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ORIGINAL ARTICLE

Ruptured dissecting aneurysms arising from non-vertebral arteries of the posterior circulation: endovascular treatment perspective

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PURPOSE
Most intracranial dissecting aneurysms involve the posterior circulation, and the intradural segment of the vertebral artery is affected in majority of these. The aim of this report is to summarize the results of endovascular treatment in patients with ruptured dissecting aneurysms of the non-vertebral posterior circulation.

MATERIALS AND METHODS
During the past six years, the medical records of 23 patients with subarachnoid hemorrhage related to dissecting aneurysm arising from non-vertebral arteries of the posterior circulation were reviewed retrospectively.

RESULTS
The locations of the aneurysms were as follows: seven in the posterior cerebral artery, five in the superior cerebellar artery, six in the basilar artery trunk, and five in the posterior inferior cerebellar artery. Two basilar artery aneurysms were treated in the chronic stage with stent-assisted coil embolization. In the remaining patients, the aneurysm was coiled with or without parent vessel occlusion in the acute stage. One patient re-bled and died 20 days after initial treatment. At follow-up, recanalization had occurred in two patients, whose aneurysms were re-embolized successfully. Overall, three patients had permanent neurological sequelae, two had transient neurological sequelae, and one patient died.

CONCLUSION
Embolization with or without parent artery occlusion is feasible with an acceptable morbidity and mortality rate in the treatment of dissecting aneurysms confined to non-vertebral arteries of the posterior circulation.

Key words: • *aneurysm* • *subarachnoid hemorrhage* • *embolization, therapeutic*

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Recently, intracranial arterial dissection has been recognized more frequently to be a cause of subarachnoid hemorrhage (SAH). It receives special attention due to the challenges of its clinical course and therapies. Most intracranial arterial dissections involve the posterior circulation, predominantly the distal vertebral arteries (1, 2). We retrospectively investigated patients who presented with a diagnosis of SAH from dissecting aneurysms arising from non-vertebral arteries of the posterior circulation.

Materials and methods

During the past six years, 23 patients who had SAH related to dissecting aneurysm arising from the posterior circulation arteries other than vertebral artery were reviewed retrospectively (Table 1). The study population consisted of patients who either presented at our hospital or were referred directly to endovascular therapy from peripheral hospitals. The age range was from 36 to 65 years (mean, 52.2 years). Sixteen patients were female, and the remaining seven were male. Seventeen patients were admitted with Hunt and Hess grades I and II, three with grade III, and three with grade IV.

SAH was confirmed by computed tomography (CT) or a lumbar puncture. Intracranial aneurysmal dilatation accompanied by SAH was further confirmed by magnetic resonance imaging (MRI) in one patient who was initially accepted as having suspicious CT findings. Intradural aneurysm was confirmed as a source of bleeding by four-vessel digital subtraction angiography in all patients. The angiographic features of arterial dissection were divided into the following groups (3); "pearl and string sign" (corresponding to a fusiform dilatation associated with proximal or distal narrowing), "double lumen" (corresponding to the visualization of two channels), and "fusiform dilatation". There were no trauma-related dissections. Mycotic aneurysm was ruled out by extensively searching the patient's history, clinical evidence, and possible predisposing factors.

Treatment was performed either in the acute (three to 10 days) or chronic (later than two weeks) phase after ictus. When possible, the aim was to occlude the aneurysm with coils while preserving the parent artery. When necessary, test occlusion was performed to visualize collateral pathways during general anesthesia. Amytal test was not performed in any patient. The reason behind waiting for at least two weeks (treatment in chronic stage) was to accomplish treatment with an intravascular stent. These chronic patients were pre-medicated with aspirin 300 mg/d and clopidogrel 75 mg/d for five days prior to the embolization. Clopidogrel 75 mg/d for three months and aspirin 300 mg/d for one year were prescribed after the embolization procedure.

Results

In addition to 23 dissecting aneurysms in 23 patients, a cerebellar arteriovenous malformation was discovered in one patient (patient 15). The distribution of aneurysms by angiographic features was as follows: pearl and string sign in 8 aneurysms, double lumen in 5 aneurysms, and fusiform dilatation in 10 aneurysms.

Treatment in the acute stage

Endovascular treatment was performed in 21 of 23 patients in the acute phase. Test occlusion was performed in one patient (patient 11) with a lower basilar fusiform aneurysm. This patient had good collateral circulation and sufficient retrograde opacification of the basilar artery on angiography, while the left vertebral artery remained occluded during test occlusion (the right vertebral artery had already been occluded due to atherosclerosis).

