Treatment of Dissecting Distal Vertebral Artery (V4) Aneurysms With Flow Diversers

BACKGROUND: Dissecting aneurysms of the intracranial vertebral arteries are rare; however, treatment of these presents multiple challenges, including high risk of rebleeding, development of thromboembolic strokes, and progressive partial thrombosis. Flow diveters, such as Pipeline Endovascular Devices (PEDs; Covidien, Medtronic Inc, Dublin, Ireland), have emerged as a potential treatment option.

OBJECTIVE: To present our experience with patients treated at our institution with PEDs for dissecting distal vertebral artery (V4 segment) aneurysms.

METHODS: A retrospective search of our prospectively maintained database was performed between January 2014 and December 2016. We queried our database for all patients treated with PED for dissecting aneurysms of the V4 segment. Information was gathered including demographics, the location and morphology of the aneurysm, the clinical presentation, specific form of treatment, complications, antiplatelet medication regimen, and follow-up time.

RESULTS: There were a total of 9 patients with dissecting V4 aneurysms treated with PED during the study period. All were treated initially with an average of 1.2 PEDs. All patients were followed with at least one repeat diagnostic angiogram and there was no residual aneurysm seen in 8 of 9 cases. In those that presented with neurological deficits, there was an average improvement in modified Rankin Scale of 2.85 points.

CONCLUSION: PED is a safe and effective tool that can be used to treat ruptured dissecting aneurysms of this specific segment of the posterior circulation, but it does require close management of antiplatelet therapy in the setting of subarachnoid hemorrhage and close angiographic follow-up.

KEY WORDS: Aneurysm, Dissecting, Embolization, Therapeutic, Intracranial embolism, Stent, Vertebral artery

Dissecting aneurysms of the intracranial vertebral arteries are rare, and either present with ischemic symptoms caused by posterior fossa strokes or as a subarachnoid hemorrhage (SAH). In total, they account for about 3% to 7% of SAHs. These present multiple challenges, including high risk of rebleeding, development of thromboembolic strokes, and progressive partial thrombosis that can generate mass effect on the adjacent brainstem. The clinical manifestations can be diverse, and presentation may vary from acute onset headache, hydrocephalus, altered consciousness in the case of ruptured aneurysms, or cranial neuropathies or Horner’s sign caused by mass effect of the lesion.

The distal vertebral artery segment (V4) is especially troublesome considering the presence of side arterial branches such as the posterior inferior cerebellar artery (PICA), the anterior spinal artery, and the posterior spinal arteries. The V4 segment represents the transition between the intracranial and extracranial segments that can be affected with extreme motion at the craniovertebral junction and also vulnerable to direct trauma against the foramen magnum.
Traditional neurosurgical approaches include vessel wrapping or internal deconstruction with and without revascularization. More recently, endovascular options have included coil embolization of larger saccular components and the use of intracranial stents to support the coils. As endovascular technology has evolved, the use of standalone stents (flow diverters) or in addition to coils are able to provide complete vessel remodeling and definitive cure of these complex lesions.

We aim to present our retrospective experience with a series of consecutive patients treated at our institution with Pipeline Embolization Device or PED (Covidien, Medtronic, Dublin, Ireland) for dissecting V4 segment aneurysms during the time period of 31 months. Clinical presentation, pertinent imaging, vascular anatomy, treatment strategies, complications, and outcomes are evaluated as well as highlights of key points on a few educational cases.

METHODS

Data Collection
A retrospective search of our prospectively maintained database was performed between January 2014 and December 2016. We queried our database for all patients treated with PED (Medtronic Inc) for dissecting aneurysms of the V4 segment. This was defined as fusiform aneurysms where the lesion compromises a 360° segmental arterial defect, lesions with a false lumen and/or very small “blister” like lesions. This protocol was approved by the Duke Institutional Review Board. Electronic records and images were accessed and information was gathered, including demographics, the location and morphology of the aneurysm, the clinical presentation, specific form of treatment, complications, antiplatelet medication regimen, and follow-up time.

