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Int J Case Rep Images 2025 Jul-Dec;16(2):7–9.
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A case of hemi facial spasm that resolved with the onset of Wallenberg syndrome

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ABSTRACT

Introduction: A rare case of hemi facial spasm that disappeared with the onset of ipsilateral Wallenberg syndrome was observed.

Case Report: A 74-year-old woman had a left vertebral artery occlusion. The patient presented with left Wallenberg syndrome by a left medullo-lateral infarction. In addition, the left hemi facial spasm, from which had been suffered for last six months, disappeared.

Conclusion: The patient had ipsilateral vertebral artery occlusion. The occluded artery had caused the facial spasm with pulsatile stimulation against the root exit zone of VII nerve. Because of lateral area of medulla oblongata was perfused with contralateral posterior inferior cerebellar artery, ipsilateral medulla infarction had been left only slight neurological deficits. This report presents a fortunate case with anatomical and hemodynamic features.

Keywords: Hemi facial spasm, Posterior inferior cerebellar artery, Root exit zone, Vertebral artery, Wallenberg syndrome

How to cite this article

Tempaku A. A case of hemi facial spasm that resolved with the onset of Wallenberg syndrome. Int J Case Rep Images 2025;16(2):7–9.

Article ID: 101513Z01AT2025

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Received: 19 May 2025
Accepted: 28 June 2025
Published: 26 July 2025

doi: 10.5348/101513Z01AT2025CR

INTRODUCTION

A patient was experienced a rare case of left hemi facial spasm that disappeared with the onset of left Wallenberg syndrome. This report outlines the vascular structure and pathophysiology of the posterior circulation that provided the background for this case.

CASE REPORT

A 74-year-old woman with a history of diabetes mellitus (DM) had suffered from left hemi facial spasm for the past six months and had been treated conservatively. She then developed ataxia and left sensory disturbance with sudden onset. She was admitted to the emergency department. She had no headache, neck pain, or motor dysfunction of the extremities. Her blood pressure was elevated at 185/91 mmHg. Neurologically, the patient presented with left Wallenberg syndrome. However, there were no concomitant lower cranial nerve disorders. In addition, the left hemi facial spasm, from which she had suffered for the last six months, had disappeared. A head magnetic resonance imaging (MRI) scan revealed a left medullary infarction on a diffusion-weighted image (DWI) (Figure 1A). A time-of-flight (TOF) image confirmed a left vertebral artery (VA) occlusion (Figure 1B). Cerebral angiography showed that the left VA was disrupted beyond V2 portion (Figure 1C). Right VA imaging showed that the right posterior inferior cerebellar artery (PICA) was perfusing both cerebellar hemispheres (Figure 1D). The patient appeared to have a left medullary infarction associated with left VA occlusion. The hemi facial spasm disappeared to the immediately after the onset of Wallenberg syndrome. Antithrombotic therapy involving the oral administration of two antiplatelet drugs (including aspirin and clopidogrel) and the intravenous infusion of ozagrel was introduced alongside edaravone infusion. Then, the patient underwent acute stroke rehabilitation and was discharged home without any neurological deficits.

