Endovascular management of intracranial vertebral artery dissecting aneurysms

FELIPE C. ALBUQUERQUE, M.D., DAVID J. FIORELLA, M.D., PH.D., PATRICK P. HAN, M.D., VIVEK R. DESHMUKH, M.D., LOUIS J. KIM, M.D., AND CAMERON G. MCDougall, M.D.

Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph’s Hospital and Medical Center, Phoenix, Arizona

Object. Intracranial vertebral artery (VA) dissecting aneurysms often present with severe subarachnoid hemorrhage (SAH) and dramatic neurological injury. The authors reviewed the management of 23 cases in an effort to evaluate treatment efficacy and outcomes.

Methods. The records of 23 patients who underwent endovascular treatment were reviewed to determine symptoms, type of therapy, complications, and clinical outcomes. All patients were evaluated using records kept in a prospectively maintained database.

Ten men and 13 women (age range 35–72 years; mean age 49 years) were treated over an 8-year period. Twelve patients presented with poor-grade SAH, five with good-grade SAH, three with headache, and two with stroke. The other patient’s aneurysm was discovered incidentally. Treatment included coil occlusion of the artery at the aneurysm in 21 patients and stent-assisted coil placement in two. Parent artery sacrifice was successful in all cases, whereas both patients treated with stent-assisted coil insertion suffered recurrences. No patient sustained permanent complications as a result of treatment. Two patients died due to the severity of their original SAH. Findings were normal in 14 patients on follow-up review (including five of the 12 presenting with poor-grade SAH), five had fixed neurological deficits but were able to care for themselves, and one was permanently disabled.

Conclusions. Despite their often aggressive neurological presentation, intracranial VA dissecting aneurysms can be managed safely with coil occlusion of the lesion and/or parent artery. Even patients presenting in poor neurological condition may improve dramatically.

KEY WORDS • dissecting aneurysm • vertebral artery aneurysm • endovascular treatment

Dissecting aneurysms of the intracranial VA may manifest with severe SAH and devastating neurological sequelae. Similarly, unruptured lesions can enlarge and produce symptoms due to both compressive and embolic mechanisms. These lesions are also associated with a propensity for rerupture in the acute setting and consequently, urgent treatment is usually pursued. Endovascular procedures, including coil occlusion and intracranial stent placement, may obviate the need for complex skull base approaches. Nonetheless, the anatomy of the V4 segment, specifically the location of the PICA and the spinal artery in relation to the aneurysm, determines the optimal treatment. We reviewed the efficacy of our interventional management in a consecutive series of 23 patients who were followed using a prospectively maintained database. We specifically analyzed the type and success of therapy in a subset of patients who presented in moribund condition with severe SAH.

CLINICAL MATERIAL AND METHODS

Patient Population

The records of 23 patients with dissecting aneurysms of the fourth segment of the VA, as documented by cerebral angiography, were reviewed. The factors assessed included clinical history, presentation, presence of SAH and its severity, aneurysm location, type of endovascular therapy, radiographically documented success of therapy, and clinical outcome. All patients were evaluated using records kept in a prospectively maintained database that was designed to track outcomes and treatment-related complications.

As shown in Table 1, the patients included 10 men and 13 women treated over an 8-year period by two endovascular neurosurgeons (F.C.A. and C.G.M.). The patients’ ages ranged from 35 to 72 years (mean age 49 years). Twelve patients presented with poor-grade SAH (Hunt and Hess Grade IV or V), whereas five presented with good-grade SAH (Hunt and Hess Grade I–III). Of the six remaining patients, three presented with headache and two with embolic strokes of the posterior circulation.
The other patient's aneurysm was incidentally detected on computerized tomography scans obtained after blunt head trauma.

**Endovascular Procedures**

Standard endovascular methods of parent artery coil occlusion, aneurysm coil occlusion, stent placement, and transfemoral coil delivery were used and are well described in the current literature.\(^5\) The type of endovascular therapy used varied with the location of the aneurysm in relation to major arterial branches and with the relative dominance of the affected VA. In all cases, careful angiographic analysis of the contralateral VA both preceded and followed endovascular treatment of the ipsilateral artery.

