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Case Report

Improvement of Hemifacial Spasm Following Palliative Embolization of an Unruptured Cerebellar Arteriovenous Malformation

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Introduction: Posterior fossa arteriovenous malformations (AVMs) are uncommon, accounting for 7–15% of all intracranial AVMs. These malformations typically present with symptoms such as headaches, seizure, and intracerebral hemorrhage. Reports of hemifacial spasm–characterized by involuntary contractions of the facial muscles–as a presenting symptom of AVMs are extremely rare and usually occur only when the facial nerve is compressed. **Case:** A 35-year-old male presented with worsening left-sided hemifacial spasm, unresponsive to medication for around one year, followed by progressive headache and dizziness. T2-weighted MRI revealed contact between the facial nerve root and a tortuous posterior inferior cerebellar artery (PICA), along with an AVM in the left cerebellar hemisphere. Cerebral angiography demonstrated a left cerebellar AVM with feeding arteries from superior cerebellar artery (SCA) and PICA. The patient underwent successful embolization of the SCA using glue (n-BCA:lipiodol). An intraprocedural thrombus developed but was managed appropriately. Post-procedural cerebral angiography revealed recanalization of the basilar artery and left PICA, with a 30% reduction in nidus size. The hemifacial spasm improved significantly after embolization with an HFS-7 score reduction of six points in the first week post-procedure. **Conclusion:** Palliative embolization has shown potential in alleviating symptoms associated with hemifacial spasm and improving quality of life. Careful patient selection is essential to rule out secondary causes of hemifacial spasm and to identify underlying neurovascular contacts.

Keywords: Cerebellar AVM, Embolization, Endovascular treatment, Hemifacial spasm, Quality of life

Highlights
- Cerebellar region's AVM and hemifacial spasm
- Embolization AVM and hemifacial spasm

Introduction

Brain arteriovenous malformations (AVMs) are congenital vascular anomalies characterized by a direct connection between cerebral arteries and veins without an intervening capillary network. The incidence of AVMs is 1.21 per100,000 person-years (95% confidence interval 1.02–1.42).[1] Posterior fossa AVMs are rare, accounting for 7–15% of all intracranial AVMs. The most common presentations include headache, seizure, and intracerebral hemorrhage. AVM presenting with hemifacial spasm (HFS) is extremely rare.[1,2]

HFS is a neurological disorder characterized by unilateral involuntary tonic and clonic contractions of the

facial muscles. It is most commonly caused by compression of the facial nerve at its root exit zone by adjacent vascular structures. However, secondary causes of HFS should be thoroughly investigated using various diagnostic modalities, including neuroimaging, to identify potential interventions.[3–5] While headache, seizure, and hemorrhage are the typical manifestations of AVMs, their association with HFS is exceptionally rare, with fewer than 50 documented cases in literature to date.[3,6]

Management of HFS is challenging due to the limited efficacy of available oral medications. Alternative treatment options include botulinum toxin injections, which offer temporary relief, and microvascular decompression (MVD)–a minimally invasive procedure with a high success rate but associated with potential serious complications, including permanent cranial nerve palsy, stroke, and death.[7] In cases of secondary HFS with a confirmed vascular etiology, MVD alone may not be sufficient. Combined approaches involving AVM resection or endovascular treatment may be necessary. The endovascular approach is often preferred over open surgery when the AVM is located in eloquent areas of the brain, such as the brainstem, cerebellar peduncles, or deep nuclei.[8]

In this report, we present a case of hemifacial spasm secondary to an extensive cerebellar AVM that showed improvement following palliative embolization.

Case

A 35-year-old male presented to the outpatient clinic with complaints of left hemifacial spasm, which had begun one year prior. Initially, he experienced involuntary contractions around the left eye that intensified under stress. After six months, the spasms progressed to involve the perioral region, with increasing severity. He reported minimal relief from conservative treatments, including carbamazepine, trihexyphenidyl, and haloperidol. The symptoms interfered with his daily activities and negatively impacted his quality of life, as reflected by a total HFS-7 score of 17.

Additional symptoms that developed alongside worsening HFS included progressive headache and dizziness. Neurological examination revealedtonic-clonic facial spasms on the left side, graded as IV on the Samsung Medical Center (SMC) HFS scale.

MRI revealed abnormal vasculature in the left cerebellum, suspected to be an AVM with a feeder artery from the left vertebral artery and drainage to the left transverse sinus (Figure 1).

