ENDOVASCULAR TREATMENT OF RUPTURED POSTERIOR CIRCULATION DISSECTING ANEURYSMS

S. PURKAYASTHA, K. GUPTA A, T. KRISHNAMOORTHY, N.K. BODHEY

Department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology, Trivandrum, Kerala, 695011 India.

SUMMARY

Background and aim: Dissecting aneurysms of the posterior circulation constitute a relatively uncommon subgroup of aneurysms. They account for 3-7% of cases of nontraumatic subarachnoid hemorrhage. Because of high risk, in most cases the patients require surgical or endovascular therapy. In this study we discuss the clinical efficacy of endovascular treatment with long-term follow-up in ruptured dissecting aneurysms of the posterior circulation.

Materials and methods: This retrospective study was conducted at our institution between January 1995 and June 2005. Eight patients (4 male; 4 females) ranging in age from 24 to 65 years (mean, 46.75 years), were included. All presented with SAH. Endovascular treatment was based on the configuration of the dissecting aneurysm. Attempt was made to occlude the dissecting aneurysm.

Result: A total of 8 ruptured dissecting aneurysms in the posterior circulation were treated. Out of them 5 were in the intradural vertebral artery, 2 in the basilar trunk and one in the proximal PCA. All the cases were technically successful. We have seen only two complications. The pre and post procedure (at the time of discharge) mean modified Rankin scores in the patients were 4.6 (SD 0.51) and 1.7 (SD 1.98). This improvement in Rankin score after endovascular treatment was statistically significant (Wilcoxon signed rank test, \( P = .017 \)).

Conclusion: Endovascular management of these lesions is safe and effective mode of treatment and gives adequate protection from rebleed.

Key words: dissecting aneurysm, intracranial artery, endovascular treatment.

INTRODUCTION

Dissecting aneurysms of the posterior circulation constitute a relatively uncommon subgroup of aneurysms. They account for 3-7% of cases of nontraumatic subarachnoid hemorrhage (SAH) [1]. Histologically, a thin media, adventitia and less vasa vasorum of the intradural segment of the vertebral artery predisposes it to result in SAH with pseudoaneurysm formation compared with dissections of the extradural segment [1-6]. Recurrent hemorrhage from a dissecting vertebral artery aneurysm ranges from 30% to 70% with 46% mortality [7-10]. Because of the high risk, patients usually require surgical or endovascular therapy [5, 11-15].

In this study we discuss the clinical efficacy of endovascular treatment with long-term follow-up in ruptured dissecting aneurysms of the posterior circulation.

MATERIAL AND METHODS

This retrospective study was conducted at our institution between January 1995 and June 2005. We reviewed the interventional neuroradiology databases for patients referred for evaluation and treatment of ruptured dissecting aneurysms of the posterior circulation. Eight patients (4 male; 4 females) ranging...
in age from 24 to 65 years (mean, 46.75 years), were included. All presented with SAH. Two patients also had associated right hemiparesis (power grade 3/5). One patient had ipsilateral 3rd cranial nerve palsy and another had bilateral 6th cranial nerve palsy. Hypertension was present in 7 patients, diabetes in 4 patients. Two patients had evidence of systemic autoimmune disease. Only intracranial dissecting aneurysms of the posterior circulation were included. Aneurysms were considered dissecting if one or both of the following imaging features were present: 1) the aneurysm was associated with an intimal flap, or irregular or beaded parent arterial narrowing or dilation, 2) MR or CT confirmed a false lumen involving the parent artery.

All patients underwent four- vessel angiography prior to endovascular treatment. When parent vessel occlusion was contemplated, patients were evaluated by test occlusion for 20 to 30 minutes by using a non-detachable silicone balloon. This was done when total occlusion of PCA, basilar artery segment, or in case of contralateral hypoplastic vertebral artery. If patients remained at their neurologic baseline, and the follow-up angiogram demonstrated adequate collateral flow, permanent coil occlusion (Guglielmi detachable coils (GDC), fiber coils; Target Therapeutics, Boston Scientific, Fremont, CA) of the affected artery was performed. Patients were anticoagulated during the procedure with a bolus of 5000U of heparin and then 1000U administered every hour if needed to keep the ACT around 300.

