Case Lesson 45-2026

Dual-Vessel Neurovascular Conflict in Hemifacial Spasm: A Case Report and Systematic Review of Surgical Outcomes

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Introduction

Hemifacial spasm (HFS) is a neurovascular compression disorder characterized by involuntary unilateral contractions of the facial musculature. The condition results from chronic pulsatile irritation of the centrally myelinated segment of the facial nerve, most commonly at the root exit zone (REZ) or proximal attached segment. Arterial loops of the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), and vertebral artery (VA) are the most frequently implicated vessels.

Microvascular decompression (MVD) remains the only curative treatment for HFS, despite all the patients are oriented for Botox. While single-vessel compression is typically identified, multiple-vessel conflicts may coexist and represent an underrecognized cause of persistent or recurrent symptoms. Incomplete exploration of the facial nerve REZ and proximal segment may result in failure to address additional offending vessels.

We report a case of dual arterial compression in a series of four patients treated at our department. This unusual dual conflict in a young woman with medically refractory HFS, emphasize the importance of complete anatomical exploration during MVD.

Case Presentation

A 27-year-old woman presented with a 3–4-year history of progressive, continuous left-sided hemifacial spasm. The spasms initially involved the periorbital musculature and gradually

extended to the lower face. She reported significant social distress and functional impairment. Despite several treatment sessions with botulinum toxin injections, symptom relief was temporary and progressively diminished.

Neurological examination revealed tonic and clonic contractions of the left hemiface without facial weakness, sensory deficits, or hearing impairment. Magnetic resonance imaging suggested vascular contact at the facial nerve REZ.

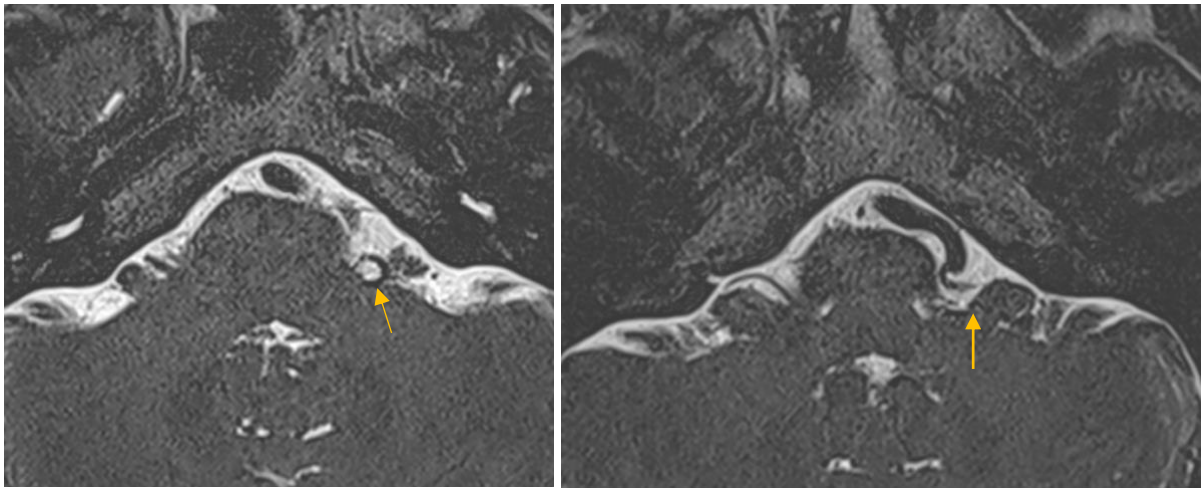

Fig 1: A: *PICA conflict at REZ* ***B:*** *Small vessel conflict, branch of AICA*

The patient underwent left retrosigmoid microvascular decompression. Intraoperatively, a PICA loop was identified compressing the facial nerve at the proximal REZ. The vessel was mobilized and separated from the nerve using Teflon felt interposition.

Following initial decompression, further exploration of the proximal facial nerve and its exit point from the brainstem revealed an additional small arterial branch exerting focal compression at the nerve's exit zone. This second vessel was also mobilized and separated using additional muscle patches.