Cerebral angiography performed using a Philips Azurion 7 biplane system, confirmed a high-flow AVM in the left cerebellar hemisphere measuring 3.5 x 2.8 x 2.5 cm. The AVM was of a mixed plexiform-fistulous type, supplied by feeder arteries from the superior cerebellar artery (SCA) and posterior inferior cerebellar artery (PICA), with drainage into the veins of the left cerebellar hemisphere and the left transverse sinus. Venous ectasia was present, but no intranidal aneurysm was identified.

Interventional management was selected for this case due to the refractory nature of the HFS in this particular case. Based on the medium nidus size and anatomical location, the AVM was classified as Spetzler-Martin Grade III. Due to the moderate surgical risk associated with this location, palliative embolization was chosen as the preferred treatment modality.

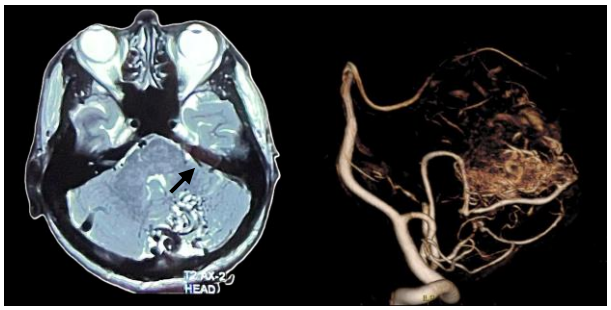

Figure 1. T2W1 MRI (left) showing contact between facial nerve root and a tortuous PICA (black arrow), and an AVM in the left cerebellum. Three-dimensional (3D) cerebral angiography (right) showed a left cerebellar AVM with feeders from the SCA and PICA.

The patient underwent successful palliative embolization of the AVM. Cannulation of the left subclavian artery was achieved using 6-F Ballast long sheath with a 5-F Vertebral diagnostic catheter over a 0.035-inch guidewire. Embolization of the left SCA was performed three sessions using a Sofia Plus intermediate catheter, MagicMP. and Magic 1.5 microcatheters, and a Hybrid 0.08-inch microwire.

A microcatheter over microwire was advanced into the SCA, and embolization was performed using adhesive liquid glue (N-Butyl Cyanoacrylate [N-BCA]:Lipiodol 1:1) with 50% concentration, followed by 30% and 25%, respectively (Figure 2).

During the procedure, a thrombus developed in the proximal basilar artery and the left PICA. Thrombectomy was performed using a 6 x 40 mm stent retriever. Post-procedural cerebral angiography demonstrated successful recanalization of the basilar artery and the left PICA, along with a 30% reduction in nidus size. (Figure 3 and Figure 4). Clinical evaluation on the following day revealed significant improvement in HFS with the SMC grade decreasing from IV to III. One week after the procedure, the patient's quality of life had improved with a reduction of six points in the total HFS-7 score.

Discussion

This case report adds to the growing body of evidence supporting a neurovascular etiology–particularly AVM and its feeding arteries–as a potential, though rare, cause of secondary HFS. Endovascular embolization reduced the feeding arterial flow and subsequently decreased the size

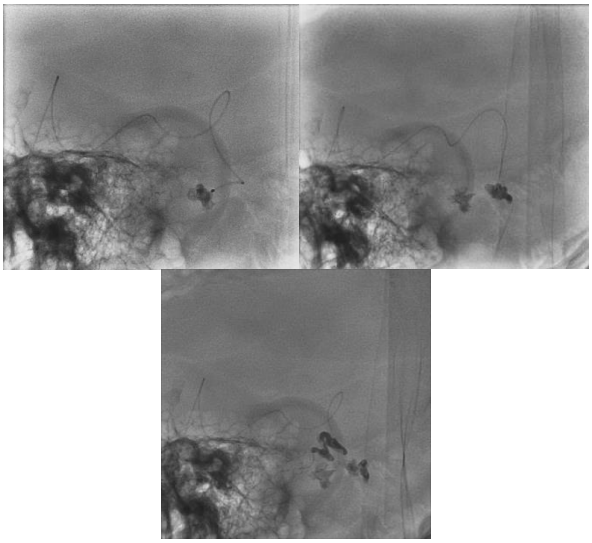

Figure 2. Microcatheter selective injection and glue casting in the SCA.