Angiograms were assessed for size, shape, and location of the dissecting aneurysm with respect to the major branches and collaterals. Each lesion was examined for evidence of extension of the dissection into adjacent arterial segments. An attempt was made to identify perforator vessels. Each dissecting aneurysm was classified as follows: lesion inferior to the origin of the posterior inferior cerebellar artery (PICA), lesion superior to the origin of the PICA, lesions involving the PICA origin, lesions at the vertebrobasilar junction, lesion of the basilar trunk and lesion in proximal posterior cerebral artery (PCA).

Endovascular treatment was based on the configuration of the dissecting aneurysm. Attempt was made to occlude the dissecting aneurysm if the lesion did not incorporate a branch such as the anterior inferior cerebellar artery (AICA), PICA, or anterior spinal artery. This was accomplished with GDCs. Also, if the dissected segment incorporated important branches such as AICA, PICA, or anterior spinal artery, proximal occlusion was used. Stent supported coil occlusion was done in case of unfavorable aneurysm anatomy, no definable neck and where parent vessel occlusion were considered dangerous. All the coiling was done by placing the microcatheter in the aneurysm sac by coaxial technique with guiding catheter in the proximal part of vertebral artery. In case of stent supported coiling, it was done either through the struts of the stents or by placing the microcatheter in the aneurysm next to the stent.

Follow-up angiography, gadolinium-enhanced MR angiography, or CT angiography was performed at 6 months to determine whether the affected segment was smaller or healed. Further examinations were obtained yearly if needed. At each follow-up detailed neurological examination were performed.

Two experienced radiologists were involved in the procedures, retrieval of data and image analysis. All the clinical data were stored in individual case files in the medical record department and the images were stored in hard copies and optical discs.

RESULTS

A total of 8 ruptured dissecting aneurysms in the posterior circulation were treated. Out of them 5 were in the intradural vertebral artery, 2 in the basilar trunk and one in the proximal PCA. An attempt was made to occlude the aneurysm sac itself and to keep the patency of the parent artery wherever possible. Total occlusion of the aneurysm sac and the parent artery were done in 6 cases and stent assisted coiling of the aneurysm was done in 2 cases. All the cases were technically successful. In the PCA aneurysm the patient passed the balloon occlusion test and the segment of the parent artery was coiled. There was pial-pial collateral filling of the PCA territory from MCA territory. In the basilar trunk aneurysms, one procedure was done with stent assistance with preservation of the parent artery, but in the other because of poor aneurysm morphology, the segment of the artery had to be sacrificed. For vertebral aneurysms, only one procedure was done with stent assistance, parent vessels were also sacrificed for all remaining lesions.

We have seen only two complications. Despite having good collateral circulation in the patient with PCA aneurysm, the patient developed a small infarct in that region 48 hours after the procedure. We kept the blood pressure little elevated (20% above mean) in that patient to avoid hypoperfusion. She was managed conservatively. One patient with vertebral aneurysm (case 4) developed severe electrolyte imbalance in the post procedure period and developed cardiac arrest and died.

The pre and post procedure (at the time of discharge) mean modified Rankin scores in the patients were 4.6 (SD 0.51) and 1.7 (SD 1.98). This improvement in Rankin score after endovascular treatment was statistically significant (Wilcoxon signed rank test, \(P = .017\)). The follow-up in the patients ranged from 5-62 months (mean 37.15 months). One patient developed mild homonymous hemianopsia due to infarction. The remaining patients were asymptomatic. There was no rebleed or recurrence of aneurysm on follow-up angiogram. The patient demography, endovascular treatment and clinical outcome in dissecting aneurysms are summarized in Table 1.

<table>
<thead>
<tr>
<th>Rankin Score</th>
<th>Patients</th>
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<tr>
<td>0-1</td>
<td>Excellent</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>Severe</td>
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Table 1: Overall outcomes were defined as excellent, Rankin score 0-1; good, Rankin score 2; moderate, Rankin score 3; poor, Rankin score 4-5; or death, Rankin score 6. The Wilcoxon signed rank test, a nonparametric test for paired samples, was used to evaluate statistical significance for any change in Rankin score.
ILLUSTRATIVE CASES

Case 4

This 56-year-old diabetic and hypertensive female presented with sudden onset of holocranial headache, more in the occipital region. It was associated with vomiting. On examination she had neck rigidity. Her GCS was 8. She had no obvious motor deficits. CT head showed evidence of SAH in the quadrigeminal and perimesencephalic cistern along with hydrocephalus. Her DSA showed a dissecting aneurysm arising from the intradural segment of right vertebral artery distal to PICA. Then it was decided to occlude the aneurysm as proximal occlusion could compromise PICA flow. With coaxial technique a microcatheter was negotiated to the aneurysmal sac which was occluded with GDC coils of various sizes. Postprocedure angiogram showed total occlusion of the aneurysmal sac with preservation of PICA flow (figure 1). The procedure was uneventful. In the post procedure period she had electrolyte imbalance, developed cardiac arrest and died.