Procedure
All patients had a cerebral digital subtraction angiography (DSA) to confirm the diagnosis found on magnetic resonance angiogram (MRA) or computed tomography angiogram (CTA). The treatment was performed once images were reviewed and it was considered not amenable for surgical treatment. Written and informed consent was obtained from all patients or the patient's power of attorney. The off-label character of flow diversion for this location was emphasized. Preoperatively, patients were loaded on antiplatelet medications and were maintained on this postoperatively. Aspirin (ASA) and clopidogrel were the initial treatment choices. P2Y12 (VerifyNow, Accriva, California) levels were measured to evaluate the responsiveness to clopidogrel and the need to either increase dose or convert to other antiplatelet agents. Initially, either 1 or 2 stents were deployed depending on the length of coverage needed, morphology of the aneurysm, and if any residual filling was seen. All patients had an angiographic and clinical follow-up. In some cases, if residual aneurysm was seen at follow-up angiogram, an additional stent was deployed.

RESULTS
There were a total of 9 patients (Table 1) with dissecting V4 aneurysms treated with PED (Medtronic Inc) during the study period. During this period, no patient was treated surgically. There were 6 men and 3 women, and the mean age at presentation was 57.3 yr. Five of 9 patients presented with SAH. The average largest diameter of the nonblister aneurysms was 10.6 mm. All were treated initially with an average of 1.3 PEDs and in 2 patients an additional PED was deployed to treat residual aneurysm at follow-up. One patient had an 8-mo follow-up that demonstrated complete occlusion of the ipsilateral vertebral artery (patient 6). In those cases where coadjuvant coils were used (n = 4), a second microcatheter was placed in the aneurysm and “jailed” before the PED was deployed. The aneurysm was then coiled after the PED was deployed. All patients were followed with at least one repeat diagnostic angiogram and there was no residual aneurysm seen in any of the cases except for one (patient 9) at the most recent angiographic follow-up. The average follow-up time was 15.1 mo (range 4-29 mo). One patient was lost to follow-up after 4 mo and another patient died at 4 mo due to unrelated causes.

Outcome and Complications
In those that presented with neurological deficits, there was an average improvement in modified Rankin Scale (mRS) of 2.85 points, and all patients at follow-up had an mRS of either 0, 1, or 2. There was only one complication related to the procedure itself (development of a lower extremity pseudoaneurysm in patient 3) and 2 long-term complications occurred related to the antiplatelet therapy (intracranial hemorrhage following ventriculoperitoneal shunt placement in patient 6 and delayed minimally symptomatic perforator stroke seen in patient 5).

Table 1 shows the individual patients who presented with V4 segment dissecting aneurysms and information regarding their presentation and treatment.