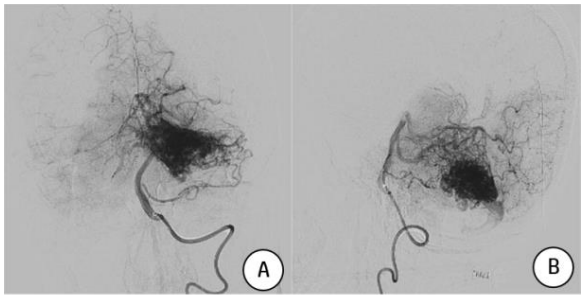

Figure 3. Antero-posterior (AP) (left) and lateral (right) projection of the left vertebral artery cerebral angiography prior to embolization, showing the AVM in the left cerebellar hemisphere.

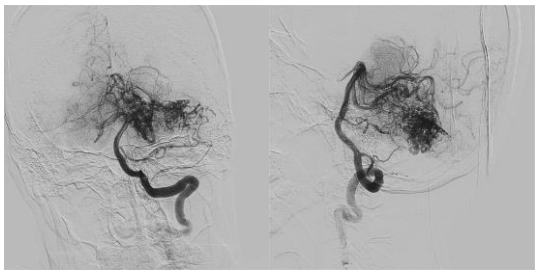

Figure 4. AP (left) and lateral projection (right) of left vertebral artery DSA after embolization, revealing 30% reduction of nidus size

of the AVM, as confirmed by post-procedural angiography. This reduction likely alleviated compression of the facial nerve root, resulting in notable clinical improvement.

Further evaluation is warranted in patients with HFS unresponsive to conservative management, especially when there is a change in the symptom pattern or emergence of additional neurological deficits.

HFS and Cerebellar AVM

In this case, left-sided hemifacial spasm was attributed to compression of the facial nerve, indicative of a neurovascular compression syndrome–a particularly rare phenomenon.[5,9,10] Decompression of the affected nerve is expected to alleviatesymptoms. By reducing blood flow to the AVM, it is hypothesized that the nidus shrinks, thereby lessening nerve compression and improving cranial nerve function.[6]

Post-procedural angiography confirmed reduced AVM blood flow, which correlated with symptom relief.[6,11] Studies have demonstrated a strong correlation between clinical improvement (as assessed by the SMC grading system) and enhanced quality of life, evaluated using the HFS-7 score.[12]

Palliative Embolization

Palliative embolization of an AVM is typically performed to reduce its size, alleviate symptoms, or lower the risk of hemorrhage. Although not always curative, embolization can improve symptoms by reducing mass effect or reducing hemodynamic stress on surrounding neural structures.[6,11] The most recent similar case was published in 1989 by Yang *et al*, in which endovascular embolization was employed to relieve HFS. Embolization was performed using hand-cut polyvinyl alcohol (PVA) particles (1–2 mm in diameter), and no complications were reported.[13]

Although particulate embolic agents like PVA were commonly used in earlier years, they have largely been replaced by liquid embolic agents (LEAs) due to the higher recurrence rates. Among LEAs, Onyx has largely supplanted N-BCA owing to its higher angiographic cure rates. Nevertheless, N-BCA remains a viable option due to its lower rates of permanent complications, broader availability and greater accessibility.[14]

By reducing the size or flow of the AVM, embolization may relieve facial nerve compression or irritation, thereby improving or resolving HFS. Patient selection is critical, as not all patients with concurrent HFS and AVM will benefit from embolization.[15] Detailed evaluation of the anatomical relationship between the AVM and facial nerve is essential.

Embolization carries inherent risks, including stroke, hemorrhage, and incomplete treatment.[11,15] The decision to proceed must carefully weigh potential benefits against associated risks. In this case, palliative embolization was preferred due to the higher risk of hemorrhage associated with posterior fossa AVMs and the lesion's location within eloquent brain areas. Although a thrombus developed intra-procedurally, it was successfully managed without resulting in postoperative deficits.

While this report presents only short-term outcomes, longer-term follow-up is necessary to monitor for AVM recurrence and assess rupture risk.[16] Continued surveillance should be rigorous, and decision regarding further intervention–such as surgery–should involve a multidisciplinary team, including neurologists, neuroradiologists, and interventional neurologists, to ensure an optimal, individualizedtreatment plan.

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Conclusion

We report a case of left-sided HFS caused by a cerebellar AVM that significantly improved followingpalliative embolization. Although AVM-associated HFS is rare, careful evaluation of the anatomical relationship is essential in determining whether intervention may yield clinical benefit.

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Conflict of Interest
The authors of this article declared no conflict of interest

Patient consent for publication
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Author contribution
All authors contributed to the conception, data collection, analysis, drafting, and critical revision of this case report. All authors have read and approved the final manuscript.

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