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Review Article

Endovascular Treatment of Intracranial Vertebral Artery Dissection

Joonho Chung,[1] Yong Cheol Lim,[2] and Yong Sam Shin[3]

Intracranial vertebral artery dissection (VAD) is the most common arterial dissection in intracranial arteries. Some types of VAD can heal spontaneously after reconstitution of the vessel lumen with excellent prognosis, whereas others can progress to stroke that needs treatment. Recently, endovascular treatment (EVT) has emerged and is suggested as a treatment option for VADs due to perceived low rates of procedure-related morbidity with good efficacy. In the last decade, we have accumulated our strategies to treat those VADs. Here, we try to share our experiences about VADs, including indications and methods of treatment of VADs using EVT. We perform EVT for ruptured VADs presenting with SAH and some of unruptured VADs such as VAD with recurrent or progressive ischemia, dissecting aneurysm larger than 7 mm or with mass effect, early ugly change of VADs in shape and size during follow-up period, involving the basilar artery (BA) and bilateral VADs. We present how we have done in our real practice for the last decade for treating VADs by EVT rather than reviewing and organizing so-far-published literature. We tended to occlude the rupture point by vertebral artery (VA) occlusion in non-dominant VA or stent-assisted coiling in dominant VA for ruptured VADs. We tended to reconstruct original hemodynamics using various stents for unruptured VADs. To decide what to treat and how to treat are very complicated for VADs. However, we believe that EVT is the current mainstay for treating VADs. Each technique of EVT should be determined on a case-by-case basis at the discretion of endovascular neurosurgeons and/or interventional neuroradiologists according to presenting symptoms, hemodynamic status, including sufficiency of the collateral supply and anatomic features of the vertebrobasilar artery as well as the posterior inferior cerebellar artery, anterior spinal artery, and medullary perforators.

Keywords ▶ endovascular treatment, dissection, dissecting aneurysm, vertebral artery

Introduction

Intracranial vertebral artery dissection (VAD) is the most common arterial dissection in intracranial arteries. Some types of VAD can heal spontaneously after reconstitution of the vessel lumen with excellent prognosis, whereas others can progress to stroke that needs treatment.[1)] Stroke can manifest as brainstem involvement, cerebellar infarction, or subarachnoid hemorrhage (SAH).[2)] Spontaneous intracranial VAD is classified into three major clinical types: headache, non-hemorrhagic ischemic symptoms, and SAH.[3)] Arterial dissection is initiated by sudden disruption of internal elastic lamina and media. Circulating blood accumulation in the arterial wall following wall disruption can promote intramural hematoma formation.[4)] In patients having arterial dissection with a steno-occlusive pattern, subintimal hematoma and intimal flap might cause luminal narrowing and flow restriction.[5,6)]

Recently, endovascular treatment (EVT) has emerged and is suggested as a treatment option for VADs due to perceived low rates of procedure-related morbidity with good efficacy.[3,7–10)] The choice of treatment method for patients with VAD depends on the patency of the contralateral vertebral artery (VA) as well as the relationship between the

[1]*Department of Neurosurgery, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea*
[2]*Department of Neurosurgery, Ajou University College of Medicine and Hospital, Suwon, Korea*
[3]*Department of Neurosurgery, Seoul St. Mary's Hospital, The Catholic University of Korea College of Medicine, Seoul, Korea*

Received: July 22, 2020; Accepted: August 23, 2020
Corresponding author: Yong Sam Shin. Department of Neurosurgery, St. Mary's Hospital, The Catholic University of Korea College of Medicine, 222, Banpo-daero, Seocho-gu, Seoul, 137-701, Korea
Email: nsshin@gmail.com
J. Chung and Y.C. Lim contributed equally to this study as co-first author.

dissection segment and the location of the posterior inferior cerebellar artery (PICA) origin. It has been known that complete occlusion of the dissecting segment is an optimal treatment for VAD.[7–9,11] If the dissection is located proximal or distal to the PICA without hypoplasia of the contralateral VA, then the lesion can be treated with total occlusion of the dissecting segment using coils (internal trapping) so that the PICA could be filled from the contralateral or ipsilateral VA, respectively.[12] If the VADs involve the PICA origin, complete isolation of the dissected segment can be achieved by internal coil trapping with revascularization of the PICA through bypass surgery[12–14] or PICA stenting.[7–9] In such circumstances, EVT has shown good results owing to the development of devices and methods such as VA trapping with VA-PICA stenting, multiple stenting, flow-diverting stenting (FDS), and stent-assisted coil embolization (SAC).[7–9,15–17] Previous studies have concluded that these EVT methods can lead to favorable outcomes. In the last decade, we have accumulated our strategies to treat those VADs. Here, we try to share our experiences about VADs, including indications and methods of treatment of VADs using EVT.

Diagnosis and Treatment Indications

We make a diagnosis of VAD if patients meet the following inclusion criteria: (a) a history of acute clinical symptoms and/or signs relevant to intracranial VAD, (b) angiographic evidence of VAD (i.e., aneurysmal dilatation of the intracranial VA, pearl-and-string sign, or tapered steno-occlusion), and (c) available results from digital subtraction angiography (DSA) as well as magnetic resonance (MR) imaging and/or computed tomographic angiography (CTA) performed at the time of symptom onset. We distinguish VADs with tapered steno-occlusion from atherosclerosis using MR vessel wall images, CTA source images, or contrast-enhanced MR source images. We exclude patients if they meet the following: (a) definitively traumatic VAD secondary to major trauma, (b) iatrogenic VAD, (c) incidentally found asymptomatic fusiform dilatations of the VAs, (d) laboratory or angiographic findings suggestive of vasculitis or fibromuscular dysplasia, or (e) the lack of documented MR or CTA during the initial evaluation.

We perform EVT for intracranial VADs with the following indications:

(1) ruptured VADs presenting with SAH

(2) unruptured VADs

- VAD with recurrent or progressive ischemia
- Dissecting aneurysm larger than 7 mm or with mass effect
- early ugly change of VADs in shape and size during follow-up period
- Involving the basilar artery (BA)
- Bilateral VADs

Ruptured VADs presenting with SAH

VADs presenting with SAH are unstable with a high risk of rebleeding.[14,18,19] Hemodynamic stress on the vessel wall can produce rebleeding. The rebleeding rate has been reported to be as high as 71.4% in a group of 42 untreated patients.[19] The mortality rate of these rebleeding cases was high, being 46.7%.[19] Therefore, therapeutic intervention should be performed as soon as possible to prevent any subsequent bleeding. EVT can decrease hemodynamic stress and provide a suitable environment for healing. An ideal treatment for patients with SAH from VADs is complete occlusion of the dissecting segment from circulation by performing EVT, such as internal coil trapping.

Unruptured VADs

Currently, there is no consensus on the proper management strategies for unruptured VADs. However, EVT has been favored as a treatment option for VADs because microsurgery carries a significant risk of cranial nerve palsy and brainstem injury. Intracranial VADs may lead to narrowing of the VA and subsequent occlusion, resulting in posterior circulation thromboembolic ischemia.[2] Ischemic stroke in posterior fossa owing to brainstem involvement may lead to high mortality and morbidity with various neurologic deficits.[20] In addition, patients with ischemic symptoms have unfavorable outcomes compared to patients without ischemic presentation.[21] These observations suggest that EVT for VADs in the acute phase should be focused more on preventing progression or recurrence of ischemic stroke than on preventing SAH, especially in patients with ischemic presentations. In addition, the preventive EVT can be performed in patients whose angiographic results showed increased risk of rupture (e.g., an enlarging dissecting aneurysm). Although most unruptured VADs can heal spontaneously, several cases of unruptured VADs that subsequently cause SAH have been reported.[10,22–24] If there are ugly changes in shape and size of VADs during follow-up or if dissecting aneurysm is larger than 7 mm and/or with mass effect, the risk of rupture may increase. Therefore, EVT should be performed for the cases with progressive ischemia or recurrent ischemic symptoms despite medication[25,26] and for those with an enlarged dissecting aneurysm on follow-up angiographic imaging.[27,28]

For unruptured VADs, whether the dissection affects the BA appears to be of critical importance in clinical outcomes. Previously published reports have shown that BA involvement of VADs is an independent predictor of unfavorable outcome.[21,29–32] Additionally, the initial severity of ischemic symptoms (higher baseline NIHSS score), bilateral VA involvement of the dissection, and intracranial VA involvement have been suggested as predictors of unfavorable outcomes in unruptured VADs.[21,33,34] Thus, we can perform EVT for VADs involving BA and bilateral VADs.

