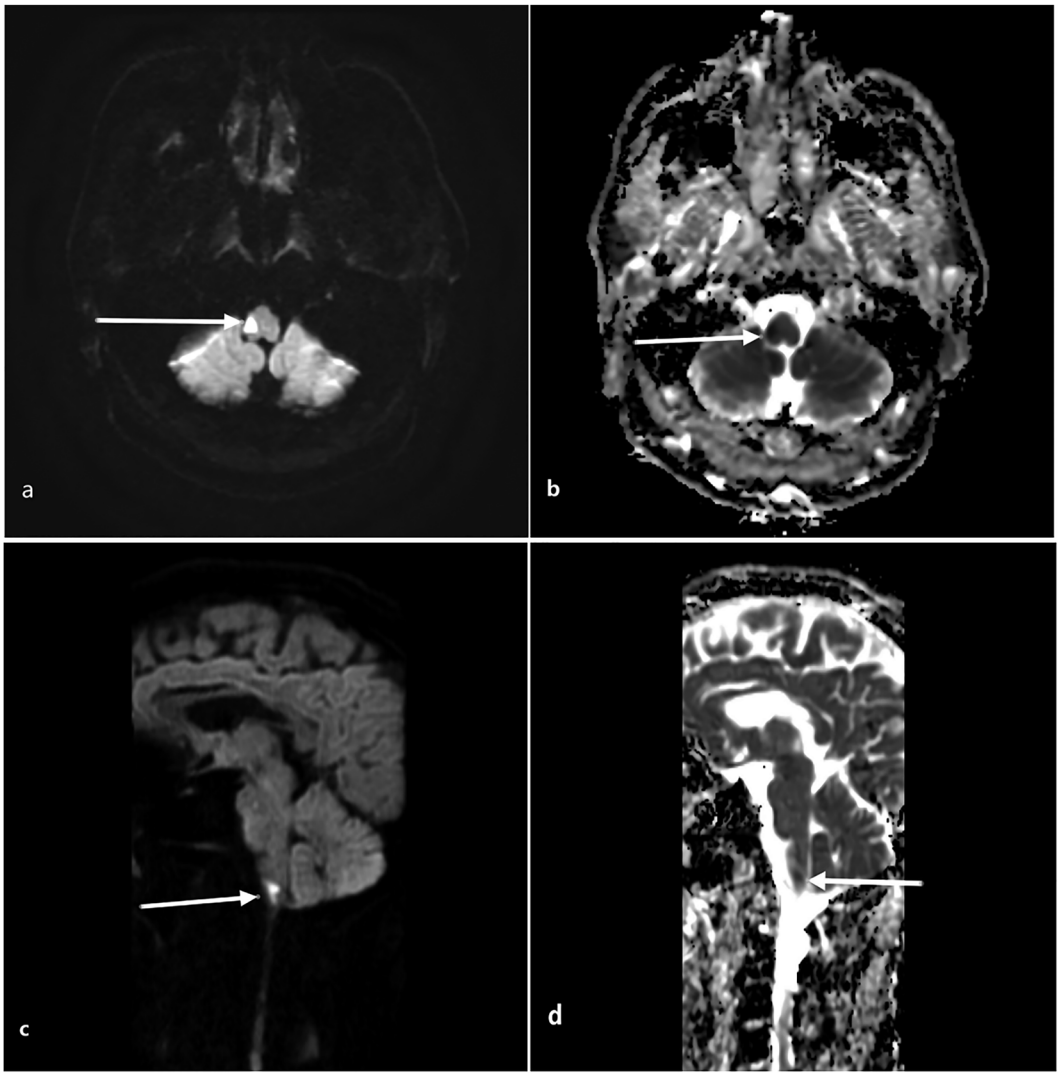

Figure 1: Axial diffusion-weighted MR images (a) showing a focal area of diffusion restriction in the right dorsolateral medulla (white arrow), suggestive of an acute infarct. Axial diffusion-weighted imaging of the middle medulla demonstrating the acute infarct of the right medulla (c). The ADC map (b and d) reveals a corresponding positive lesion (arrow) in the medulla.