**RESULTS**

Although endovascular therapy was typically instituted within hours of the patient’s arrival at our institution, four individuals suffered rerupture of their lesions before treatment. Fourteen aneurysms were located on the right VA; of these, eight were proximal to the PICA, four were distal, and one was located at the origin of the PICA. In the other patient with a right-sided aneurysm, no ipsilateral PICA was visualized. In the nine patients with left-sided aneurysms, seven lesions were distal to the PICA and two were proximal.

**Endovascular Treatment**

Occlusion of the parent artery was performed using transarterial detachable coils deposited at the aneurysm site in 21 of 23 patients. In the remaining two, stent-assisted coil occlusion of the aneurysm was undertaken because the PICA originated from the segment containing the lesion in one patient and the affected VA was dominant in the other. According to radiographic studies, treatment was successful in all cases of parent artery occlusion. Both patients treated with stent-supported coil occlusion, however, had recurrences. Of these, one individual presented with recurrent headaches and the other was asymptomatic. The former, in whom the affected artery was dominant, was treated with parent artery coil occlusion after tolerating a balloon test occlusion. The latter patient, whose aneurysm affected the origin of the PICA, was treated with additional stent placement and coil insertion and remains both clinically and radiographically stable.

**Complications and Clinical Outcomes**

No patient suffered a permanent complication as a result of endovascular treatment. The patient who required additional stent placement and coil insertion for a recurrent aneurysm sustained an iatrogenic, initially flow-limiting dissection of the V\(_3\) segment as a result of an attempt to pass a relatively stiff coronary stent into the distal artery. The patient suffered no clinical sequelae of this iatrogenic dissection, and in fact demonstrated spontaneous reopening and normal patency of the dissected segment on follow-up angiography performed 24 hours later. Despite treatment, two of the patients who presented with poor-grade SAH died due to the severity of their original hemorrhage.

Of the 21 surviving patients, clinical follow-up data were obtained in 20. The follow-up duration ranged from 15 days to 49 months (mean 14.6 months). At follow up, findings in 14 patients were normal, five had fixed neurological deficits but were functionally capable of caring for themselves, and one was permanently disabled and dependent on nursing care. The 12 patients who presented with poor-grade SAH accounted for the five cases of fixed neurological deficits and the two deaths. The remaining five patients in this subset, however, made normal recoveries.

**ILLUSTRATIVE CASES**

**Case 11**

This 48-year-old man presented with severe SAH, obtundation, and myocardial infarction. Cerebral angiography was performed after the patient’s medical condition was stabilized and an external ventricular drain was placed. Angiography revealed a V\(_3\) dissecting aneurysm involving the origin of the PICA (Fig. 1A). In an effort to preserve this vessel, we elected to perform stent-supported coil embolization (Fig. 1B and C). Despite the gravity of his original presentation, the patient made a complete neurological recovery. Follow-up angiography performed 2 years later, however, demonstrated a significant recurrence of his aneurysm (Fig. 1D). This recurrence was treated with additional stent placement and coil insertion (Fig. 1E and F). The patient remains clinically and radiographically stable 3 years after his ictus.

**Case 17**

This 38-year-old man was found unconscious at home after suffering an SAH. He presented with obtundation re-