Endovascular Techniques

While treating ruptured VADs, heparin is not injected intravenously or subcutaneously. Instead, it is mixed into the saline flushes during the procedure. Thus, the activated clotting time is not checked during the procedure. When we use stents, loading doses of clopidogrel (300 mg) and aspirin (300 mg) are given via the nasogastric tube after femoral artery puncture.[35] Post-procedural surgical management such as external ventricular drainage and ventriculo-peritoneal shunt insertion can be conducted without discontinuation of the antiplatelet agents.

For symptomatic unruptured VADs, patients take a daily dose of 75 mg of clopidogrel and 100 mg of aspirin for more than 5 days before EVT. We perform the platelet aggregation test preoperatively after prescribing dual-antiplatelet agents. However, we do not modify the medication for poor responders because aspirin or clopidogrel resistance did not show significant relationships with acute and delayed thromboembolism in SAC of unruptured intracranial aneurysms in our series. During the procedure, patients receive an intravenous heparin load of 50 IU/kg immediately after guiding catheter placement and activated clotting time is maintained at twice that of the baseline throughout the endovascular procedure. Heparin is stopped immediately after completion of the procedure.

If thromboembolic complications are encountered during the procedure, 1 mg or less of glycoprotein IIb/IIIa antagonist (tirofiban) is injected intra-arterially. After the procedure, patients who have undergone EVT using stents are prescribed 75 mg of clopidogrel daily for 3 months and 100 mg of aspirin daily for at least 12 months. Patients who have previously used anticoagulants are prescribed the same anticoagulant and aspirin (100 mg/day). In cases of FDS, patients take 75 mg of clopidogrel daily for at least 6 months and 100 mg of aspirin daily for at least 24 months. Follow-up angiography is usually performed within a month, at 3–6 months, and then at 12–24 months for ruptured VADs. It is performed at 3–6 months and then at 12–24 months for symptomatic unruptured VADs.

Deconstructive Technique

A deconstructive technique means parent artery occlusion by EVT, such as internal coil trapping or proximal coil occlusion, in which blood that flows into the dissected segment of VA is stopped. Preferably, the dissected segment is occluded both proximally and distally to prevent rebleeding through retrograde filling of a dissecting aneurysm. A deconstructive technique has a risk of ischemic stroke in case of insufficient collateral supply. Thus, it is recommended for VADs in non-dominant VA or VA with sufficient collaterals. If the dissection is not completely excluded from the antegrade arterial circulation following proximal occlusion, the potential for rebleeding still exists. In addition, rebleeding can occur when the dissection cavity increases in size after proximal occlusion. Thus, a deconstructive technique is preferred and has benefits for ruptured VADs because it can prevent rebleeding, reduce recurrence, and allow additional surgical procedures such as external ventricular drainage and decompressive surgery. SAC and flow diversion (multiple stenting or FDS) can preserve parent artery flow. However, patients must strictly adhere to antiplatelet therapy to prevent in-stent thrombosis. In the acute period, antiplatelet therapy carries some risk of parenchymal hemorrhage if placement of a ventricular drain is necessary. Additionally, with flow diversion, the risk of re-hemorrhage in the acute period prior to endothelial remodeling is unknown. Therefore, we prefer deconstructive methods for ruptured VADs in non-dominant VA.

VA trapping (occlusion) by coiling

If the dissection is located proximal or distal to the PICA without hypoplasia of the contralateral VA, then the lesion can be treated with total occlusion of the dissecting segment with coils (internal trapping) so that the PICA could be filled from the contralateral or ipsilateral VA, respectively, after embolization.[12] We perform VA trapping by coiling. It sacrifices the VA, making complete isolation of the dissected segment from the circulation. This method is usually applied for VADs that do not involve the PICA origin (post-PICA or pre-PICA segment) in non-dominant VA by either internal coil trapping or proximal coil occlusion. A proximal occlusion of the VA can be performed using

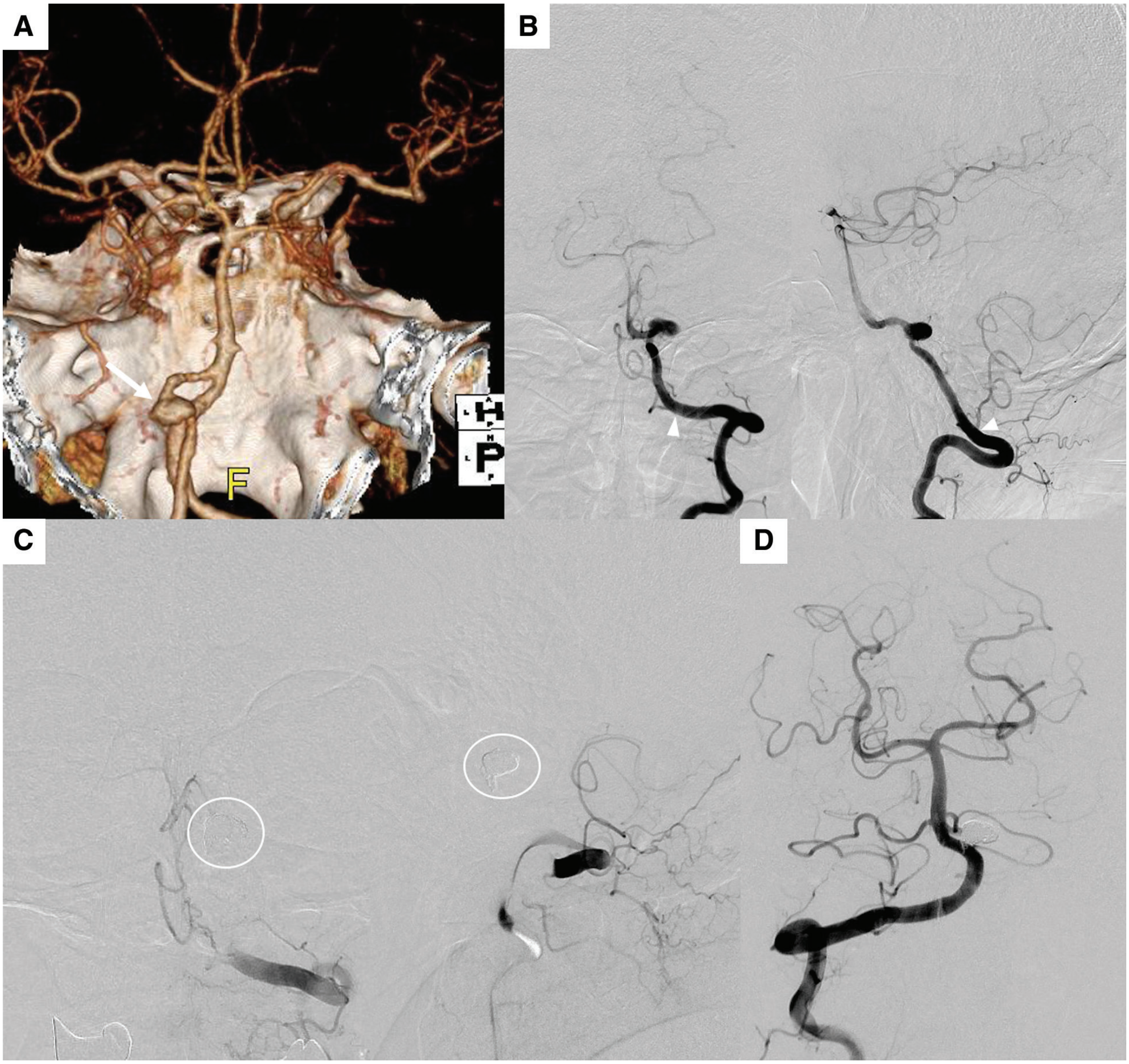

Fig. 1 (**A**) CTA showing a ruptured dissecting aneurysm (white arrow) in non-dominant left VA. (**B**) DSA showing a dissecting aneurysm distal to the extradural origin of the PICA (white arrowheads). (**C**) Dissecting aneurysm of the left VA is completely occluded by internal coil trapping. Coils are seen in *white circles*. (**D**) The flow of the posterior circulation seems to be sufficient from the dominant right VA on DSA. CTA: computed tomographic angiography; DSA: digital subtraction angiography; PICA: posterior inferior cerebellar artery; VA: vertebral artery

coils at the segment proximal to the VAD. Internal coil trapping is coil embolization of the VA at the dissected segment (**Fig. 1**). When we consider a deconstructive method for cases in dominant VA, we perform a balloon test occlusion at the ipsilateral VA proximal to the involved segment.