Preservation of the parent vessel was accomplished in five acute cases (Fig. 1). Parent vessel occlusion (PVO) was inevitable in 15 acute cases (Figs. 2–6). Two of these 15 patients underwent PVO only with coil packing just proximal to the aneurysm orifice. In the remaining 13 acute patients, both the aneurysm and parent vessel were occluded successfully with embolizing agents: dense coil packing in 12 patients, and glue in one. The reason for using glue was to avoid having to navigate a coil-delivering microcatheter to this very distal aneurysm. We therefore used a thinner and softer flow-directed microcatheter to reach the aneurysm, and occluded both the aneurysm and the parent vessel with a small droplet of glue.

Table 1. Summary of 23 patients with non-vertebral dissecting aneurysm of the posterior circulation

Patient no./ Age/Sex	Aneurysm location	Aneurysm type	Treatment[a]	Treatment complication	Final GOS	Follow-up
1/46/F	PCA	PS	Selective coil	-	5	39 mo. (1-year DSA)
2/58/M	PCA	PS	PVO (selective coil)	-	5	22 mo. (re-opening at the 1-year DSA)
3/44/M	PCA	PS	PVO	Hemianopsia	4	33 mo. (1-year MRI)
4/36/F	PCA	FU	PVO	-	5	30 mo. (1-year DSA)
5/37/F	PCA	FU	Spontaneous PVO	-	5	14 mo. (6-mo. MRI)
6/50/F	PCA	FU	PVO	Hemiparesis	4	10 mo.
7/51/F	PCA	FU	PVO	-	5	6 mo.
8/60/M	BA	DL	Selective stent+coil (PVO)	Hemiparesis in re-treatment	4	62 mo. (re-opening at the 14 mo. DSA)
9/50/M	BA	DL	Selective stent+coil	-	4	27 mo. (1-year DSA)
10/59/F	BA	DL	Selective coil	-	5	20 mo. (1-year DSA)
11/54/M	BA	FU	PVO	-	5	42 mo. (1-year DSA)
12/65/M	BA	DL	Selective coil	-	4	14 mo. (1-year DSA)
13/56/F	BA-SCA	DL	Selective coil	-	Exitus	Re-bleeding (20-day DSA)
14/54/F	SCA	PS	PVO	-	5	31 mo. (1-year DSA)
15/56/M	SCA	FU	PVO	Transient cerebellar synd.	4	84 mo. (2-year DSA)
16/65/F	SCA	PS	PVO	-	4	12 mo.
17/45/F	SCA	FU	PVO	-	4	12 mo.
18/42/F	SCA	FU	PVO	-	5	6 mo.
19/45/F	PICA	PS	PVO	-	4	36 mo.
20/64/F	PICA	FU	PVO	-	3	66 mo.
21/50/F	PICA	FU	PVO	Transient CN-9 paralysis	5	9 mo.
22/50/F	PICA	FU	Selective coil	-	5	9 mo.
23/62/F	PICA	PS	PVO	-	4	3 mo.

[a]re-treatment mentioned in parantheses.
M, male; F, female; PCA, posterior cerebral artery; BA, basilar artery; SCA, superior cerebellar artery; PICA, posterior inferior cerebellar artery; PS, pearl and string; FU, fusiform; DL, double lumen; PVO, parent vessel occlusion; CN, cranial nevre; GOS, Glasgow Outcome Scale; mo., month.