Illustrative Case #1
Patient 2 is a 58-yr-old male who presented to the Emergency Department (ED) with worst headache of life and was found to have a Fisher 2 Hunt Hess 1 SAH. He underwent a diagnostic angiogram that revealed a right vertebral artery “blister” proximal to the vertebrobasilar junction. Incidentally, it was found to have a left subclavian artery occlusion secondary to previous chest radiation (Figure 1). He was loaded on 600 mg clopidogrel and ASA on postbleed day 1 and taken to the angio suite the next day for PED stent embolization. This was complicated by a brief period of hypotension requiring cardiopulmonary resuscitation prior to intubation that was not related to the procedure. He recovered and the procedure was resumed later that day. He did well postoperatively. Given that he was a clopidogrel nonresponder (no drop in the P2Y12 values despite being loaded on clopidogrel), he was switched to prasugrel for maintenance antiplatelet therapy along with the ASA. At 6-mo follow-up, a diagnostic angiogram demonstrated no residual aneurysm, and he was asymptomatic and at full functional status.
## TABLE 1.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>SAH Presentation</th>
<th>Size of nonblester aneurysm</th>
<th>Number of PED devices</th>
<th>Coils placed</th>
<th>Complications</th>
<th>Follow-up time</th>
<th>Outcome on most recent angiogram</th>
<th>Initial mRS</th>
<th>Post procedure mRS</th>
<th>Change in mRS</th>
<th>Antiplatelet medications</th>
<th>Days after bleed that antiplatelet therapy was started (if applicable)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>67</td>
<td>Incidental</td>
<td>17 mm</td>
<td>1</td>
<td>Y</td>
<td>None</td>
<td>4 mo</td>
<td>No residual aneurysm, patent stent</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Clopidogrel (prior to PED due to recent 4 drug eluting stents). ASA initially, then changed to apixaban after 3 mo for new diagnosis of atrial fibrillation</td>
<td>N/A</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>58</td>
<td>F2H1 SAH</td>
<td>N/A</td>
<td>1</td>
<td>N</td>
<td>Prior to intubation, patient became hypotensive and required CPR briefly. Procedure was aborted and successfully preformed later same day</td>
<td>25 mo</td>
<td>No residual aneurysm, patent stent</td>
<td>1</td>
<td>0</td>
<td>−1</td>
<td>ASA indefinitely. Initially on clopidogrel but was nonresponder, so switched to prasugrel for 6 mo</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>66</td>
<td>Incidental, WHOL 2 wk prior</td>
<td>5 mm, pseudoaneurysm with near complete occlusion of the vertebral artery</td>
<td>1 placed initially, a second placed at 2 mo follow-up for residual aneurysm</td>
<td>N</td>
<td>Hematoma and pseudoaneurysm of right femoral artery and development of right femoral DVT. Required vascular surgery repair in OR and 3 mo anticoagulation</td>
<td>25 mo</td>
<td>No residual aneurysm, patent stent</td>
<td>1</td>
<td>0</td>
<td>−1</td>
<td>ASA indefinitely. Clopidogrel for 2 mo but changed to rivaroxaban for 3 mo for DVT</td>
<td>N/A</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>73</td>
<td>F3H3 SAH</td>
<td>N/A</td>
<td>1</td>
<td>N</td>
<td>None</td>
<td>17 mo</td>
<td>No residual aneurysm, Patent stent</td>
<td>5</td>
<td>1</td>
<td>−4</td>
<td>Clopidogrel indefinitely. Clopidogel for 6 mo</td>
<td>1</td>
</tr>
<tr>
<td>Patient</td>
<td>Sex</td>
<td>Age (yr)</td>
<td>SAH Presentation</td>
<td>Size of nonblister aneurysm</td>
<td>Number of PED devices</td>
<td>Coils placed</td>
<td>Complications</td>
<td>Follow-up time</td>
<td>Outcome on most recent angiogram</td>
<td>Initial mRS</td>
<td>Post procedure mRS</td>
<td>Change in mRS</td>
<td>Antiplatelet medications</td>
<td>Days after bleed that antiplatelet therapy was started (if applicable)</td>
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<tr>
<td>5</td>
<td>M</td>
<td>59</td>
<td>F3H3 SAH</td>
<td>5 mm</td>
<td>2 placed initially, then a third placed at 16-d follow-up for aneurysm enlargement</td>
<td>N</td>
<td>Small perforator stroke 3 mo later, asymptomatic</td>
<td>13 mo</td>
<td>No residual aneurysm, patent stent</td>
<td>5</td>
<td>0</td>
<td>−5</td>
<td>ASA and clopidogrel indefinitely</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>50</td>
<td>F4H5 SAH</td>
<td>10 mm</td>
<td>1</td>
<td>Y</td>
<td>No procedure complications. Developed a frontal ICH during VPS placement</td>
<td>29 mo</td>
<td>Complete occlusion of ipsilateral vertebral artery</td>
<td>5</td>
<td>0</td>
<td>−5</td>
<td>ASA indefinitely. Initially started on clopidogrel but was non-responder so switched to ticagrelor for 8 mo</td>
<td>21 d</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>42</td>
<td>H3F4 SAH</td>
<td>10 mm</td>
<td>1</td>
<td>Y</td>
<td>None</td>
<td>4 mo</td>
<td>No residual aneurysm, patent stent</td>
<td>4</td>
<td>0</td>
<td>−4</td>
<td>ASA indefinitely and clopidogrel, for 4 mo</td>
<td>10</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>55</td>
<td>Incidental</td>
<td>15 mm</td>
<td>2</td>
<td>Y</td>
<td>None</td>
<td>11 mo</td>
<td>No residual, patent stent</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Clopidogrel and ASA indefinitely (was also on coumadin for mechanical heart valve)</td>
<td>N/A</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>46</td>
<td>Posterior circulation infarction</td>
<td>12 mm</td>
<td>2</td>
<td>N</td>
<td>None</td>
<td>8 mo</td>
<td>Minimal residual aneurysm opacification, patent stent</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>Clopidogrel for 4 mo, ASA indefinitely</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Abbreviations: ASA = Aspirin; N/A = not applicable; F = Fisher Score; H = Hunt Hess Score; SAH = subarachnoid hemorrhage; WHOL = worst headache of life; DVT = deep venous thrombosis; ICH = intracranial hemorrhage; VPS = ventriculoperitoneal shunt; mRS = modified Rankin Scale.
Illustrative Case #2