VA trapping (occlusion) with PICA stenting

When a VAD involves the origin of the PICA in non-dominant VA, there is a high risk of PICA infarction accompanied by internal coil trapping.[8,36] In terms of EVT, reconstruction therapy is indicated for patients with VADs involving the origin of the PICA.[37] However, we perform internal coil trapping for VAD that involves the PICA origin in non-dominant VA. The dissected segment, including the ruptured portion that either had a daughter sac or was the most notably dilated, can be occluded by coil embolization while preserving the PICA by the PICA stenting. In 2010, for the first time, we reported on a patient who was treated with a self-expanding closed-cell Enterprise stent (Codman Neurovascular, Miami Lakes, FL, USA) from the proximal VA to the PICA. We completely occluded the dissected segment by coiling (**Fig. 2**). When the origin of the

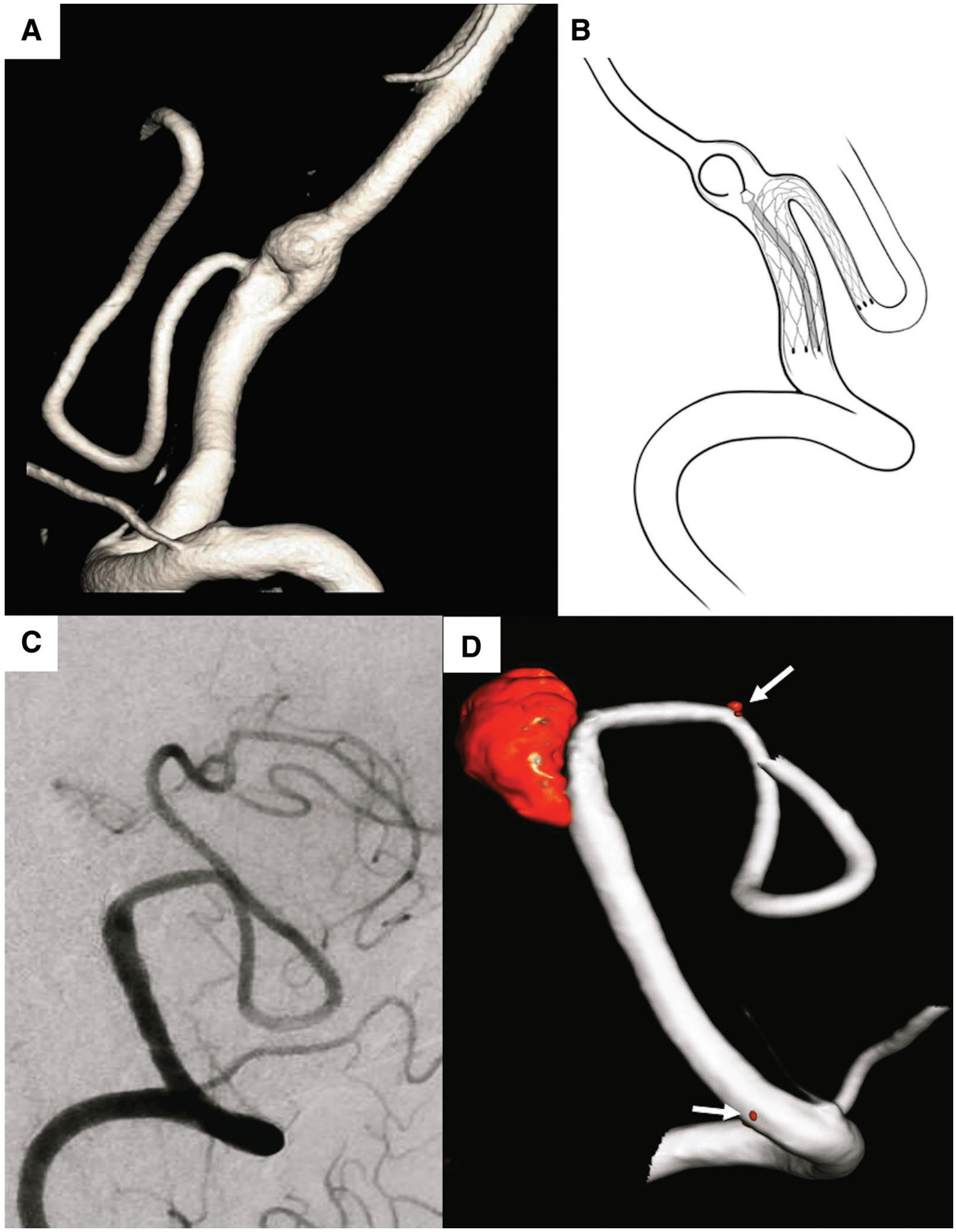

Fig. 2 (**A**) Three-dimensional reconstruction image showing a ruptured VAD with involvement of the origin of the PICA in a non-dominant VA. (**B**) Illustration of VA-to-PICA stenting with coil embolization. (**C**) One-year follow-up angiography. An Enterprise stent was placed through the proximal VA to the PICA. The dissected segment was completely occluded by coil embolization. It was found that the VA-PICA angle became larger as the stent had been unfolded by its returning force to become straight, resulting in an increase in the diameter of the PICA. (**D**) Three-dimensional reconstruction image showing PICA patency. The arrows indicate proximal and distal stent markers in the VA and the PICA. PICA: posterior inferior cerebellar artery; VA: vertebral artery; VAD: vertebral artery dissection

PICA involves the distal segment of VAD, we can perform internal coil trapping of VAD combined with PICA stenting by placing an Enterprise stent from the distal VA to the PICA approached from contralateral VA (**Fig. 3**).

When we evaluated clinical and radiologic outcomes of VADs involving the PICA origin according to different types of EVT (VA trapping with VA-PICA stenting, multiple stenting, SAC, and FDS), VA trapping with VA-PICA stenting showed the lowest rate of recurrence with a high rate of minor infarction and favorable neurologic outcome. Stent-assisted coil embolization shows high recurrence rates with possible fatal disabling infarction.[7)]