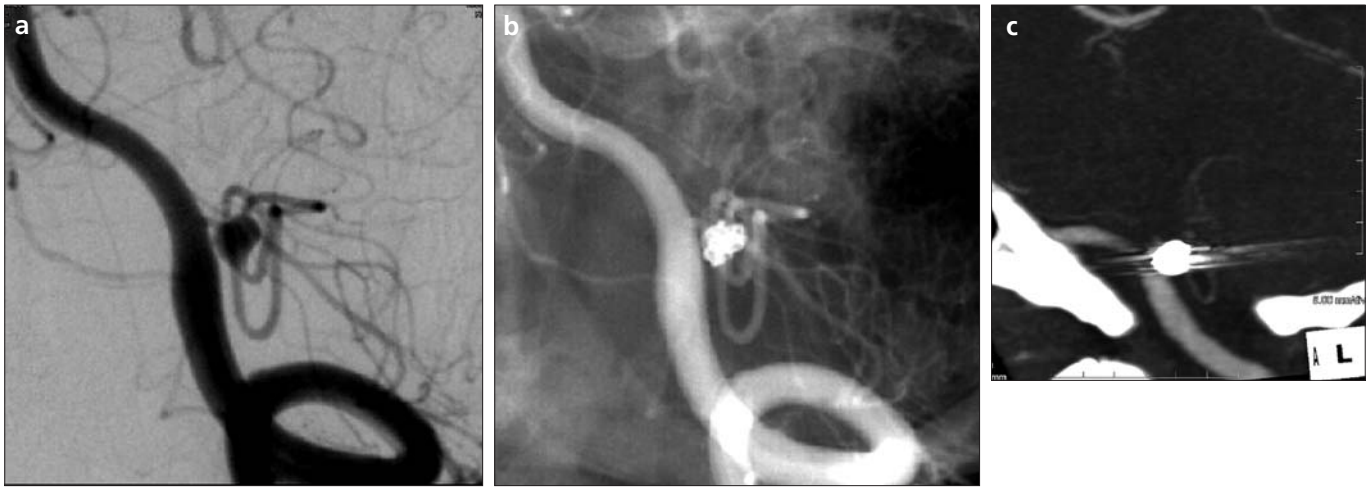

Figure 1. a–c. DSA images of patient 22 with fusiform distal posterior inferior cerebellar artery (PICA) aneurysm before **(a)** and after **(b)** embolization. Note the patent parent vessel even after full coil packing of the aneurysm. CT angiography **(c)** image obtained three months later confirms patent PICA.

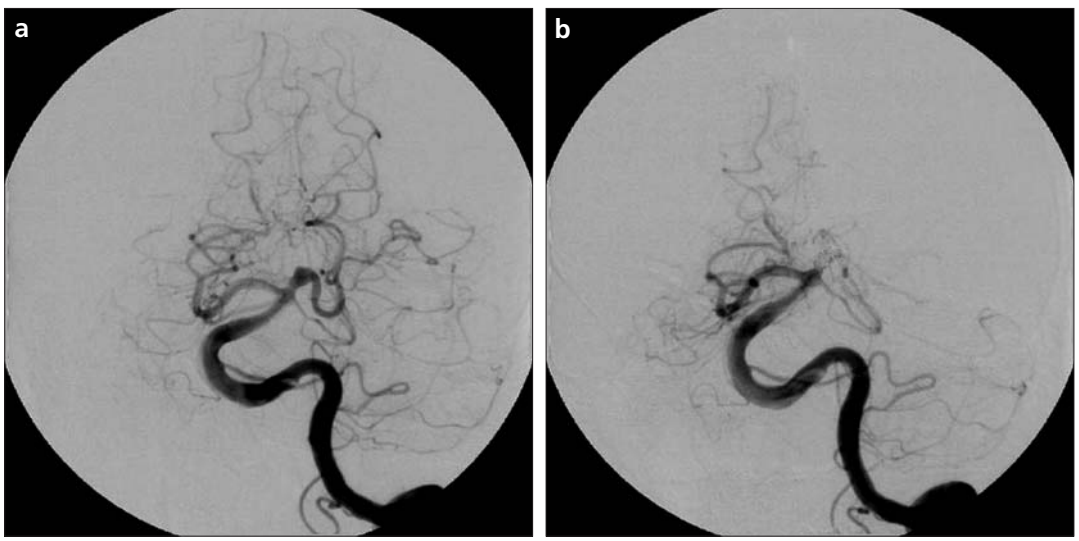

Figure 2. a, b. Patient 9 with fusiform aneurysm involving P1 and the proximal P2 segment of the left posterior cerebral artery. Anteroposterior vertebrobasilar DSA views before **(a)** and after **(b)** embolization with parent vessel occlusion.

