Ruptured dissecting aneurysm of the vertebral artery requires a rapid treatment because of the high rate of fatal early rebleeding. Stent-assisted coil embolization has been introduced as an effective procedure for both aneurysmal occlusion and parent artery preservation. We report a case of successful urgent treatment of ruptured dissecting aneurysm using a self-expandable, intracranial stent (Neuroform®) and a coated coil (HydroCoil®).

Key Words: Vertebral artery; Coil embolization; Dissecting Aneurysm; Stent

Ruptured dissecting aneurysm of the vertebral artery (VADA) necessitates an urgent treatment to prevent a fatal early rebleeding (1-3). The endovascular procedure has been suggested as a rational approach in the management of acute ruptured VADAs (1-4). Although stent-assisted coil embolization has been introduced as an effective endovascular procedure for both durable aneurysmal occlusion and parent artery preservation, some limitations were reported as well (5, 6). We report an urgently treated case of a ruptured VADA in a patient with a contralateral hypoplastic vertebral artery, using a self-expandable, flexible, intracranial nitinol stent (Neuroform®: Boston Scientific/Target, Fremont, CA) and a self-expandable bioactive hydrophilic coil (HydroCoil®: MicroVention, Aliso Viejo, CA).

CASE REPORT

A 40-year-old woman was admitted to our emergency room because of a sudden, severe headache and altered mental status. The brain CT scans showed a subarachnoid hemorrhage. Emergency cerebral angiograms demonstrated a dissecting aneurysm, 8mm long and 3mm wide, at the left vertebral artery (VA) distal to the origin of the cranial minor loop of the duplicated left posterior inferior cerebellar artery (PICA). The right VA was hypoplastic, and collateral blood flow from the posterior communicating artery was not satisfactory (Fig. 1A). After obtaining informed consent, we performed emergency stent-assisted coil embolization. Under general anesthesia, we catheterized the left VA by using microcatheter and wire. We introduced a 300-cm Transend exchange wire with its tip placed in the P2 portion of the right posterior cerebral artery and withdrew the microcatheter. A 3×20-mm Neuroform stent was...
advanced over the exchange wire and the stent was placed across the neck of the aneurysmal sac and over the intimal flap, using roadmap guidance. The control angiogram showed good coverage of the neck of the pseudoaneurysmal sac and well-preserved distal blood flow. There was no visible in-stent thrombosis or distal embolization. On the follow-up angiogram at 20 minutes, the contrast-filling of the aneurysmal sac was decreased, but residual sac was still visible. A Prowler 14 microcatheter was then introduced over a Synchro wire and placed through the struts of the stent into the residual pseudoaneurysmal sac, and a $2 \times 20$ helical HydroCoil 10 was placed in the sac without difficulty (Fig. 1B). We then initiated intravenous heparinization to achieve an activated clotting time (ACT) of 250-300 seconds. Control angiograms 20 minutes later showed progressive occlusion of the pseudoaneurysmal sac due to coil expansion, with faint neck filling. The patient's vital signs were stable. Her heparin anticoagulation was stopped at the end of the procedure. The final ACT was 280 seconds. A loading dose of clopidogrel (325 mg) and aspirin (100 mg) for antiplatelet therapy were administrated through the nasogastric tube on the same day of the procedure. On the next day, she received 75 mg clopidogrel and 100 mg aspirin as the beginning of maintenance. There were no newly detected neurologic symptoms or bleeding episodes during admission. Follow-up angiograms 1 week later demonstrated preserved anatomic blood flow of the left VA, a well-visible small cranial loop of the left PICA, and total obliteration of pseudoaneurysmal sac (Fig. 1C). There were no visible in-stent thromboses or distal thromboembolic complications. She was discharged 1 week after the procedure. After maintaining dual antiplatelet therapy for 3 months, follow-up angiography will be performed. After that, our plan will be to maintain aspirin at 100 mg per day as lifelong therapy.