Patient 3 is a 66-yr-old male who experienced chronic migraines headaches and presented to the ED with an acute headache and neck pain. Work-up was negative for acute SAH but an MRA with dissection protocol demonstrated a mural thrombus of a dissecting right V4 aneurysm. On initial diagnostic angiogram (Figure 2), the presence of a right V4 dissecting aneurysm was confirmed, and found to be pointing anteriorly and superiorly at the takeoff of the PICA with a narrow distal true lumen and flow limiting stenosis. He underwent a placement of PED and was discharged without complications on ASA and Plavix. At 3-mo follow-up, there was residual aneurysm and a second telescoping stent was placed uneventfully. His postoperative course was complicated by the development of a hematoma and pseudoaneurysm of the right common femoral artery that required surgical repair, and also the development of a right femoral deep vein thrombosis that required 3 mo of anticoagulation. At last follow-up at 8 mo, he was asymptomatic with no neurological sequelae and no evidence of the aneurysm.

Illustrative Case #3

Patient 5 is a 59-yr-old male who presented to the ED with worst headache of life and was found to have a Fisher 3 Hunt Hess 3 SAH. He was intubated and an external ventricular catheter (EVD) was placed shortly after admission. Angiography (Figure 3) demonstrated a fusiform dissecting aneurysm involving the right vertebral artery V4 segment. The patient was loaded with clopidogrel and ASA prior to treatment later that day. Two PED devices were placed in a telescoping fashion. The EVD was eventually weaned and removed. On 2-wk follow-up angiogram, he was found to have some enlargement of the residual aneurysm so a third PED Flex was deployed. Later, he was discharged with no complications on ASA and clopidogrel. Overall, from time of presentation to time of follow-up, he had an improvement in mRS of 5 points, and was at his baseline when last seen. Three months later, he experienced a small perforator medullary stroke and was admitted to an outside hospital for a brief period. During this admission, he had an angiogram that demonstrated complete aneurysm occlusion. His symptoms resolved and he was asymptomatic when discharged home 2 d later.
DISCUSSION

The vertebral artery is particularly vulnerable to the development of dissecting aneurysms compared to the anterior vasculature. Potential reasons are that the intradural segment of the vertebral artery is thin and the adventitia has few elastic fibers. Thus, the intradural segment is more likely to rupture and cause SAH with possible pseudoaneurysm formation than the extradural segments. There is also less vasa vasorum that might contribute to decreased potential for healing and strengthening of the vessel wall. The peak age for dissecting aneurysms is typically in the fourth decade with a slight male predominance.