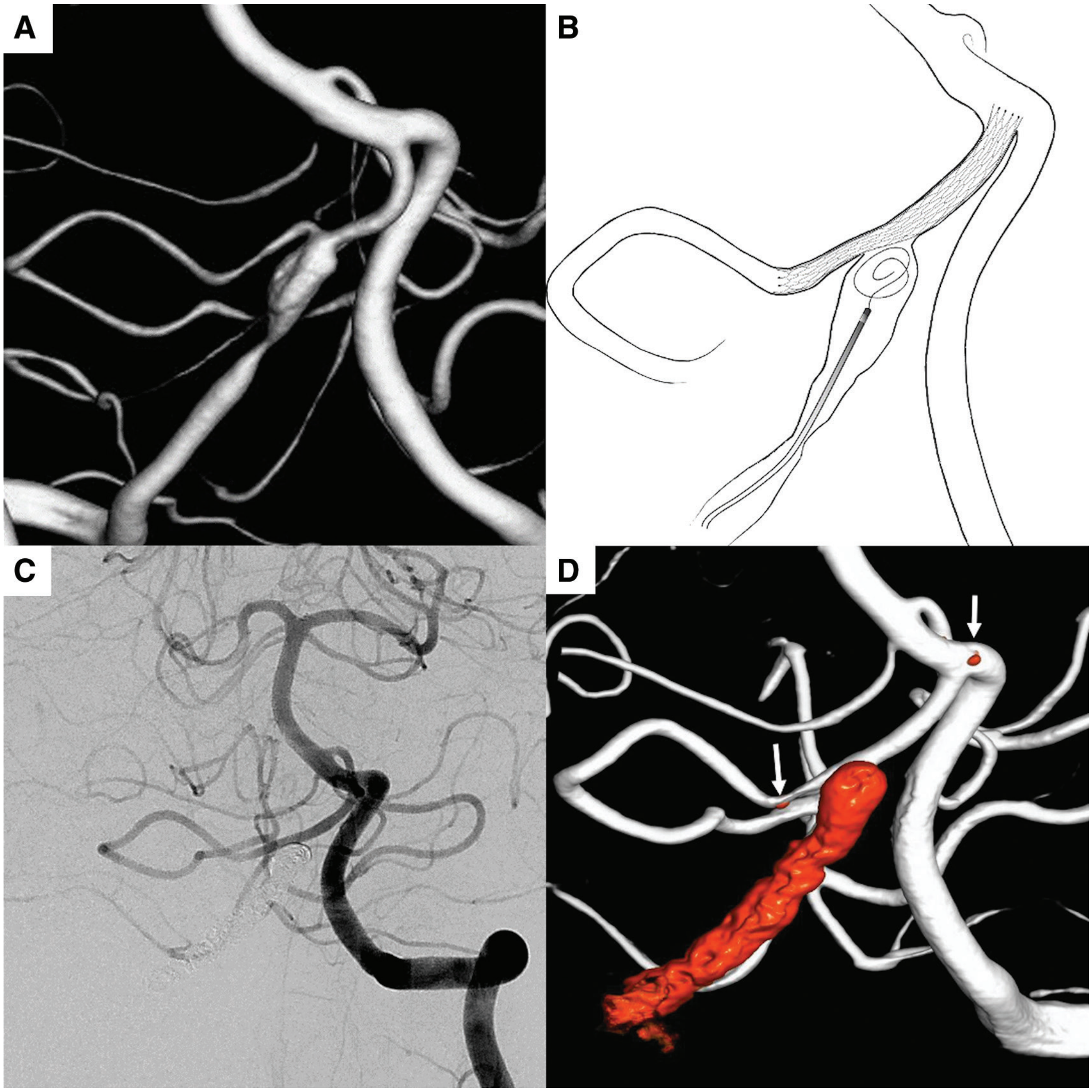

Fig. 3 (**A**) Three-dimensional reconstruction image showing a ruptured VAD and the origin of the PICA involving the distal segment of dissection in a non-dominant VA. (**B**) Illustration of the VA-to-PICA stenting with coil embolization. (**C**) Six-month follow-up angiography. An Enterprise stent was placed through the distal VA to the PICA approached from the contralateral VA. The dissected segment was completely occluded by coil embolization, revealing good PICA patency. (**D**) Three-dimensional reconstruction image showing good PICA patency. The arrows indicate proximal and distal stent markers in the VA and the PICA. PICA: posterior inferior cerebellar artery; VA: vertebral artery; VAD: vertebral artery dissection

In terms of PICA patency after VA-PICA stenting, we have reported our experience using Enterprise stents deployed in small arteries (<2 mm in diameter).[38] Among 31 patients enrolled, 3 (9.7%) patients experienced procedure-related complications. They were all asymptomatic. Follow-up angiography was performed in 27 (87.1%) patients at a mean of 15.5 months after EVT. Parent arteries with two acute angles (n = 4) were occluded in three (75.0%) cases. Those with no acute angles (n = 13) or one acute angle (n = 6) showed 100% patency on follow-up angiography. Follow-up sizes of parent arteries and their sizes before EVT were significantly different ($P = 0.037$).[38] On multivariate logistic regression analysis, the tortuosity (the number of acute angles) of parent arteries was determined to be the only predisposing factor for size increments of parent arteries on follow-up angiography. Furthermore, successful navigation and deployment of stents were achieved (100%) without symptomatic procedure-related complications. These results suggest that angiographic vascular configuration and traveling course of parent arteries might be important factors for maintaining the patency of parent arteries with diameter <2 mm. Stent deployment in small parent arteries seems to be safe from a technical viewpoint. According to our results, the PICA usually does not have more than two acute angles. Therefore, we believe that PICA patency after VA-PICA stenting can be guaranteed.

Reconstructive Technique

Deconstructive technique of the affected VA is not always possible, such as for cases of VAD in the dominant or single VA with poor collateral flow or bilateral VADs. For such cases, reconstructive techniques that can preserve the parent artery using stents alone or with coiling have been increasing used. They might be appropriate alternatives to vessel deconstruction.[39] While a deconstructive technique shows a higher rate of complete angiographic occlusion, a reconstructive technique is associated with less perioperative morbidity. Thus, endovascular reconstruction of VADs has been performed more recently using multiple stenting (stent-in-stent technique), SAC, or employing FDS with the aim to prevent ischemic sequelae while restoring original hemodynamics.[40–44] As mentioned above, all reconstructive techniques require a relevant period of dual-antiplatelet therapy in the setting of SAH which may jeopardize further neurosurgical interventions such as external ventricular drainage and craniectomy. However, reconstructive technique is believed to be beneficial in terms of maintaining normal physiological blood flow (original hemodynamics) in the ipsilateral VA as well as eliminating additional flow loading (hemodynamic stress) in the contralateral VA after ipsilateral deconstruction that might lead to subsequent dissection of the contralateral VA. Identification of the relationship of the anterior spinal artery, PICA, and medullary perforators with the dissected segment of the VA is of paramount importance for a safe and permanent cure after EVT. For good vessel wall apposition of stents, we usually deploy most of stents using dynamic push–pull technique, that is a combination of microcatheter pull-back and microwire push. According to the vessel tortuosity, we sometimes deploy open-cell stents using microcatheter pull-back technique.

Multiple stenting

We usually perform multiple stenting for symptomatic unruptured VADs, especially VADs with recurrent or progressive ischemia showing steno-occlusive lesions such as pearl-and-string signs. We deploy a single stent for VADs with steno-occlusive lesions involving a short segment. When a self-expanding closed-cell stent covers the dissected segment, it makes it easier to perform reconstitution of the vessel lumen using stabilizing intimal flaps and vessel wall repair by promoting neointima formation. If an ischemic presenting VAD involves very long segment extended to BA without large dissecting aneurysm, multiple stenting with a telescoping technique can be one effective treatment method (**Fig. 4**). We prefer to use a laser-cut closed-cell Enterprise stent for multiple stenting not only because it is easy to deploy but also because the Enterprise has good chronic outward force (= outward radial force) that is changes in diameter versus force for both expansion and contraction during circumferential compression of the stent throughout 360°. When oversizing a closed-cell design stent, the stent tends to exhibit a higher chronic outward force.[45]

LVIS Blue stent-within-an-Enterprise stent

As one way of multiple stenting without coiling among reconstruction methods, we perform double stenting by deploying an Low-profile Visualized Intraluminal Support (LVIS) Blue stent within an Enterprise stent for VADs in dominant or single VA.[46] A LVIS device (LVIS Blue; Microvention, Tustin, CA, USA) is a braided stent that provides a high degree of metal coverage about 22%–28%, suggesting that the LVIS Blue stent might be beneficial for complete obliteration of a dissecting aneurysm due to its support of a higher occlusion rate using coils inside of the aneurysm and its flow diversion effect. In addition, overlapping of LVIS blue stents might potentially work as a flow diverter because of their high metal coverage surface area. However, the transition zone located at both ends of the fusiform dissecting aneurysm neck and device transitions from a constrained diameter to its unconstrained state generally result in a low metal coverage surface area.[47–49] To overcome this limitation and demonstrate the flow diversion effect of the LVIS Blue stent, we can take advantage of the structural properties of a laser-cut closed-cell Enterprise stent and use an LVIS Blue stent-within-an-Enterprise stent technique. An Enterprise stent has a relatively minimal outward expansion at the unconstrained segment. In contrast, the LVIS Blue stent has an apparent outward expansion. Thus, the Enterprise stent offers a scaffold to reduce the size of the unconstrained segment across the fusiform dissecting aneurysm neck, thus maintaining even distribution of high metal coverage surface area and porosity without having a transition zone at either end of the aneurysm neck. Consequently, the flow-diverting effect of the LVIS Blue stent can be maintained by suppressing blood flow through the "opening" of cells at the transition zone into the aneurysm. An Enterprise stent has a metal coverage surface area of 8%–11%. After overlapping with an LVIS Blue stent, the metal coverage surface area may increase up to around 33% in a straight vessel such as the