lower facial weakness, right-sided hemiparesis, right-sided truncal ataxia, and a positive Babinski sign on the right side. These neurological deficits, combined with MRI findings of an ischemic infarct in the right dorsolateral medulla, consistent with LMS, confirmed the diagnosis. Despite ischemic infarction being the most common cause of LMS, the patient's imaging raised the possibility of an underlying non-vascular etiology, particularly MS. Imaging revealed several features that pointed towards a demyelinating process. Apart from the medullary infarct, multiple FLAIR hyperintensities were identified in the basal ganglia, white matter, corpus callosum, and cervical spinal cord. Furthermore, conventional MRI sequences revealed prolonged T1WI and FLAIR signals with "black hole" changes, indicative of chronic demyelination (Figures 3 and 4). Contrast-enhanced MRI also demonstrated an open-ring sign in the medullary lesions, which is characteristic of demyelinating diseases, such as MS (Figure 4c).

Differentiating Opalski syndrome from other conditions with similar presentations was crucial in this case. Other variants of Wallenberg syndrome, brainstem infarcts in regions other than the lateral medulla, as well as potential neoplastic, infectious, and inflammatory etiologies were considered. The patient's recurrent neurological events and MRI findings strongly favored MS as the most plausible underlying diagnosis. Notably, the patient had a suspected cerebral infarction 8 years earlier, and current imaging revealed disseminated lesions in the periventricular and cortical regions, cerebellum, and spinal cord. According to the 2024 revisions of the McDonald criteria [15], MS can be diagnosed in typical cases when a compatible clinical