Small unruptured dissecting aneurysms typically have a benign course, and conservative measures are often advocated, such as anticoagulation or antiplatelet therapy. However, once ruptured, vertebrobasilar aneurysms carry a grim prognosis. The mortality rate is about 50%, and recurrent hemorrhage occurs in 24% to 70% of cases.

Ruptured dissecting vertebrobasilar aneurysms are treacherous due to high risk of rebleeding and poor prognosis if rebleeding occurs. The strategy of therapy depends heavily on the relationship of the aneurysm with branching arteries, especially the PICA, the presence of a more saccular component within the dissected segment, and the presence of a contralateral vertebral artery.

Historically, treatment options include Hunterian ligation, wrapping, and reconstructions with surgical bypass. Until recently, endovascular treatment was limited to parent vessel sacrifice by trapping the affected segment with coils. However, further advancements of endovascular technology have broadened the therapeutic arsenal and emerged as the favored treatment modality. The 2 most common techniques are vessel deconstruction and stent coiling when a saccular component is present. Patients treated with trapping techniques have a higher rate of complete occlusion compared to those that were stent coiled, but recurrence rates are similar between the 2 groups.

Advocates for proximal artery occlusion argue that there is a decreased risk of complication by avoiding the passage of a catheter through a narrow or irregular segment. However, obliteration of the parent vessel can cause lateral medullary or cerebellar
infarction, which can occur even on the nondominant side. In cases where PICA origin was not involved with codominance with the contralateral artery, the main treatment option has been vessel deconstruction via microsurgical clipping or coil occlusion. In cases where PICA was involved within the dissection, the surgical option was limited to occipital to PICA bypass or an in situ PICA–PICA anastomosis with deconstruction or trapping of affected vertebral artery, which carries the risk of affecting the origin of the segmental perforators such as the anterior and posterior spinal arteries.

Flow diverters have emerged as an attractive treatment option for these challenging lesions. These devices are placed in the parent artery and affect the hemodynamics such that there is an eventual remodeling of the affected segment via endothelial proliferation. Thus, treating aneurysms without occluding functional perforators.

The PED consists of a tightly braided chromium cobalt nickel alloy and has low porosity. The metal surface area acts as a scaffold for endothelialization and intraluminal reconstruction that occurs in a delayed fashion. Originally, PEDs have been used to treat wide necked aneurysms of the anterior circulation. However, as interventionalists have become more experienced and comfortable with flow diverters, they have shown benefits for posterior circulation dissecting aneurysms and met with favorable outcomes. The use of PED for posterior circulation aneurysms in the United States is considered off-label, while its use is broader in Europe, Asia, and South America.

Our series shows that PEDs with or without coils can be a first line treatment option for dissecting V4 segment aneurysms. In our experience, adding coils facilitates and expedites aneurysm occlusion and decreases risk of rebleeding while the PED provides a definitive cure by arterial remodeling that prevents recurrence (Cases 1, 6-8). We used coils as adjuvants only when larger (>10 mm) saccular components were present. All patients had complete resolution of the aneurysm on the latest angiographic follow-up except for 1 case (Case 9). In cases where the saccular component does not allow coil placement (blister aneurysms, patient 2 and 4), the use of flow diversion as standalone treatment provided a definitive cure while minimizing the risk of procedural rupture since the aneurysm itself was not manipulated (Cases 2-5).