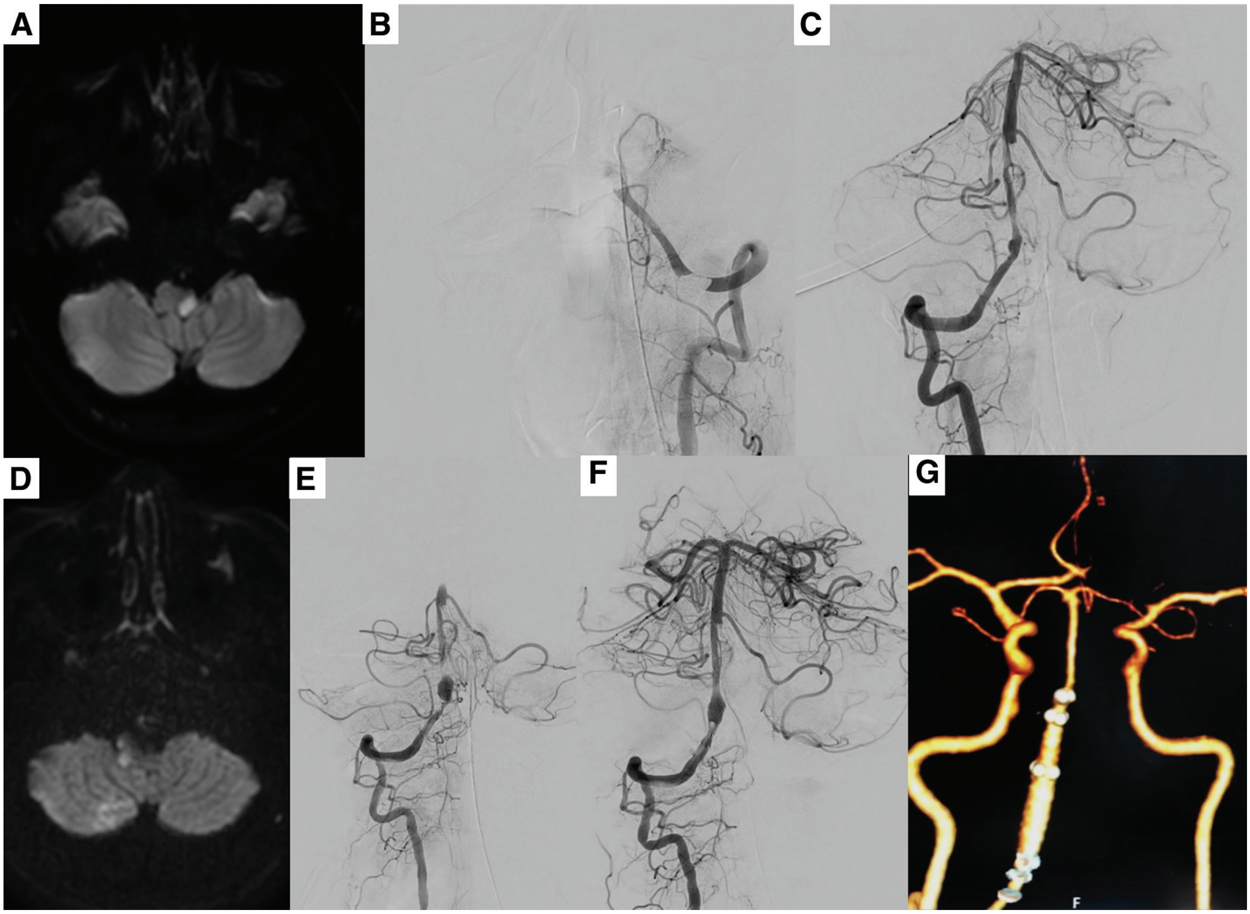

Fig. 4 Bilateral lateral medullary infarction due to bilateral VADs. (**A**) Patients were presented with left lateral medullary syndrome. (**B**) Left VAD with dissecting aneurysm was occluded spontaneously. (**C**) Right VA had small sized dissecting aneurysm that was asymptomatic. (**D**) At 3 days after admission, right lateral medullary infarction with PICA and PCA territory infarction occurred. (**E**) Right VAD aggravated the enlarging dissecting aneurysm and extending to BA. (**F**) Three self-expanding closed-cell stents that had been deployed by a telescoping technique, covered the dissected segment from BA-to-right VA. (**G**) Follow-up CTA showing complete healing of dissected segment with good patency. CTA: computed tomographic angiography; BA: basilar artery; PICA: posterior inferior cerebellar artery; VA: vertebral artery; VAD: vertebral artery dissection

VA. Using this technique, the flow-diverting effect can facilitate complete obliteration of VA dissecting aneurysms with endothelial healing of the VA (**Fig. 5**). Compared to deploying flow diverters, this technique offers a clinically reasonable alternative that is more cost-effective. In addition, this technique, as explained earlier, can use the LVIS Blue stent with an even distribution of metal coverage at the unconstrained segment. Furthermore, our technique for delivery and deployment may be easy because it involves small profiles and simple delivery systems. However, our technique requires sufficient time to achieve complete healing of the dissecting segment. Thus, it may not be proper for ruptured VADs because of the challenge of obliterating the rupture point immediately and the risk of hemorrhagic complications due to the use of dual-antiplatelet agents in the acute period.

Stent-assisted coiling

In cases of ruptured VAD with dissecting aneurysm in the dominant or single VA with poor collateral flow, reconstructive treatment without coils, such as multiple stenting or FDS, may result in re-rupture (rebleeding) of VAD during a latent period of dissecting flap stabilization and thrombose formation in the dissecting aneurysm. Because reconstructive techniques without coils are often difficult for ruptured VADs given the non-saccular shape of the dissecting aneurysm, we usually perform SAC in patients with ruptured VADs and aneurysmal dilatation in the dominant or single VA (**Fig. 6**).

When performing SAC for VADs in dominant or single VA, we should consider if the VA involves anterior spinal artery, PICA origin, or medullary perforators. Saving those critical branches is very important for safe reconstruction