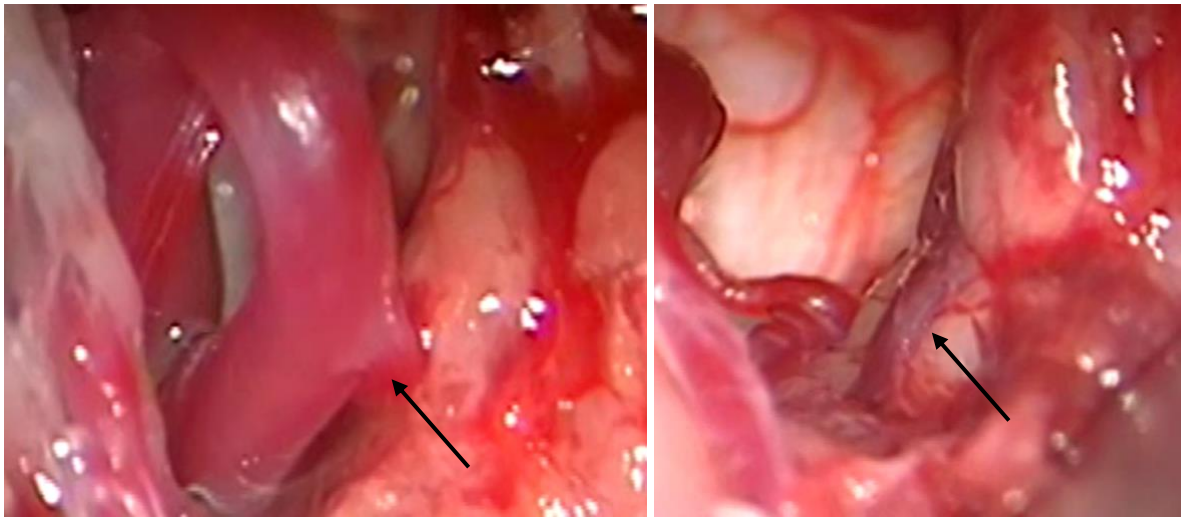

Fig 2: A: *PICA conflict at REZ* ***B:*** *Small vessel conflict, branch of AICA*

No venous compression was observed. The postoperative course was uneventful. The patient experienced immediate and complete resolution of facial spasms. At 3-month follow-up, she remained asymptomatic without neurological deficits.

Results

Complete decompression of both offending vessels resulted in sustained symptom resolution. No complications occurred, including facial nerve palsy, hearing impairment, cerebrospinal fluid leakage, or lower cranial nerve dysfunction.

Within our institutional series of four patients treated for facial nerve neurovascular conflict during the study period, dual-vessel compression was identified in one patient. Although limited by small sample size, this observation underscores the clinical relevance of multiple-vessel involvement.

Discussion

Hemifacial spasm is a classical neurovascular compression syndrome caused by arterial contact with the centrally myelinated portion of the facial nerve. Contemporary anatomical studies emphasize that compression may occur anywhere along the facial REZ and attached segment, not exclusively at the classical transition zone between central and peripheral myelin. Proper exposure of the proximal nerve segment is therefore essential to achieve complete decompression.

While single-vessel compression is often described, multiple-vessel involvement is reported in 10-15% in the two large series[4,9]. In vertebral artery–associated cases, additional small arterial contributors have been reported in the majority of patients. These findings highlight the importance of meticulous exploration beyond the dominant offending vessel.

In a long-term follow-up, patients sustained spasm relief was achieved in approximately 90% of cases. Similarly, a 22-year institutional experience reported excellent or good outcomes in 85–92% of cases, with low rates of permanent morbidity, further reinforcing MVD as the definitive treatment for HFS.

PICA is frequently implicated either alone or in combination with other vessels. Studies examining vascular patterns have demonstrated that PICA involvement may be particularly

common in complex or multi-vessel configurations. The presence of a secondary small arterial branch, as observed in our case, may represent a significant compressive contributor even after decompression of a larger vessel.

Failure to identify and address additional compressive branches may lead to incomplete decompression and persistent symptoms. Surgical failures reported in the literature often result from inadequate exposure of the proximal REZ and failure to explore the entire attached segment of the facial nerve, according to the recent anatomical description by Kaufman in JNS. Thorough dissection of the arachnoid planes and systematic inspection of the exit point from the brainstem are therefore mandatory.

Complication rates across major series remain low, with permanent facial weakness typically reported in less than 2–3% of patients and hearing impairment in 2–4%. Importantly, outcomes in cases involving multiple vessels do not appear significantly worse when meticulous microsurgical technique is employed.

Our case supports these observations. Despite imaging suggesting a single conflict, intraoperative exploration revealed dual arterial compression. Complete decompression of both vessels resulted in immediate and sustained symptom resolution.

Conclusion

Dual-vessel neurovascular conflict represents an important mechanism of hemifacial spasm and may be underrecognized preoperatively. Complete exploration of the facial nerve root exit zone and proximal brainstem exit point during microvascular decompression is essential to identify additional offending vessels with safe and highly effective curative treatment for hemifacial spasm when meticulous anatomical inspection and vessel mobilization are performed.

References

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