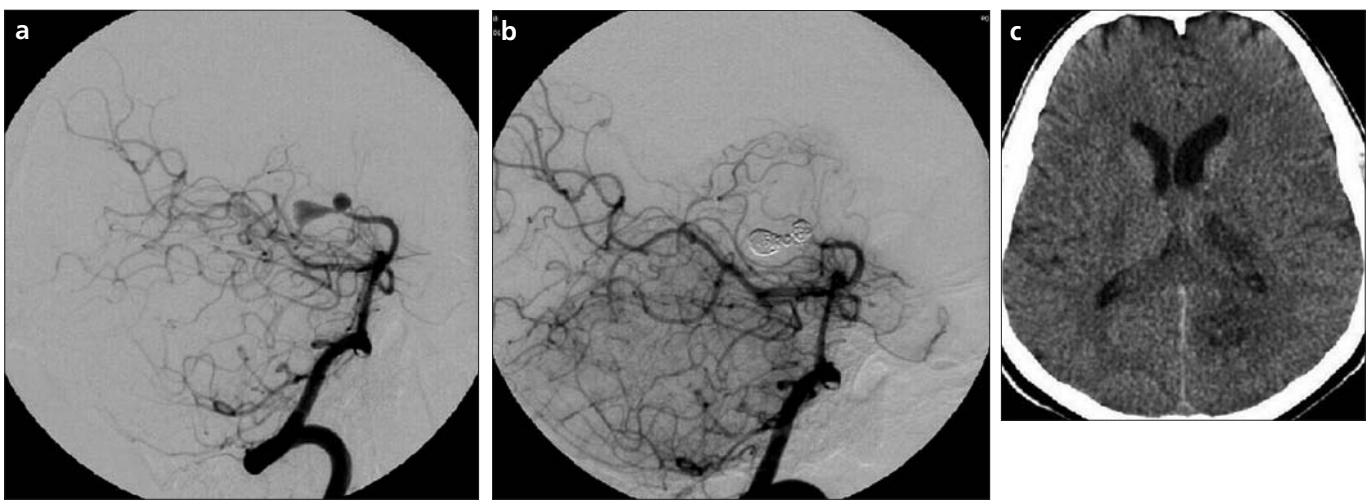

Figure 3. a–c. Patient 4 with aneurysm of the P2-P3 segment of the left posterior cerebral artery. Vertebrobasilar DSA view **(a)** at oblique-lateral projection shows dissecting aneurysm with pearl and string sign. After embolization with parent vessel occlusion **(b)** the patient experienced hemianopsia. Axial CT image **(c)** shows left occipital and thalamic infarction.

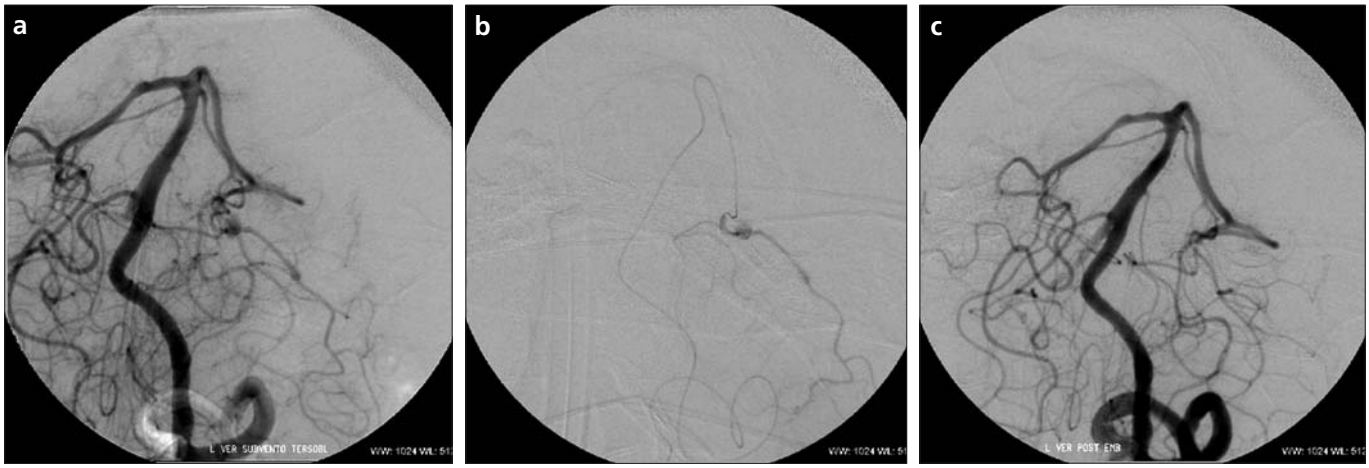

Figure 4. a–c. Patient 18 with left superior cerebellar artery (SCA) aneurysm. Vertebrobasilar angiography at submentovertical projection **(a)** shows distally located small aneurysm of the left distal SCA. Microcatheter injection just proximal to the aneurysm **(b)** demonstrates fusiform aneurysmal dilatation. Compare the post-aneurysmal 2-cm segment of the left SCA to the more distal segment in **a** and **b**. The post-aneurysmal segment has a larger caliber with a hazy contour, which confirms dissection. Final angiography **(c)** shows occlusion of both the aneurysm and parent vessel.