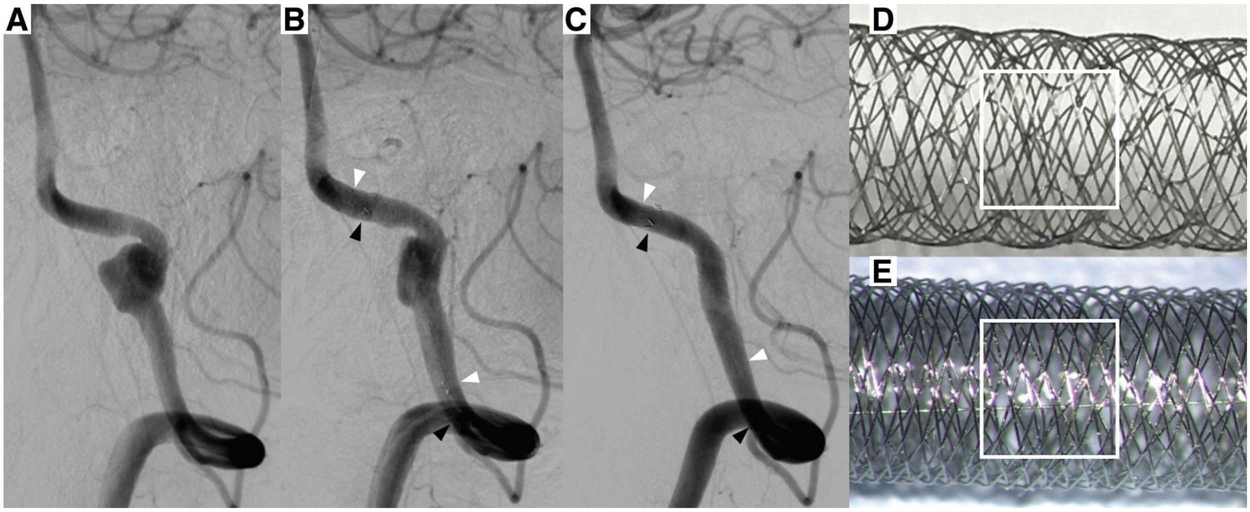

Fig. 5 (**A**) Right VA angiography showing the dissecting aneurysm involving 360 degree of the dominant VA. (**B**) Flow stagnation inside the aneurysm immediately after performing a LVIS blue stent-with-in-an-Enterprise stent technique. (**C**) Right VA angiography at 6-month follow-up revealing complete obliteration of the aneurysm and remodeling of the VA. The VA shows good patency without in-stent stenosis. The white arrowheads and black arrowheads indicate proximal and distal ends of the LVIS Blue and Enterprise, respectively. We used 4.5-mm-sized LVIS Blue stents and 4.0-mm-sized Enterprise stents to show metal coverage surface area of (**D**) An LVIS Blue-within-an-Enterprise stent (33.4%) and (**E**) double LVIS Blue stent (36.6%). LVIS: Low-profile Visualized Intraluminal Support; VA: vertebral artery

of the VA. In such cases, it may be difficult to perform SAC completely because a dissecting aneurysm tends to form fusiform-like dilatation rather than saccular-type aneurysm, resulting in incomplete obliteration by SAC. In such cases, we can make dense coil packing in the rupture point of the dissecting aneurysm if identified. For those reasons, SAC of VADs involving PICA tends to recur more frequently than other treatment options during follow-up. Cho et al.[7] have determined clinical and radiologic outcomes of VADs involving PICA according to different types of EVT. They found that multiple stenting and VA-PICA stenting groups had the highest rate of postoperative infarction, followed by SAC group. However, no infarction occurred in the FDS group during follow-up. Recurrence of dissecting aneurysms was seen in 25% of patients treated with SAC. When treating VADs involving the PICA origin with SAC, a small portion of the dissecting aneurysm from which the PICA originated was left without coil packing. That is, when the PICA origin was involved, the dissecting aneurysm was left partially open or entirely open to preserve blood flow to the PICA.

To save those branches from the VA, we can perform SAC with partial overlapping technique using two LVIS Blue stents (**Fig. 7**). In this technique, two LVIS Blue stents are overlapped around the dissecting segment while proximal and distal normal segments of the VA are covered by one LVIS Blue stent each. It may reduce perforator infarction around the normal segment of the VA. It may also enhance flow-diverting effect and increase complete healing of the dissecting segment by overlapping two stents.

Flow-diverting stenting

FDS can be used to treat VADs. Reconstructive procedures using FDS have become first-line treatment options worldwide, taking the place of multiple stenting. More recently, deconstruction procedures have been the last choice in the treatment of armamentarium. However, the use of FDS is limited in Korea due to our reimbursement system that allows one flow diverter per case without concomitant usage of coils. For stand-alone FDS in a ruptured VAD, it requires close angiographic follow-up considering the severity and increased risk of re-rupture. However, in those with symptomatic unruptured VADs, FDS may cover the origin of PICA without impairing flow through the branching artery when it is appropriately sized to the vessel wall and positioned in the VA (**Fig. 8**).

One advantage of the FDS is its potential to maintain patency of branching vessels and perforators arising from the parent artery.[50–54] The presence of interstices between stent strands facilitates flow to branching vessels while disrupting flow into a dissecting aneurysm and causing intra-saccular thrombosis. A recent meta-analysis has reported a perforator infarction rate of 3%, with a higher rate in patients with posterior circulation aneurysms than in those with anterior circulation aneurysms.[55] The wall reconstruction concept appears logical if the ruptured

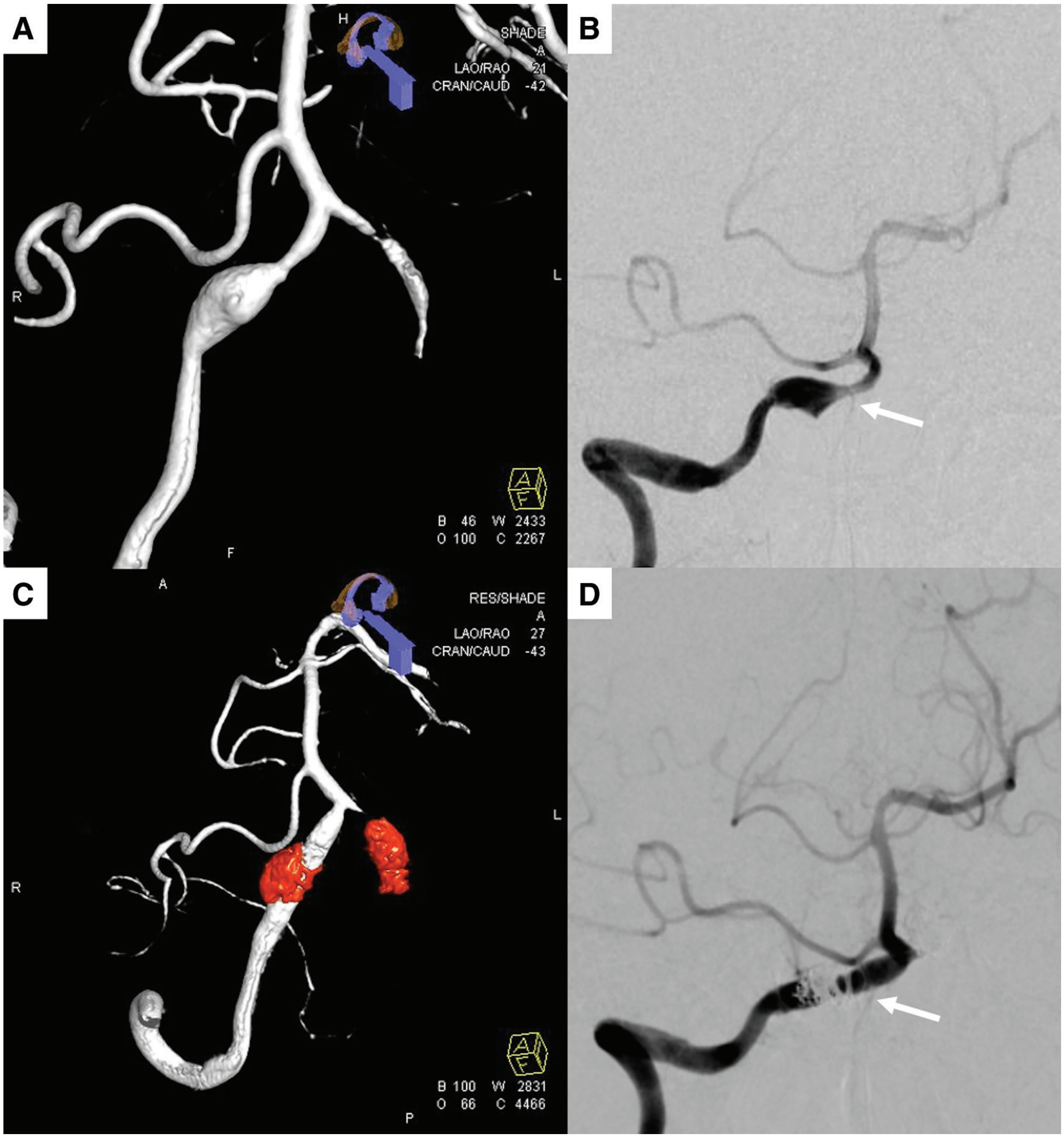

Fig. 6 (**A**) Ruptured bilateral VADs with dominant VA on the right. (**B**) Right dominant VA had fusiform aneurysmal dilatation with the rupture point on the inferior part. (**C**) Left non-dominant VA was occluded by internal coil trapping and SAC with triple stenting was performed for the right dominant VA. (**D**) Follow-up angiography at 12-month showing complete obliteration of the dissecting aneurysm and well reconstruction of right dominant VA. The white arrow indicates the anterior spinal artery is originated just distal to the dissecting aneurysm. SAC: stent-assisted coil embolization; VA: vertebral artery; VADs: vertebral artery dissections

VAD is considered to be a diffuse defect of the vessel wall. However, large multicenter studies regarding outcomes when employing reconstructive techniques are not currently available, with most outcome data coming from small published case series.[54,56–59] As the use of FDS for VADs increases, larger studies are needed to determine long-term outcomes such as rates of aneurysm occlusion, thromboembolic complications from device insertion, and sequelae of covered branch vessels and perforators.

