Hemifacial Spasm Caused by Compression of a Vertebral Artery Aneurysm Consequently Improved by Stent-assisted Coil Embolization

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Objective: We described a rare hemifacial spasm (HFS) caused by compression of a vertebral artery (VA) aneurysm that was consequently improved by stent-assisted coil embolization.

Case Presentation: A 60-year-old man presented with a chief complaint of left HFS that had persisted for 1 month. It had initially appeared in the orbicularis oculi, spread to the orbicularis oris, and severely disrupted his quality of life. Both MRI and MRA revealed a wide-necked aneurysm of the left VA (neck 8.5 mm, dome 6.0 mm) compressing the left facial nerve root exit zone (REZ). We performed stent-assisted coil embolization because the VA was dominant at this side of the aneurysm and we tried to preserve normal antegrade flow. The HFS disappeared immediately after embolization without complications. After 6 month follow-up, the patient had no recurrence of symptoms and MRA showed no recurrence of the aneurysm.

Conclusion: Stent-assisted coil embolization was effective for treating HFS caused by compression of a VA aneurysm and it might be the treatment of choice for this type of aneurysmal HFS.

Keywords: hemifacial spasm, vertebral artery, aneurysm, endovascular, stent-assisted

Introduction

Hemifacial spasm (HFS) is characterized by unintentional intermittent unilateral painless contractions of facial muscles that are typically caused by arterial compression of the facial nerve root exit zone (REZ). Microvascular decompression (MVD) is the standard of choice for treatment of typical HFS caused by contributed arteries. Intracranial aneurysms are rare causes of HFS. Several reports have indicated that endovascular parent artery occlusion is useful for treating HFS caused by aneurysms. We describe the first case of stent-assisted coil embolization improving HFS caused by VA aneurysm.

Case Presentation

A 60-year-old previously healthy man presented with a chief complaint of left HFS that had persisted for 1 month. The spasm had initially appeared in the orbicularis oculi, then spread to the orbicularis oris, and severely disrupted his quality of life. Both MRI and MRA showed a wide-necked aneurysm of the left VA (neck 8.5 mm, dome 6.0 mm) compressing the left facial nerve REZ (Fig. 1).

The posterior inferior cerebellar artery was not involved in the aneurysm. Considering the quality of life of the patient disturbed by HFS and the potential risk of rupture, we decided to treat the patient. Direct aneurysm trapping and MVD by open craniotomy or endovascular internal trapping with parent artery occlusion or stent-assisted coil embolization were considered. Because the VA was dominant at this side of the aneurysm and we tried to preserve normal antegrade flow, we performed stent-assisted coil embolization.
embolization were included as treatment options. We selected stent-assisted coil embolization instead of internal trapping with parent artery occlusion because the affected VA was dominant and the aneurysm was unruptured and we tried to preserve normal antegrade flow. We also expected that the HFS might be improved because the slight change of the arterial course and the attenuation of continuous pulsation of the aneurysm to the REZ could be achieved by stent-assisted coil embolization. We obtained written, informed consent from the patient.

The antiplatelet agents (aspirin 100 mg/day and clopidogrel 75 mg/day) were administered for 14 days before the procedure. Under general anesthesia, a 6 Fr FUBUKI 90-cm guiding catheter (Asahi Intecc, Aichi, Japan) was inserted via the right femoral artery into the left VA (Fig. 2A and 2B). Heparin was administered and activated clotting time was controlled between 250 and 300 s. A J-shaped Excelsior SL-10 straight 150-cm catheter (Stryker, Kalamazoo, MI, USA) was navigated into the aneurysm for coil delivery under roadmap guidance. An Excelsior SL-10 straight 150-cm catheter was then placed in the basilar artery for stent delivery. A Neuroform atlas 4.5 mm × 30 mm stent (Stryker) was deployed from just proximal to the vertebrobasilar junction across the aneurysmal orifice. The stent-vessel wall apposition was assessed by cone-beam CT (Fig. 2C). The first coil was a Target Soft 6 × 20 (Stryker) and then five Target Ultrasoft (Stryker), six Axium Prime (Medtronic, Minneapolis, MN, USA), and seven Target Ultrasoft (Stryker) were delivered, respectively. A total of 19 coils (total length of coils; 108 cm) were delivered and an adequate embolization for prevention of aneurysmal rupture could be achieved (Fig. 2D and 2E). We intentionally finished embolization with body filling avoiding tight packing of the aneurysmal dome because we considered that the mass effect for REZ by coils could worsen the HFS though the possibility of aneurysmal recurrence would be higher than tight packing. The HFS immediately disappeared after the procedure. MRI and MRA obtained the day after the procedure confirmed the absence of aneurysmal flow (Fig. 3A). The patient was discharged after 12 days after the procedure without any neurological deficits. Six months after the procedure, the patient had no recurrence of symptoms and MRA showed no recurrence of the aneurysm (Fig. 3B).

Discussion

Stent-assisted coil embolization was useful as a treatment for aneurysm and HFS in this case. We found 15 patients with HFS caused by VA aneurysm in literatures, in which five were treated using an endovascular procedure. All of these patients had some type of parent artery occlusion. One had only proximal occlusion, one had endovascular trapping without an aneurysmal embolization, and one and two had proximal occlusion after rough and tight embolization of aneurysms, respectively. All the reports indicated that the symptoms disappeared within 6 months.

In the present case, we should treat both unruptured VA aneurysm and life disturbing HFS. We performed stent-assisted coil embolization for preserving antegrade flow of
hearing disturbance and lower cranial nerve damage is slightly higher in MVD for VA-associated HFS because of the difficulties with transposition of VA. Therefore, endovascular treatment might be preferable for the treatment of the patient.

Nagashima et al. reported that endovascular treatment often improves HFS after about 3–6 months of follow-up. Some reports have indicated that the risk of a
We therefore planned to observe for 6 months. Then, taking the patient’s symptom and the state of the aneurysm into account, we would make additional treatment such as endovascular internal trapping or direct MVD and VA trapping with occipital artery to posterior inferior cerebellar artery bypass or only direct MVD or botulinum neurotoxin injections.

Stent-assisted coil embolization may have a possibility to worsen HFS because stents and coils might increase the mass effect of the REZ; however, the HFS rapidly disappeared after the procedure in our patient. The minimal change of running course of the VA could affect the REZ. Additionally, attenuated continuous pulsation of the aneurysm to the REZ by decreasing blood flow in the aneurysm might improve the HFS. Considering the mass effect of coils, avoiding tight packing might be important for symptom relief. Anatomical relationships among the parent artery, the aneurysm, and the REZ should be considered in detail when deciding the treatment strategy.

We could find no reports of stent-assisted coil embolization for HFS caused by a VA aneurysm. The present case report is, therefore, the first one.

**Conclusion**

We treated HFS caused by compression of a VA aneurysm with stent-assisted coil embolization. Stent-assisted coil embolization was effective for treating HFS caused by compression of a VA aneurysm and it might be the treatment of choice for this type of aneurysmal HFS.

**Disclosure Statement**

All authors have no conflict of interest.

**References**