Case 3

This 24-year-old male presented with sudden onset of headache and right-sided weakness. On examination he had GCS of 12 and motor strength of 3/5 in both right upper and lower limbs. He was detected to be mildly hypertensive but he had no other risk factors. His CT showed SAH in the pre-pontine cistern and a saccular dilation arising from the mid basilar artery. DSA showed a dissecting aneurysm arising from the mid basilar artery extending almost into the basilar bifurcation. PCAs and superior cerebellar arteries (SCA) were seen separate from the aneurysm. It was decided to occlude the aneurysm with parent vessel at the level of the aneurysm. Cross circulation studies showed filling of distal vertebral through posterior communicating. Using a coaxial technique, a microcatheter was taken to the aneurysm sac which was packed with GDC coils and the basilar artery was occluded at that level. Proximal basilar was filling from vertebral circulation and the SCAs, PCAs were filling from the anterior circulation. The procedure was uneventful and the patient was kept on anti-platelet therapy. He regained his strength completely at the time of discharge and had no problem on follow up.

Case 7

This 37-year-old female presented with sudden onset of headache, vomiting and loss of consciousness. CT showed SAH in the pre-pontine cistern. DSA
showed a wide necked dissecting aneurysm arising from the distal basilar artery with ectatic changes and vessel contour irregularity. MRI showed evidence of layered thrombus. There was diffuse vasospasm involving the basilar artery. As the SCAs were arising just distal to the aneurysm it was decided to occlude the aneurysm sac with stent assistance, preserving the parent artery. At first a LEO stent (3.5X18 mm) (BALT Extrusion, France) was placed across the aneurysmal sac and then the aneurysm was packed with GDC coils. Post procedure angiogram showed total occlusion of the aneurysm with preservation of SCA flow (figure 3). Mild persistent vessel irregularity was noted in the basilar artery. The procedure was uneventful and the patient recovered completely.

**Case 8**

This 34-year-old female presented with recurrent ischemic symptoms in the left PCA territory for last one year. She was on antiplatelet medication. She presented with sudden onset of headache with loss of consciousness and CT revealed SAH in the left perimesencephalic cistern. DSA showed a dissecting aneurysm involving the P2 segment of left PCA. Then balloon test occlusion was done in the left PCA territory and she tolerated it well. The aneurysm was then occluded with GDC coils. Post procedure angiogram showed total occlusion of the aneurysm and filling of the left PCA territory through pial-pial collaterals from the left MCA territory (figure 4). After 48 hours the patient developed signs of left PCA territory ischemia but she was managed conservatively.

**DISCUSSION**

Fusiform and dissecting aneurysms in the posterior circulation typically have a poor prognosis if left untreated. One group is comprised of those with acute dissecting aneurysms, which involve a disruption of the internal elastic lamina and intramural hemorrhage. These dissecting aneurysms typically present with subarachnoid hemorrhage (SAH) or ischemia secondary to compromise of the vessel lumen. In our series, all patients presented with hemorrhage. The second group is comprised of those with chronic fusiform aneurysms, which may arise from various vascular abnormalities that cause a defect in the arterial media [16]. These gradually enlarging aneurysms may result from congenital, acquired, or iatrogenic causes, and it is thought that atherosclerosis and hypertension may play a role in their development [17]. Chronic fusiform aneurysms typically involve the posterior circulation [18, 19].
Most cases of dissecting aneurysm are idiopathic in origin, but numerous causes have been reported in the literature including trauma, hypertension, syphilis and other arteritides, fibromuscular dysplasia, and polyarteritis nodosa [20, 21]. In almost all of our patients, these risk factors were identified.

Symptoms of vertebrobasilar dissecting aneurysms depend on factors such as size and location, including whether the lesion is intradural or extradural. Typical presentations include SAH, thromboembolic events, and cranial neuropathy from mass effect [22-24]. One patient in this series presented with thromboembolic event.