The use of antiplatelet medications in the setting of SAH is controversial and has been reported before for both more porous stents and flow diverters. However, new data suggest that these medications are safe in the acute setting after SAH and may even have independent benefits of reducing delayed cerebral ischemia. Our strategy included placement of an external ventricular drain prior to the procedure, and also loading the antiplatelet agent prior to the treatment procedure. During this time, platelet functionality with P2Y12 (VerifyNow) tests were monitored pre- and postloading dose, with a goal of a decrease in the P2Y12 Reaction Units (PRU) to 40% to 60% from a baseline PRU levels; others use a wide range P2Y12 level (60-240) with no baseline required. We prefer the former due to large variability in response. Albuquerque et al7 and Mazur et al14 described the use of the intravenous agent abciximab, as opposed to our strategy where none of our patients received an intravenous antiplatelet (abciximab or epifibatide) loading dose due to the inability to follow platelet inhibition function (VerifyNow) for a few weeks, therefore preventing us from identifying clopidogrel nonresponders. Using this strategy, we identified 2 clopidogrel nonresponder patients.

Patients had remarkable recovery in our series as quantified by the mRS designations at presentation and at follow-up time. Overall, there were no series complications and no patient developed any new or worsening neurological deficits. There are others who have reported similar findings. Albuquerque et al17 in a series of 17 patients with posterior fossa aneurysms, including only one with a ruptured aneurysm, used an average of 2.1 devices per patient. Our strategy differed in that we utilized only one device initially, and an additional one if there was no adequate coverage immediately postprocedure (Cases 5, 8, and 9) or evidence of aneurysm growth on short follow-up DSA (Case 5). All were treated with dual antiplatelet medications for at least 6 mo.7 Mazur et al14 reported a mixed series including patients with aneurysms that occurred in V4 segment, saccular aneurysms including the PICA origin, and dissections, and only 2 presented with SAH. Our series refers exclusively to dissecting V4 aneurysms and most of them presented with SAH.

Similarly, Phillips et al10 presented data from a multi-institutional prospective case registry of 32 patients with posterior circulation aneurysms treated with PED, although there was no clear description of the location and presence of SAH, except in one case. There were no deaths or poor neurological outcomes. They had no aneurysm rerupture or PED thrombosis events. There were permanent neurological complications in 2 patients (9.4%) but they were considered mild and still had good clinical outcome.10

Use of PED on the vertebral and basilar artery system is not without risks. In-stent thrombosis and thromboembolic events are feared complications, and up to 52% of patients can have diffusion weighted imaging (DWI) changes on magnetic resonance imaging and up to 14% experience symptomatic infarctions. Occlusion of perforators can occur acutely by direct mechanical blockage from a tine or strut of the PED, a thrombus from the device migrating to the branching artery, or chronically by excessive neointimal proliferation (ie, intrastent stenosis). Some are conducive to a progressive asymptomatic occlusion of the vertebral artery as seen in patient 6, or a subacute perforator stroke like in patient 5. Fiorella and colleagues15 report a case where a patient developed late construct thrombosis a year after PED was placed for fusiform aneurysm of the left vertebral artery. The patient developed stroke like symptoms 1 yr later that eventually progressed to basilar thrombosis and death.13 These complications can be due to lack of response to antiplatelet therapy, malposition of the stent, or arterial injury at time of stent placement. Other complications include perianeurysmal edema and distant and/or delayed hemorrhages.8
The use of standalone flow diverters does not provide immediate aneurysm occlusion therefore carries the potential risk of early rebleeding. Close angiography follow-up is highly recommended to rule out aneurysm enlargement (patient 5). To mitigate this, we prefer adding coils when a large enough saccular component is present or telescoping additional devices, although Tan and colleagues⁹ suggest avoidance of overlapping PEDs in the basilar artery and placement of unnecessary stents to decrease the risk of in-stent thrombosis. For nonruptured dissecting aneurysms, we perform catheter angiography at 3 mo, and if the aneurysm disappeared, we switch them to ASA monotherapy for life.

In summary, from our experience, avoidance of these complications can be achieved by several treatment strategies. This relies on 4 pillars; namely, early placement of EVD prior to the use of antiplatelet medications, close monitoring of the platelet function (VerifyNow) with medication and dose adjustments based on response, platelet transfusion with restarting of antiplatelet medications after required procedures (such as EVD replacement, ventriculoperitoneal shunt, etc.), and early angiographic follow-up. Even in the presented series, platelet function was probably not completely restored for patient 6, resulting in a frontal intracranial hemorrhage after placement of ventriculoperitoneal shunt, despite being transfused platelets.