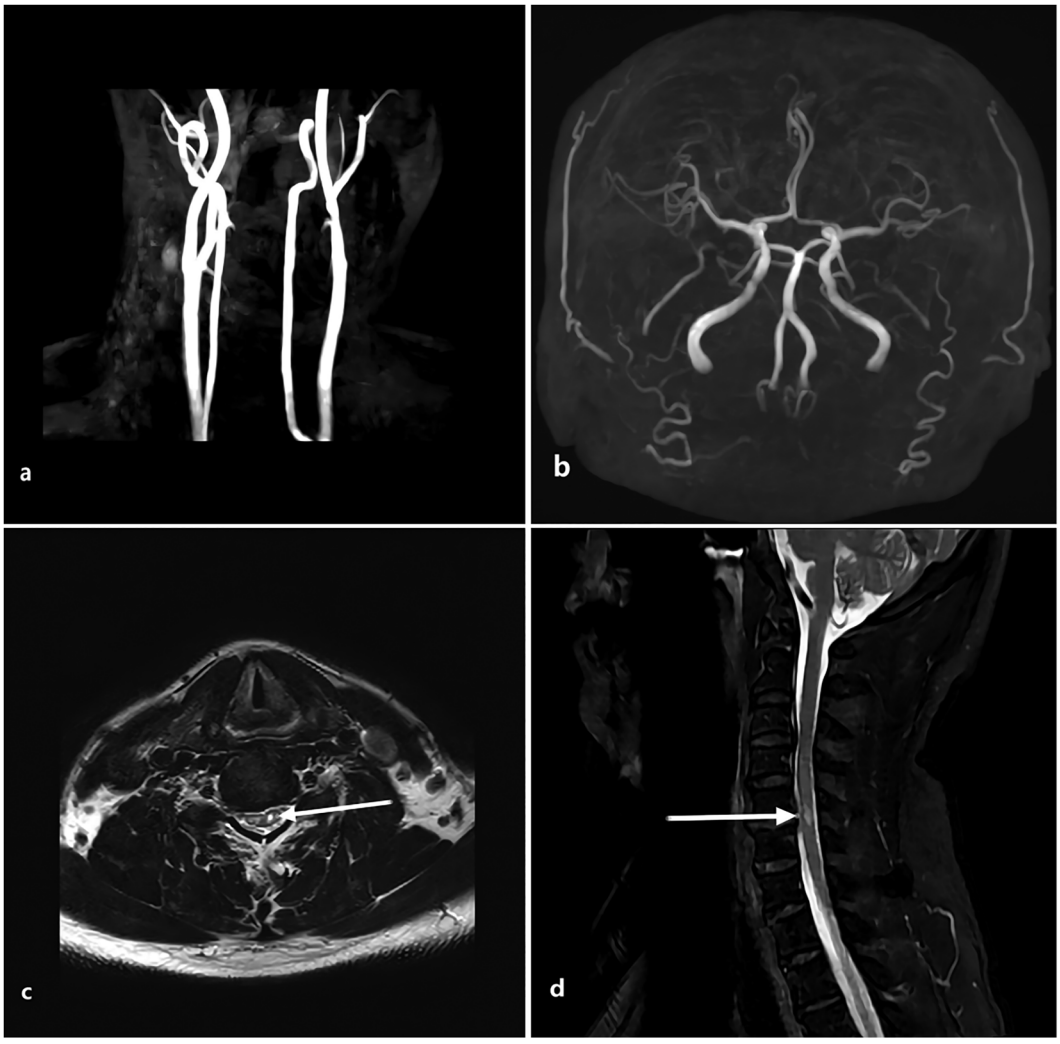

Figure 2: Magnetic resonance angiography (MRA) shows no significant stenosis or occlusion of the intracranial or extracranial vessels (a and b). T2/FLAIR hyperintensities in the cervical spinal cord at the C5-C6 levels (c and d).

syndrome is accompanied by MRI evidence of dissemination in space across characteristic CNS regions, with supportive evidence of dissemination in time, provided that alternative diagnoses are carefully excluded. In our patient, multifocal lesions involving the supratentorial white matter, infratentorial structures, and the cervical spinal cord fulfill dissemination in space, while the remote suspected vascular event and subsequent recurrent transient neurological episodes provide supportive evidence of dissemination in time. In addition, demyelinating-appearing cervical cord lesions are uncommon in chronic small-vessel ischaemic disease and therefore increase the specificity of an MS diagnosis in an individual over 50 years of age with vascular risk factors. Although CSF analysis remains an important supportive tool to increase diagnostic confidence and to exclude alternative inflammatory or infectious disorders, it is not mandatory in all patients under the updated criteria and was not performed in this case because lumbar puncture was declined.

MS, though an uncommon cause of LMS, has been documented in the literature. Smith et al. reported a case of Wallenberg syndrome due to demyelination identified postmortem, even though the patient had not fulfilled clinical MS criteria [16]. Similarly, Qiu et al. described a case of incomplete Wallenberg syndrome as the initial manifestation of MS [11]. These reports highlight the ability of MS to mimic stroke, especially in atypical presentations, lacking vascular abnormalities on imaging. In our patient, the open-ring sign found on the contrast-enhanced MRI and the scattered lesions in the white matter and corpus callosum further reinforced the possibility of MS.

The mechanisms by which MS could lead to Opalski syndrome likely involve inflammation and demyelination of