Discussion

Endovascular techniques have evolved as the treatment of choice for intracranial VADs, either ruptured or symptomatic unruptured VADs. However, a consistent strategy regarding when to reconstruct and when to occlude an affected V4 segment has not been presented yet. Most published studies in this context favor one specific technique or a singular device without balancing shortcomings with advantages in an equilibrated fashion. Hence, we present how we have done in our

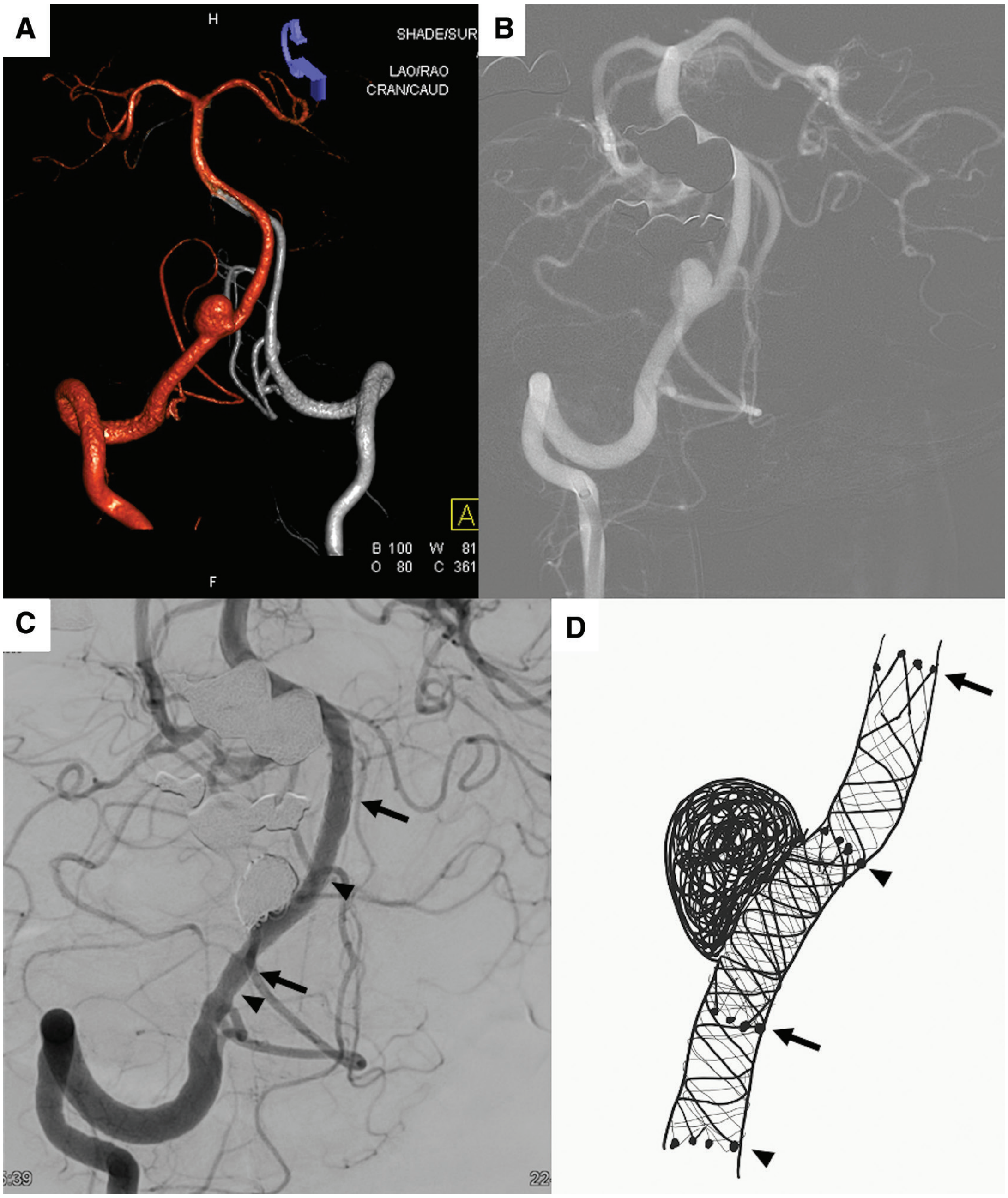

Fig. 7 Right unruptured VAD with a dissecting aneurysm with a diameter of about 8 mm on the dominant VA. (**A**) Three-dimensional reconstruction image and (**B**) two-dimensional working angle for SAC. (**C**) Complete obliteration of the dissecting aneurysm by SAC with partial overlapping technique of two LVIS Blue stents. (**D**) Illustration of SAC with partial overlapping technique of the two LVIS Blue stents. Dissecting segment of the VA was covered by double LVIS Blue stenting. Proximal and distal normal segments of the VA were covered by single LVIS blue stenting that might provide flow-diverting effect, enhance healing process of the VAD on the dissecting segment, and reduce perforator infarction around the normal segment of the VA. The black arrows and arrowheads indicate stent markers of distal and proximal LVIS Blue stents, respectively. LVIS: Low-profile Visualized Intraluminal Support; SAC: stent-assisted coil embolization; VA: vertebral artery; VAD: vertebral artery dissection

real practice for the last decade for treating VADs by EVT rather than reviewing and organizing so-far-published literature. We tended to occlude the rupture point by VA occlusion in non-dominant VA or SAC in dominant VA for ruptured VADs. We tended to reconstruct original hemodynamics using various stents for unruptured VADs.

We notice that the current trend is employing reconstructive techniques using FDS. However, reconstructive

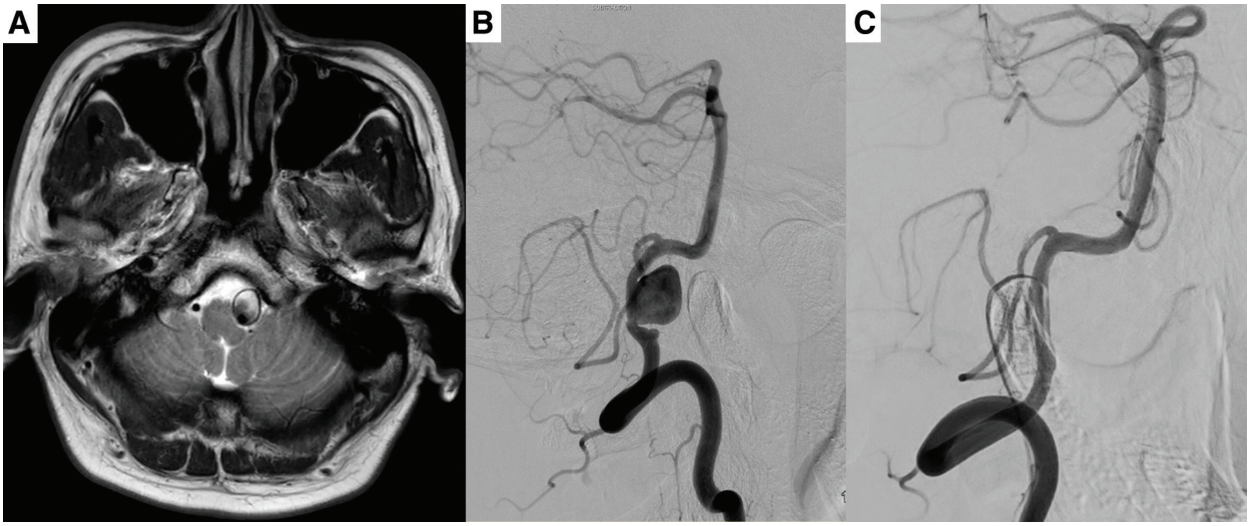

Fig. 8 (**A**) A symptomatic left VA aneurysmal dilatation is noticed on the MR T2-weighed image. (**B**) DSA of the left VA showing VAD with a dissecting aneurysm. (**C**) A 6-month follow-up angiography showing complete healing of the VAD after treatment with a flow-diverting stent, Pipeline Flex with Shield technology. Although the flow-diverting stent covered the origin of the PICA, the flow of the PICA was patent. DSA: digital subtraction angiography; MR: magnetic resonance; PICA: posterior inferior cerebellar artery; VA: vertebral artery; VAD: vertebral artery dissection