Limitations

Our study is limited by small sample size and data from a single institution. It is retrospective and consecutive thus vulnerable to confounding factors and inherent bias. Outcomes are affected by inevitable learning curves.⁶ Additionally, our follow-up is relatively short and long-term risk is not well defined.

Despite these items, we believe our case series provides more evidence that PED is a safe and effective treatment for V₄ dissecting aneurysms, even in those that present as ruptured with a subarachnoid hemorrhage.

CONCLUSION

V₄ segment aneurysms are difficult lesions, and currently the literature is lacking in long cases series or prospective trials to suggest a consensus treatment paradigm. Flow diverters provide definitive aneurysm occlusion and vessel preservation, which is vital due to the abundance of perforators in this area. PED (Medtronic Inc) is a safe and effective tool that can be used to treat ruptured dissecting aneurysms of this specific segment of the posterior circulation, but it does require close management of antiplatelet therapy in the setting of SAH and close angiographic follow-up.

Disclosures

Dr Gonzalez occasionally proctors on-label cases. The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

REFERENCES


COMMENT

In this study, the authors have reported a small series of 9 patients with vertebral artery (V₄ segment) acute dissection without hemorrhage, dissections with subarachnoid hemorrhage, or dissecting aneurysms without hemorrhage through a retrospective review. All 9 cases underwent Pipeline Embolization Device (PED) placement to restore the lumen of the artery and treat the presenting aneurysm or pseudoaneurysm, requiring co-adjunct coiling in 4. The authors address one of the challenging scenarios that cerebrovascular neurosurgeons and neuroendovascular interventionists come to encounter. This stems from the fact the V₄ segment involves the origin of posterior inferior cerebellar artery (PICA), anterior spinal artery, posterior spinal artery, and caudal brain stem perforators. The case series demonstrates the robust utility of PED in that its placement in the dissected V₄ segment can eliminate the aneurysmal or pseudoaneurysmal sac portion to prevent rehemorrhage,
and ultimately reconstruct the vascular lumen in this complex anatomic location.

One of the advantages of this work is the focus on the V4 segment versus the entire vertebrobasilar system. The authors also address the need for a protocol to assess patient response to anti-platelet therapy. To this end, they track all patients with a Plavix effect assay, commonly referred to as the PRU test (Plavix reaction units) or by the parent company name, VerifyNow. Use of the PED for treatment in this study was designed to minimize the number of stents, with more than 1 device being used at initial treatment in 2 patients for complete coverage of the lesion. Three patients required re-treatment after their aneurysms were either incompletely resolved or demonstrated growth on follow-up. Of note, one of the patients with incomplete aneurysm obliteration (patient #9) was the only patient with an aneurysm of >10 mm who did not undergo coadjuvant coiling.

However, this study raises concerns given previous literature regarding the risk of multi-device PED utilization. In this series, one of the patients who had additional implantation of a PED after 2 devices were initially placed developed small infarct of brain stem (patient #5, 3 PEDs total), likely due to perforator occlusion. While we share the authors’ strategy of PED vertebral artery lumen preservation technique at our institution, an additional strategy is to utilize overlapping intracranial porous stents to eliminate the V4 segment dissection with blister aneurysms. We believe the latter may provide better radial force to restore the lumen and reduce the long-term risk of perforator occlusion, particularly after stopping Plavix after 6 months.

Treatment of posterior circulation disease requires a high degree of skill, and continues to represent a challenge for both open and endovascular approaches. This study refines the work of others to extend the use of PEDs to posterior lesions. Given their relatively lower incidence, a multi-center collaborative data registry would help further clarify some management strategies and risks.

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