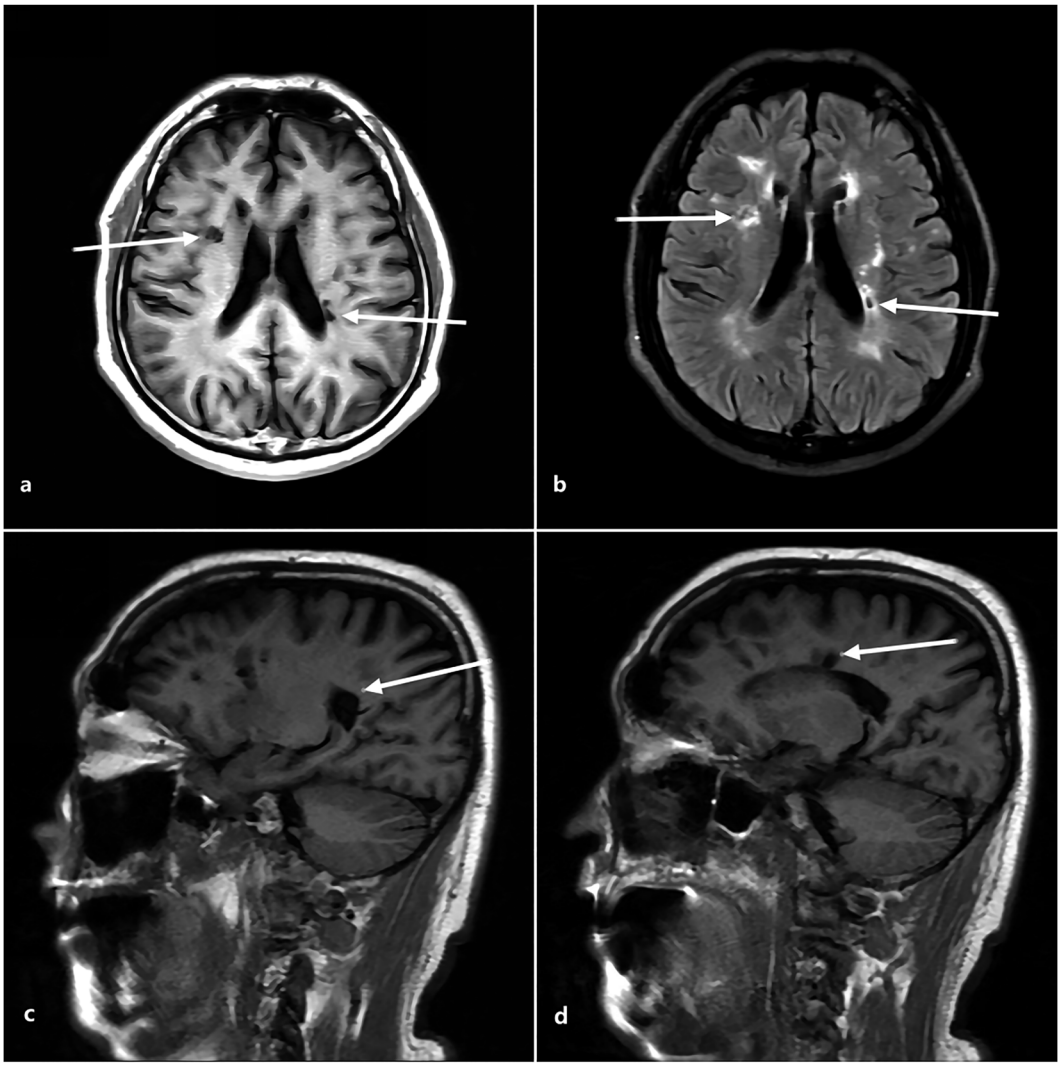

Figure 3: Magnetic resonance sequences showed round, oval, banded and patchy long T1WI (a, c, d) and flair signals (b) in the white matter around the lateral ventricles, the center of the hemioval, the corpus callosum and the cortical lobe. T1WI signals of some lesions showed "black hole" changes.

the lateral medulla, influencing key structures, such as the corticospinal tract. These processes could explain the hemiparesis and other neurological characteristic of Opalski syndrome. While vascular occlusion of the vertebral artery or posterior inferior cerebellar artery (PICA) remains the primary cause of LMS [7, 8], this case underscores the potential of non-vascular etiologies, such as MS, to cause similar syndromes.

In addition to MS, other non-vascular causes of LMS have been reported, including tumors, infections, and trauma. Ho et al. described a case of LMS caused by small-cell carcinoma, where metastatic spread to the lateral medulla led to stroke-like symptoms [13]. Kovacs et al. reported a case of LMS following varicella infection, suggesting a vasculitic mechanism [12]. Viral infections have also been implicated in LMS, as Román-Campos et al. described a fatal case of LMS caused by brainstem encephalitis due to herpesvirus [17]. Additionally, Lawson-Smith et al. documented that trauma can cause LMS, as illustrated by a case involving a low-velocity penetrating injury in an intravenous drug user [14]. These outcomes highlight the broad differential diagnosis for LMS and the importance of considering both vascular and non-vascular causes.

This case report has several limitations. The lack of long-term follow-up data and the patient's refusal to undergo lumbar puncture could hinder the ability to establish a definitive diagnosis of MS. Although the multifocal lesions and open-ring enhancement pattern raise suspicion of a demyelinating process, the long asymptomatic interval and the absence of cerebrospinal fluid analysis preclude us from further increasing the diagnostic certainty for MS and from fully excluding other demyelinating or ischaemic etiologies.