A variety of treatment strategies have been applied in patients with dissecting vertebrobasilar aneurysms, including surgical reconstruction, surgical ligation, wrapping, endovascular trapping, and various surgical bypass with occlusion strategies [5, 11-13, 25-27]. These aneurysms are difficult to treat because of their location and their morphology. Surgical access can be problematic and in some instances, complicated and technically demanding surgical procedures have been required, such as saphenous or radial artery bypass or PICA side-to-side reanastomosis [12, 26, 28]. Vessel wrapping for intradural lesions was ineffective in the small number of documented cases [11, 25].

Endovascular treatment is emerging as a first line of treatment and as an alternative to surgery in treating these lesions because of its less invasive nature associated reduced morbidity [18]. Endovascular treatment can be deconstructive or reconstructive. Sacrifice of the parent artery from which the aneurysm arises, a deconstructive procedure, has become a widely accepted approach to treatment of posterior circulation dissecting aneurysms. Advantages of endovascular treatment include the ability to monitor the neurologic status of an awake patient, to angiographically assess the adequacy of collateral flow, and to evaluate any residual flow after permanent occlusion has been performed. Reconstructive procedures involve occlusion of the aneurysm sac with preservation of the parent artery and that can be performed successfully by stent supported coil embolization. The goal of endovascular treatment is intraaneurysmal thrombosis, involution of the aneurysm, to decrease the risk of rupture, to minimize symptoms of mass effect and prevention of rebleed [13, 29, 30, 35, 36].

Results of multiple series suggest surgical ligation or coil embolization to occlude directly the affected segment of artery as the appropriate therapy for dissecting vertebral artery aneurysms [5, 11-15]. Yamaura et al. treated 19 patients with intracranial vertebral artery dissection by surgical clip-occlusion of the proximal vertebral artery. There were no postoperative deaths or rebleeding but lateral medullary syndrome developed in three patients. This might be due
to compromise of vascular supply of medulla due to clip occlusion [5]. Endovascular treatment might be less traumatic for these patients. Although, surgical or endovascular occlusion of the parent artery proximal to an aneurysm has been recommended for treatment of dissecting aneurysms of the intracranial posterior circulation, these aneurysms may rupture even after proximal occlusion because distal progression of thrombus is necessary to occlude the dissecting aneurysm completely, and this may be delayed by the presence of retrograde flow. By endovascular treatment the exact site of dissection can be occluded preserving nearby perforators. Yamaura et al. reported endovascular treatment of ruptured dissecting aneurysms aimed at occlusion of the dissected site by using Guglielmi detachable coils. They successfully treated six patients with a ruptured dissecting aneurysm in the posterior fossa by endovascular occlusion of the parent artery at or just proximal to the dissected site by using Guglielmi detachable coils. They successfully treated six patients with a ruptured dissecting aneurysm in the posterior fossa by endovascular occlusion of the aneurysm and concluded that endovascular occlusion of the dissected site is a safe, minimally invasive, and reliable treatment for dissecting aneurysms when a test occlusion is tolerated and adequate collateral circulation is present [15]. Halbach et al. also treated 15 patients with endovascular occlusion of the parent artery at or just proximal to the dissected site. In all patients angiography disclosed complete cure of the dissection. Follow-up angiography demonstrated healing of the vertebral artery dissection but persistent filling of the artery above the balloons. No hemorrhages recurred. They encountered only two minor complications related to the procedure [13]. So result of endovascular treatment is more favorable as compared to surgical treatment [5, 13, 15]. We also aimed to occlude the aneurysm sac itself rather than proximal occlusion. All procedures were technically successful. No recurrence of bleed was encountered up to the most recent follow-up.

When the aneurysm is located at the vertebrobasilar junction or in the basilar trunk, parent vessel occlusion of the involved or dominant vertebral artery is performed to decrease blood flow and facilitate shrinkage of the aneurysm. The goal is to decrease the risk of rupture and minimize symptoms of mass effect. This can be achieved by partial or complete occlusion of the parent artery. Complete angiographic occlusion gives better clinical result in these patients. Leibowitz et al. found a significant difference in outcomes for incompletely occluded patients, with relatively poor clinical recovery as compared with the subset of patients treated for angiographic cure [31]. We treated one case of basilar trunk dissecting aneurysm with complete occlusion of the affected segment (case 3). The patient recovered completely. In these cases perforators get thrombosed by luminal thrombus of the aneurysm or collaterals will develop, so complete occlusion does not pose any risk. However, attempts should be made to preserve the parent vessel as best possible.