**Fig. 1.** A. Left vertebral artery (VA) digital subtraction angiography (DSA: oblique projection) shows an intimal flap and dissecting aneurysmal sac (arrowhead) at the distal portion of the origin of the cranial minor loop of the duplicated posterior inferior cerebellar artery (PICA). Hypoplastic right VA is also noted. B. DSA after a coil insertion into the residual aneurysmal sac. A $2 \times 2$ cm Hydrocoil was placed in the residual sac through the struts of Neuroform stent. Minimal aneurysmal neck remnant is visible (arrowhead). There was no evidence of stent-coil associated complications (arrows: proximal and distal markers of the stent). C. The follow-up angiogram 1 week later demonstrated preserved anatomic blood flow and patency of the left vertebral artery, well-visible small cranial loop of the left PICA, and total obliteration of the aneurysmal sac (arrowhead).
DISCUSSION

The increasing incidences of ruptured VADA as a cause of subarachnoid hemorrhage have been documented in many reports (1, 2, 7). One of the main concerns noted in reports about ruptured VADA was a high rebleeding rate (30–71%), generally occurring within the first 24 hrs (2). Patients with rebleeding show a high morbidity and mortality rate (2). Therefore, acute treatment for preventing fatal rebleeding is highly desirable (3, 7). Several authors have reported that some surgical strategies for ruptured VADA were inappropriate in the acute stage, due to complications (2, 8). Endovascular treatment of acute ruptured VADA can be divided into deconstructive or reconstructive methods. Deconstructive methods include proximal VA occlusion or internal trapping of the dissected segment, using detachable coils. These procedures were considered to be effective and useful methods for treating ruptured VADAs in their acute stage, but there is a basic risk of ischemia or infarction in the posterior circulation, especially in the presence of a hypoplastic contralateral VA, such as occurred in our case. Therefore, our case needed a reconstructive method, including double stent placement or stent-assisted coil embolization. This stent-assisted coil embolization not only could preserve the parent artery flow but provide occlusive coil placement in the pseudoaneurysmal sac.

Stent-assisted coil embolization has been reported as a novel option for the treatment of the acute stage of a ruptured VADA, but some limitations have been reported, including inflexibility or migration of stent, difficulty in navigating the microcatheter through the stent strut, injury to the dissected arterial wall, a persistent residual aneurysmal sac on follow-up, herniation of coils, and complications such as rebleeding (5, 6). To our knowledge, this report represents the first case of urgent treatment using a Neuroform stent and a HydroCoil for a ruptured VADA in a patient with a contralateral hypoplastic VA. The Neuroform stent is a self-expandable, nitinol, intracranial stent. Its 35-μstrut thickness and open-cell design permit significant flexibility and easy microcatheter delivery through the struts into the aneurysmal sac. Their low radial force of only 9–10 mm Hg, similar to the physiological force of native vessels, may reduce injury to the labile dissected arterial wall (9).

The HydroCoil is a platinum coil with an expandable hydrogel that provides delayed progressive expansion of the coil when it contacts a water-based substance, such as blood, with a pH of 7.4. Two advantages of the HydroCoil are the increased packing density in the aneurysmal sac and advanced healing and endothelialization at the aneurysmal neck (10).

The main problems of these devices are thromboembolic complications. Thus, strict anticoagulant and antiplatelet therapy are recommended, but the optimal regimen has not yet been demonstrated. Our anticoagulation protocol for this patient was as follows: intravenous heparin injection after the introduction of the coil, to achieve an ACT of 250–300 seconds; a loading dose of clopidogrel and aspirin at the end of the procedure, and maintenance of the dual antiplatelet regimens for 3 months. No periprocedural complications, including thromboembolic events, were encountered in our case.

Combined therapy with a Neuroform stent and HydroCoil may be a new and useful option for managing an acute ruptured VADA event in patients with a hypoplastic contralateral VA. We report a case of successful urgent management for ruptured pseudoaneurysm in the dominant VA using these interventions. Further investigation and collection of additional data about the long-term safety and efficacy of this procedure and of device-related complications will be needed.

References
by using stents and coils. J Neurosurg 2001; 94:427-432

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