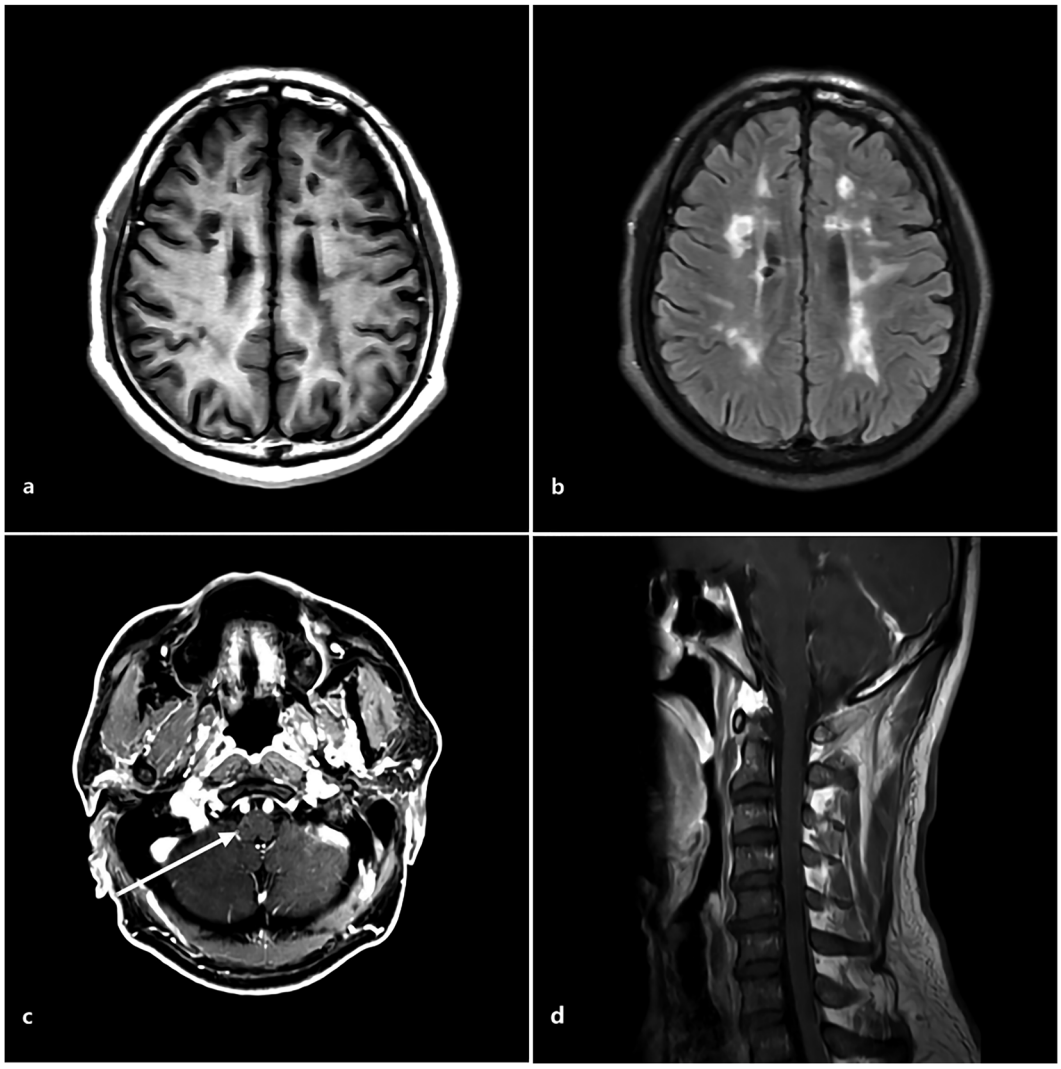

Figure 4: T1WI (a) and flair (b) sequence showed obvious hypersignal changes, including the increase of lesions, the enlargement of the scope, the rough ventricle upper wall. Head enhanced MRI showed slightly open ring sign in the medulla oblongata lesions (c). There was no significant enhancement in spinal cord enhanced MRI (d).