---

**TABLE 1**

Clinical characteristics, treatment, and outcome in 23 patients with dissecting VA aneurysms*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Treatment</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>51, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>2</td>
<td>35, F</td>
<td>coil occlusion, functional w/ deficit</td>
<td>normal</td>
</tr>
<tr>
<td>3</td>
<td>36, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>4</td>
<td>54, M</td>
<td>coil occlusion</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>50, M</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>6</td>
<td>59, F</td>
<td>coil occlusion, functional w/ deficit</td>
<td>normal</td>
</tr>
<tr>
<td>7</td>
<td>53, F</td>
<td>coil occlusion, functional w/ deficit</td>
<td>normal</td>
</tr>
<tr>
<td>8</td>
<td>38, F</td>
<td>coil occlusion, functional w/ deficit</td>
<td>normal</td>
</tr>
<tr>
<td>9</td>
<td>72, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>10</td>
<td>36, M</td>
<td>SCD</td>
<td>normal</td>
</tr>
<tr>
<td>11</td>
<td>48, M</td>
<td>SCD</td>
<td>normal</td>
</tr>
<tr>
<td>12</td>
<td>40, M</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>13</td>
<td>35, M</td>
<td>coil occlusion</td>
<td>died</td>
</tr>
<tr>
<td>14</td>
<td>48, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>15</td>
<td>45, M</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>16</td>
<td>58, M</td>
<td>coil occlusion, functional w/ deficit</td>
<td>normal</td>
</tr>
<tr>
<td>17</td>
<td>38, M</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>18</td>
<td>54, M</td>
<td>coil occlusion</td>
<td>died</td>
</tr>
<tr>
<td>19</td>
<td>45, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>20</td>
<td>46, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>21</td>
<td>50, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>22</td>
<td>55, F</td>
<td>coil occlusion</td>
<td>normal</td>
</tr>
<tr>
<td>23</td>
<td>61, M</td>
<td>coil occlusion</td>
<td>disabled</td>
</tr>
</tbody>
</table>

* NA = not available; SCD = stent-supported coil delivery.
quiring intubation and renal failure related to myoglobinuria; the latter condition was likely the result of the patient having suffered his SAH several days before his discovery. After his medical condition was stabilized, the patient underwent angiography, which revealed a dissected, dilated $V_4$ segment of the VA distal to the left PICA (Fig. 2A). The aneurysm was occluded with coils throughout its length, with preservation of the PICA (Fig. 2B and C). Right VA angiography demonstrated severe vasospasm of the basilar artery consistent with the patient’s delayed presentation (Fig. 2D). After receiving endovascular and medical management of his vasospasm, the patient made a complete recovery.

**DISCUSSION**

Dissections of the intracranial vessels are rare causes of SAH, accounting for fewer than 10% of cases. Of these, most involve the posterior circulation, especially the fourth segment of the VA. These lesions are dynamic; unruptured aneurysms have a propensity to enlarge, and ruptured lesions have a proclivity to repeated hemor-
Researchers estimate the rate of rebleeding from V₄ aneurysms to be more than 30%.9,11,14,16,20,24 As expected, patients suffering rerupture are subject to a dramatically higher mortality rate (47% compared with 8% of patients without rebleeding).24

The most common symptom associated with a dissecting V₄ aneurysm is headache related to SAH.11,16,17 In such cases, SAH may be severe, often rendering these patients moribund at admission. Nonetheless, both ruptured and unruptured lesions can manifest with neck pain, cranial neuropathies from aneurysm compression, and embolic phenomena producing strokes and transient ischemic attacks.

The origin of V₄ dissecting aneurysms is multifactorial; cases associated with trauma, hypertension, and fibromuscular dysplasia have been reported.2,20,23 Because the V₄ region represents the most proximal part of the intracranial artery, this segment is likely to be subject to a higher degree of traumatic forces at the skull base, such as head rotation and flexion.2,20,23 Furthermore, the tunica media and adventitia of the VA thin as the vessel pierces the dura.2 These physical and anatomical factors surely predispose this arterial segment to injury and aneurysm formation.

The high rate of rerupture confirms what has been observed surgically: that these aneurysms are friable and thin

Fig. 2. Case 17. Preembolization and postembolization angiographic studies. A: Left VA angiogram, lateral projection, revealing a dissecting aneurysm of the left V₄ segment just distal to the PICA as well as significant intracranial vasospasm. B: Coil occlusion of the artery at the aneurysm site was performed. C: Left VA angiogram obtained after coil occlusion of the distal parent artery reveals filling of the proximal PICA. D: Right VA angiogram demonstrating filling of the basilar artery. Note the severity of the intracranial vasospasm (arrow).
Endovascular management of intracranial VA dissecting aneurysms

allowed. Histological studies reveal widespread disruption of the internal elastic lamina. This disruption eventually produces a dissection plane between the tunica media and the adventitia. Rupture into the adventitia is a typical finding. Surgical evaluation of these aneurysms often reveals several perforating arteries arising from the diseased segment. These small arteries tend to be thrombosed or occluded, explaining why patients can usually tolerate coil occlusion of the affected segment. Autopsy analysis indicates that healing occurs through neointimal hyperplasia, starting 1 week after injury and lasting longer than 1 month. This protracted process further contributes to the high rate of repeated hemorrhage.