Some giant vertebrobasilar aneurysms require bilateral vertebral artery occlusion to reduce flow in the aneurysms for the aneurysms to shrink. Sluze-
wski et al. treated 6 patients by bilateral vertebral artery balloon occlusion for giant vertebrobasilar aneurysms. At 6-22 months follow-up, three patients had a good functional outcome and showed unchanged size or shrinkage of the aneurysm on MRI. Three other patients died; one from recurrent hemorrhage, and two probably from delayed brain-stem ischemia. They concluded that bilateral vertebral artery balloon occlusion could be considered in patients with otherwise untreatable giant vertebrobasilar aneurysms. If test occlusion is not tolerated, a surgical bypass to the posterior circulation can be considered.

Steinberg et al. reported that late neurologic complications occurred in only 4% of the patients with complete or virtually complete thrombosis, as compared with 45% of the patient subset that had incompletely thrombosed aneurysms when treated by surgical parent vessel occlusion. Eighty-five percent of late neurologic complications in the subset with incomplete aneurysm thrombosis were fatal. These data suggest that the lack of complete thrombosis in these aneurysms represents a serious impediment to effective treatment. We tried to fully occlude the aneurysms by endovascular means. We have not encountered any hemorrhagic or delayed ischemic complications in them.

The most complete treatment of a dissecting aneurysm would be to exclude the segment with a trapping procedure. Although coil occlusion of the affected segment is one strategy, it poses the risk of occlusion of side branches, further injury to the vessel wall, or dislodging emboli. If the lesion extends to the basilar artery, only a segment may be coiled. The alternative strategy of proximal occlusion limits the risk of catheter and wire manipulation across a narrow or irregular segment and may allow for better collateral circulation, especially when lesions involve the origin of the PICA or anterior spinal artery. It should also be considered for lesions with configurations unlikely to hold coils in a stable position. These include fusiform or wide-necked pseudoaneurysms. Flexible intravascular stents followed by coil embolization of the aneurysm may be used to preserve the lumen especially if preservation of the parent vessel is deemed necessary.

Relatively new therapies are currently being investigated and the use of intracranial stents in the treatment of dissecting aneurysms seems to be a promising technique, and it may be that stents will offer a better therapeutic approach when aneurysm occlusion cannot be achieved. A recent report by Phatouros et al. shows the successful use of stent-supported coil embolization in the treatment of fusiform and wide

**FIG. 4.** (a) AP view of left vertebral artery injection shows the dissecting aneurysm involving the P2 segment of left PCA. (b) Balloon occlusion test in PCA. (c) After balloon occlusion test the aneurysm sac was coiled. (d) Lateral view of skull shows the coil mass. (e) Capillary phase of the left internal carotid artery injection shows the filling of PCA territory through pial-pial collaterals.
neck aneurysms. The stent mesh allows for attenuated packing of the aneurysm with less concern for herniation of coils into the parent artery. Phatouros et al reported technical success in six of the seven treated patients, with 0% 30-day peri-procedural morbidity and mortality. After a mean follow-up of 14.5 months, all patients treated with stent-supported coil embolization were at their neurologic baseline or had improved [34]. This technique can be extended for the treatment of wide necked dissecting aneurysm. We have used this technique in two of our patients and achieved technical success in both. Both patients recovered completely and no recurrent bleed was noted. So we feel it is good strategy to treat these aneurysms with preservation of the parent artery.

Although our study represents the effectiveness of endovascular treatment, it has its limitations. It lacks the prospective randomization of a clinical trial and the limited patient population lacks the power to demonstrate the real effectiveness and complication rate of the procedure in these patients. In our study experienced operators have performed the procedures. It may be a clinically relevant limitation as in practice the level of experience varies dramatically.

To conclude, dissecting aneurysms of the posterior circulation are challenging to the physicians. Ruptured intracranial dissecting aneurysm is associated with high morbidity and mortality in the absence of intervention. The management of vertebral-basilar dissection must be carefully tailored to its type of presentation and morphology of the lesion. The high risk of rebleeding within the first 24 hours requires aggressive and rapid intervention. Conservative management in cases of dissecting aneurysm may be highly risky and after diagnosis immediate endovascular treatment should be planned. Endovascular management of these lesions is a safe and effective mode of treatment and gives adequate protection to rebleed.

REFERENCES