Further investigation and longitudinal follow-up are necessary to clarify the underlying pathology and to deeply understand the potential association between MS and Opalski syndrome.

In conclusion, this study presented a distinctive case of Opalski syndrome potentially linked to MS, which may be the first such case documented in the literature. It highlights the importance of considering non-vascular causes, particularly MS, in cases of Opalski syndrome, especially when atypical clinical or radiological findings are present.

Acknowledgments: The authors would like to thank the patient and his family for their cooperation. We thank Medjaden Inc. for the language editing of this manuscript.

Funding information: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution: SM: Writing – review & editing, Conceptualization, Methodology, Project administration; XZ: Writing – review & editing; LZ: Writing – review & editing; FP: Writing – review & editing; YX: Writing – review & editing; RW: Writing – review & editing, Formal Analysis; XC: Writing – original draft, Conceptualization, Methodology. All authors have reviewed the final version of this manuscript and agreed to be accountable for all aspects of the study.

Conflict of interest: The authors declare that they have no competing interests.

Data Availability Statement: No data were utilized for the research described in the article.

References

1. Opalski A. Syndrome partiel de l'artère vertébro-spinale postérieure. Paris Med 1946:1214–20.
2. Khan U, Ahmad B, Aslam A, Muhammad A, Iqbal J. Opalski syndrome, a rare variant of Wallenberg syndrome, the first case reported from Pakistan: a case report. Heliyon 2023;9:e21687.
3. Kk P, R K, P C, Aiyappan SK, N D. A rare variant of Wallenberg's syndrome: Opalski syndrome. J Clin Diagn Res 2014;8:Md05–6.
4. Hermann DM, Jung HH, Bassetti CL. Lateral medullary infarct with alternating and dissociated sensorimotor deficits: Opalski's syndrome revisited. Eur J Neurol 2009;16:e72–4.
5. Pandey S, Batla A. Opalski's syndrome: a rare variant of lateral medullary syndrome. J Neurosci Rural Pract 2013;4:102–4.
6. Joseph AT, Toluie A, Hrehorovich PA. Opalski syndrome and elucidation of lateral medullary syndrome. Cureus 2024;16: e54314.
7. Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126: 1864–72.
8. Hong YH, Zhou LX, Yao M, Zhu YC, Cui LY, Ni J, et al. Lesion topography and its correlation with etiology in medullary infarction: analysis from a multi-center stroke study in China. Front Neurol 2018;9813:813.
9. Kim JS, Caplan LR. Clinical stroke syndromes. Front Neurol Neurosci 2016;40:4072–92.
10. García-García J, Ayo-Martín O, Segura T. Lateral medullary syndrome and ipsilateral hemiplegia (Opalski syndrome) due to left vertebral artery dissection. Arch Neurol 2009;66:1574–5.
11. Qiu W, Wu JS, Carroll WM, Mastaglia FL, Kermode AG. Wallenberg syndrome caused by multiple sclerosis mimicking stroke. J Clin Neurosci 2009;16:1700–2.
12. Kovacs SO, Kuban K, Strand R. Lateral medullary syndrome following varicella infection. Am J Dis Child 1993;147:823–5.
13. Ho KL. Uncommon causes of the lateral medullary syndrome. report of a case of metastatic carcinoma. Arch Neurol 1980;37:669–70.
14. Lawson-Smith MJ, Smith SJ, Leach JC, Cadoux-Hudson T. Lateral medullary syndrome caused by penetrating head injury. J Clin Neurosci 2006;13: 792–4.
15. Montalban X, Lebrun-Frénay C, Oh J, Arrambide G, Moccia M, Pia AM, et al. Diagnosis of multiple sclerosis: 2024 revisions of the McDonald criteria. Lancet Neurol 2025;24:850–65.
16. Smith DB, Demasters BK. Demyelinating disease presenting as Wallenberg's syndrome. report of a patient. Stroke 1981;12:877–8.
17. Román-Campos G, Toro G. Herpetic brainstem encephalitis. Neurology 1980;30:981–5.