techniques using stents for ruptured VADs do have their own procedure-related complications with a recurrence issue. We have reported our experience in SAC of ruptured aneurysms in the acute period and evaluated the incidence and risk factors of procedure-related complications.[35] Patients with clinically poor grades or acute hydrocephalus underwent EVD before embolization to reduce the risk of catheter-related hemorrhagic complications due to antiplatelet therapy or anticoagulation. We found that the periprocedural complication rate was 19.4% among 72 patients with ruptured wide-necked aneurysms treated by SAC. The overall procedure-related thromboembolic complication rate was 12.5%; four (5.6%) were asymptomatic and five (6.9%) were symptomatic. This outweighed the 6.9% risk of hemorrhagic complications, all of which were symptomatic. At that time, we concluded that microsurgical clipping or EVT with another technique (multiple-microcatheter or balloon-assisted technique) might be a more appropriate option for first-line treatment than SAC, especially in patients with poor clinical grades or acute hydrocephalus who were likely to require EVD because of the high complication rate.[35] Additionally, we evaluated postoperative stroke risk and recurrence rate of ruptured and unruptured VADs involving PICA after different EVT modalities.[7] VA trapping with VA-PICA stenting showed the lowest rate of aneurysm recurrence with a high rate of minor infarction and favorable neurologic outcome. SAC showed high recurrence rates with possible fatal disabling infarction. Furthermore, there was a 13% recurrence rate after EVT of intracranial VADs. The rate of post-treatment recurrence did not differ between reconstructive and deconstructive techniques. Involvement of the PICA origin by VADs was the only independent risk factor for recurrence after EVT.[11]

In a meta-analysis of vertebrobasilar dissecting aneurysms in patients treated by either reconstructive or deconstructive techniques, the immediate occlusion rate was 75.0% (95% CI, 55.0%–88.0%) and the long-term occlusion rate was 87.0% (95% CI, 74.0%–94.0%).[60] Angiographic recurrence rate was 7.0% (95% CI, 5.0%–10.0%) with a retreatment rate of 3.0% (95% CI, 2.0%–6.0%). Perioperative morbidity rate was 12.0% (95% CI, 9.0%–16.0%). All-cause perioperative mortality was 8.0% (95% CI, 6.0%–11.0%). Patients with ruptured vertebrobasilar dissecting aneurysm made up the majority of patients with perioperative mortality (11.0%; 95% CI, 8.0%–16.0%). All-cause perioperative mortality for unruptured dissecting aneurysms was 4.0% (95% CI, 2.0%–9.0%). The overall rebleeding rate for patients with ruptured dissecting aneurysms was 9.0% (95% CI, 6.0%–13.0%). Patients treated with deconstructive techniques had higher rates of complete occlusion not only on immediate post-angiography than those treated with reconstructive techniques (88.0% vs 53.0%, P <0.0001) but also on long-term angiography (88.0% vs 81.0%, P <0.0001). Perioperative morbidity was lower in the reconstructive group than that in the deconstructive group (4.0% vs 12.0%, $P = 0.04$). There was a trend toward a decrease in perioperative mortality rates in the reconstructive group (4.0% vs 10.0%, $P = 0.11$) and a trend toward a higher rate of long-term good clinical outcome in the reconstructive group (92.0% vs 86.0%, $P = 0.10$). They concluded that EVT of vertebrobasilar dissecting aneurysms might be associated with a high rate of complete occlusion and good long-term neurologic outcomes. Deconstructive techniques may result in a higher rate of complete angiographic occlusion, while reconstructive techniques may be associated with less perioperative morbidity. However, long-term neurologic outcomes and retreatment rates are statistically similar between these two treatment modalities.[60]

While there is consensus that ruptured VADs should be treated reasonable and fair, proper indications to treat unruptured VADs have not been well established yet. However, we made our own indications for treating unruptured VADs. Our philosophy was melted in, according to our clinical and radiographic experiences about unruptured VADs. We treat symptomatic unruptured VADs presenting with recurrent or progressive ischemia because clinical outcomes for symptomatic unruptured VADs are favorable in all patients without ischemic symptoms and in most patients with ischemic presentation.[21] In addition, BA involvement is an independent predictors of unfavorable outcome in symptomatic unruptured VADs.[21] Furthermore, radiographic findings can provide helpful information about predicting outcomes.

Radiographically, there are three groups of VADs: dilatation-without-stenosis, pearl-and-string, and stenosis-without-dilatation.[1] Aneurysmal dilatation was more frequent than steno-occlusive type in the angiographic evaluation of symptomatic unruptured vertebrobasilar dissections. This finding differs from those associated with extracranial VAD, in which the steno-occlusive type was predominant.[61,62] One possible explanation for such difference in lesion type was the absence of external elastic lamina and decreased amount of medial elastic tissue in the intradural artery.[63,64] Subintimal dissection tends to result in luminal stenosis or occlusion, whereas subadventitial dissection often causes dilatation.[1] The primary lesion shape of symptomatic intracranial VADs might differ between unruptured and ruptured cases. Morphologies of symptomatic intracranial VADs are probably different at follow-up imaging compared to initial angiographic findings. Ruptured vertebrobasilar dissections are more likely to present with dilatation without stenosis (20 of 48 [42%]) or pearl-and-string appearance (24 of 48 [50%]) at initial angiography, whereas only 8% (four of 48) of ruptured cases show stenosis-without-dilatation.[1] Intramural hematoma was present in 33.9% (78 of 230) of cases, most frequently occurring in lesions that featured stenosis without dilatation (42 of 60 [70%]), followed by lesions with a pearl-and-string appearance (27 of 90 [30%]) and dilatation-without-stenosis appearance (nine of 80 [11%]) ($P < 0.05$).[1] The dilatation-without-stenosis group showed no change in 74% (25 of 34) of cases, whereas the stenosis-without-dilatation group showed improvement in 91% (39 of 43) of cases. Intracranial VADs without intramural hematoma at initial angiography showed improvement in 53% (32 of 60) cases. However, there was no change in 42% (25 of 60) of cases. Conversely, vertebrobasilar dissections with intramural hematoma exhibited improvement in 63% (34 of 54) of cases, progression in 20% (11 of 54) of cases, and no change in 17% (nine of 54) of cases. Intracranial vertebrobasilar dissections with intramural hematoma showed progression approximately four times more than vertebrobasilar dissection without intramural hematoma (20% vs 5%).[1]

The next step is to analyze our whole dataset of VADs treated by EVT. It is necessary to know not only our own clinical and radiographic outcomes after treatment but also procedure-related complications, morbidity, and mortality. These data may change our indications or EVT techniques in the future. In addition, large clinical trials are needed to determine long-term clinical and radiographic outcomes and determine each EVT technique's benefits and risks.

Conclusion

To decide what to treat and how to treat is very complicated for VADs. However, we believe that EVT is the current mainstay for treating VADs. Each technique of EVT should be determined on a case-by-case basis at the discretion of endovascular neurosurgeons and/or interventional neuroradiologists according to presenting symptoms, hemodynamic status, including sufficiency of the collateral supply, and anatomic features of the vertebrobasilar artery as well as the PICA, anterior spinal artery, and medullary perforators.

Disclosure Statement

The authors declare that they have no conflicts of interest.

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