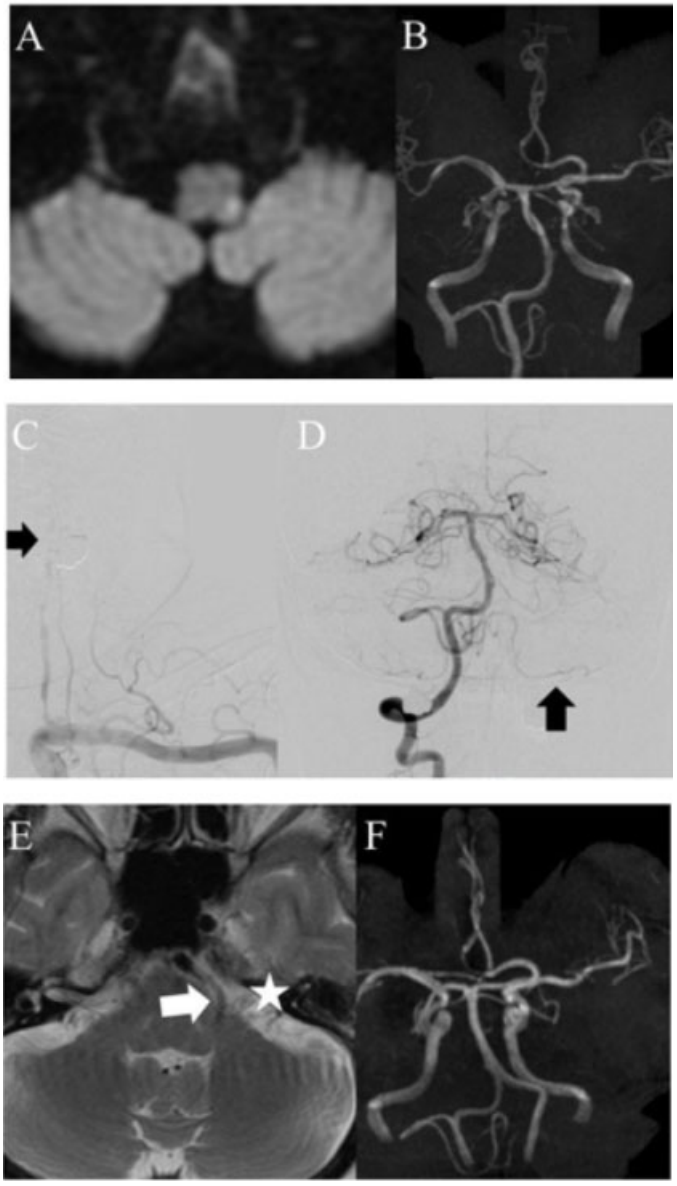

Figure 1: (A) A head magnetic resonance imaging (MRI) with diffusion weighted image (DWI) at the admission time. (B) A time of flight (TOF) image in MRI at the admission time. (C) An angiographical image from left subclavian artery. Arrow points the left vertebral artery, which flow disappears over V2 portion. (D) An angiography from right vertebral artery. Arrow points the distal flow of the left posterior inferior cerebellar artery, which is derived from the right vertebral artery. (E) The TOF in MRI at the twelve years ago. (F) The T2-weighted image in MRI at the twelve years ago. Arrow points the flow void of the left VA. Star means the cranial nerve VII.

DISCUSSION

The patient's previously affected unilateral facial spasm resolved with the onset of the stroke. Magnetic resonance imaging taken prior to the stroke showed that the left VA was running near the brainstem (Figure 1E), and was thought to be in close proximity to the root exit zone (REZ) of the VII cranial nerve (Figure 1F). It was therefore thought that the left VA was compressing the VII REZ due to arteriosclerotic changes caused by DM and other background factors, resulting in the unilateral facial spasm. However, when the left VA occluded and caused the cerebral infarction, the arterial vasopulsation of the VA disappeared. Consequently, the pressure stimulus to the VII cranial nerve disappeared, as did the facial spasm.

The outer medulla oblongata is perfused from the VA, PICA, and other sources [1, 2]. In the present case, the VA perforating branch likely perfused to the lateral medulla oblongata, since the disease was caused by left VA occlusion. Although there are few cases of bilateral dominance of the PICA [3, 4], in this case, the right PICA perfused both sides with no cerebellar infarction complications on either side. Furthermore, despite the occlusion of only the left VA, there was no decrease in blood flow to the right VA or the PICA. The patient experienced a left lateral medullary infarction, which suggests that the perforating branch from the left VA was the primary vessel responsible for perfusing the lateral medulla oblongata.

Hemi facial spasm develops when the anterior inferior cerebellar artery (AICA), VA or other vessels come into contact with the REZ of VII [5, 6]. In the present case, MRI images taken before the onset of stroke showed that the left VA ran near the brainstem, and that the left AICA showed no significant flow gain. In addition, the right PICA was perfused peripherally from the cerebellar tonsils to the cerebellar hemispheric cortex, but not to the left cerebellopontine angle. Therefore, the left VA was probably responsible for the facial spasm. Occlusion of the left VA caused blood flow to be disrupted, and the vascular pulsatile stimulation of VII to disappear. The facial spasm seemed to improve without any treatment. This provided collateral evidence that the pulsatile stimulation of the bent vessels was the cause of the facial spasms.

CONCLUSION

A rare case of hemi facial spasm that disappeared with the onset of Wallenberg syndrome was described. The REZ of the seventh nerve was stimulated directly by VA pulsation. Vertebral artery occlusion resulted in loss of REZ stimulation. However, the medulla oblongata was protected from severe ischemia by bilateral perfusion from the contra-lateral PICA.

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Acknowledgments
The author thanks the medical staff of Hokuto Hospital to support in the clinical treatment and care for the patient.

Author Contributions
Akira Tempaku – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission
The corresponding author is the guarantor of submission.

Source of Support
None.

Consent Statement
Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest
Author declares no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.