Several angiographic features must be considered before selecting the optimal treatment for V4 dissecting aneurysms. These lesions have a variable angiographic appearance, most often demonstrating a region of vessel tapering and then enlargement, also known as a “pearl and string” configuration. This particular configuration has the highest propensity for rerupture. Irregularities such as aneurysm dilation and vessel tapering actually facilitate transarterial coil placement, because both features allow the coils to conform to the diseased segment.

Other and perhaps more important angiographic features include the location of the aneurysm in relation to the PICA and the spinal artery as well as the status of the contralateral artery. Aneurysm involvement of the PICA or spinal arterial segment precludes a deconstructive treatment such as coil occlusion of the artery at the aneurysm site. Similarly, the dominance of the affected artery complicates the treatment paradigm. In such cases, balloon test occlusion is helpful in assessing the risk of stroke after vessel sacrifice.

Initially, V4 aneurysms were treated with huntarian ligation of the proximal artery. This simple alteration of flow dynamics often successfully produced thrombosis of the aneurysm. Nevertheless, aneurysm growth and rebleeding can follow proximal ligation. This possibility prompted direct surgical approaches to the aneurysm for clip placement and wrapping. The complexity of these approaches and their high attendant morbidity and mortality rates hastened the advent of endovascular treatment.

Endovascular therapy of V4 aneurysms evolved in a similar fashion to surgical endeavors. At first, the affected artery was occluded proximally by using either detachable balloons or coils. This paradigm was again limited by cases of aneurysm recurrence and repeated hemorrhage. Subsequently, coil occlusion of the artery at the lesion site was adopted in patients in whom the aneurysm location was distinct from the origin of the PICA and the spinal artery. Cases involving either of the aforementioned sites mandate a reconstructive endovascular or bypass procedure, followed by either surgical or endovascular occlusion of the aneurysm. In patients suffering from obtundation who are unable to cooperate during test occlusion, surgical or endovascular therapy should be tailored to the clinical scenario, with the goal of selecting the treatment that offers the highest benefit and poses the least risk to the patient.

A surprising finding in this study was the high number of critically ill patients who eventually recovered functionally. Of this subset of 12 patients, five actually recovered completely. Although the literature documents that patients presenting in extremis usually do poorly, this negative expectation must be tempered by the prospect of substantial recovery. An SAH within the posterior fossa is more likely to affect the brainstem and to produce a dramatically poor neurological picture. In our series, patients who recovered from severe posterior fossa SAH tended to demonstrate signs of improvement within hours of their ictus. The potential for substantial recovery and the high rate of acute rerupture should prompt urgent treatment of patients harboring these aneurysms.

CONCLUSIONS

Coil occlusion of the aneurysm and parent artery is the treatment of choice in patients harboring dissecting lesions of the fourth segment of the VA. This procedure is associated with a very low rate of complications and a high rate of technical success. In patients whose aneurysms involve critical arterial branches or the dominant VA, attention should be turned to open surgical alternatives such as bypass. Stent-supported coil insertion is associated with aneurysm recurrence and should be reserved for patients in whom surgery is contraindicated, and used only when careful follow-up angiography can be performed. Patients suffering from these lesions often recover as dramatically as they present, and most should be treated in an urgent fashion to diminish the likelihood of repeated hemorrhage.
References


Manuscript received December 22, 2004. Accepted in final form January 6, 2005.

Address reprint requests to: Felipe C. Albuquerque, M.D., Neuroscience Publications, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, Arizona 85013. email: neuropub@chw.edu.