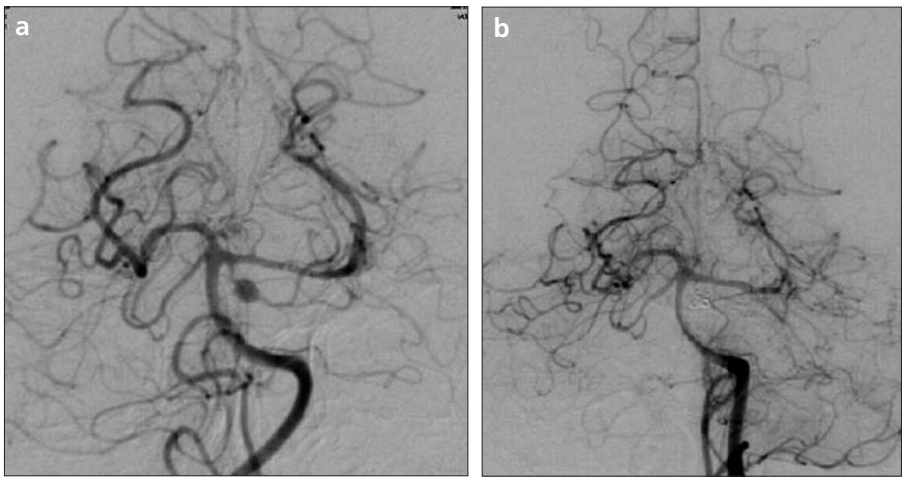

Figure 5. a, b. Patient 20 with fusiform aneurysm of the left superior cerebellar artery; initial vertebrobasilar angiography **(a)**. Final angiogram **(b)** demonstrates coil occlusion of the aneurysm and its parent vessel.

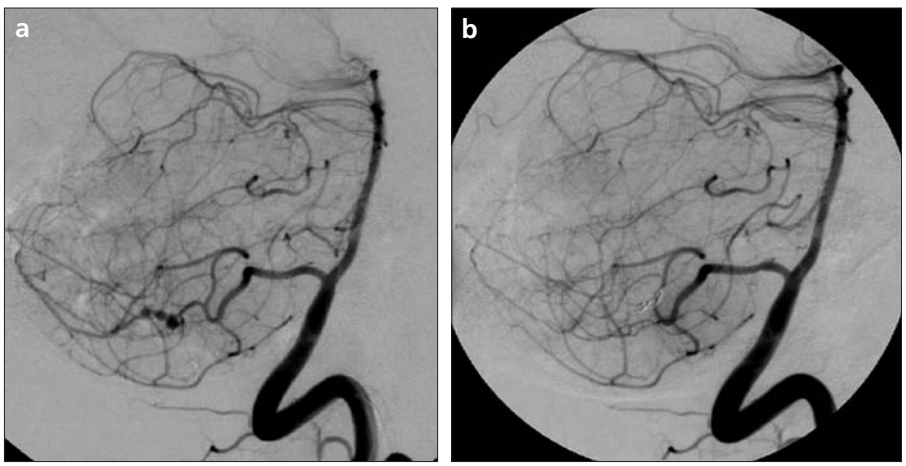

Figure 6. a, b. Patient 25 with distal posterior inferior cerebellar artery (PICA) aneurysms. Lateral vertebrobasilar angiogram **(a)** shows typical pearl and string sign. Exclusion of the aneurysms with parent vessel occlusion while sparing the main trunk and vermian branch of the PICA **(b)** is shown.

We had planned to treat by PVO the last patient (patient 5), who had a dissecting distal posterior cerebral artery (PCA) aneurysm on initial angiograms. Angiogram obtained at the beginning of the procedure surprisingly showed spontaneous occlusion of the aneurysm and its parent vessel without neurological deficits. Overall, final angiogram showed disappearance of the aneurysm in all 21 acute patients, and the parent vessel remained patent in five of them.

Treatment in the chronic stage

Endovascular treatment was performed in the chronic stage after SAH in two of 23 patients. These two patients (patients 8 and 9) had dissecting basilar trunk aneurysm necessitating prior stent implantation to accomplish stable and safe coil deposition within aneurysm. A coronary stent in patient 8, and a Neuroform stent (Boston Scientific, USA) in patient 9 (Fig. 7) were used. After stent implantation, coil embolization was accomplished via microcatheter passing through the stent mesh.

Complications of initial treatment

Two technical complications (8.5%) occurred during endovascular treatment. One technical problem was that acute thrombosis developed in patient 8 during stent-assisted coil embolization of the basilar trunk aneurysm. IV infusion of tirofiban at a dose of 2 mg in 15 minutes rapidly cleared all thrombus fragments from the circula-

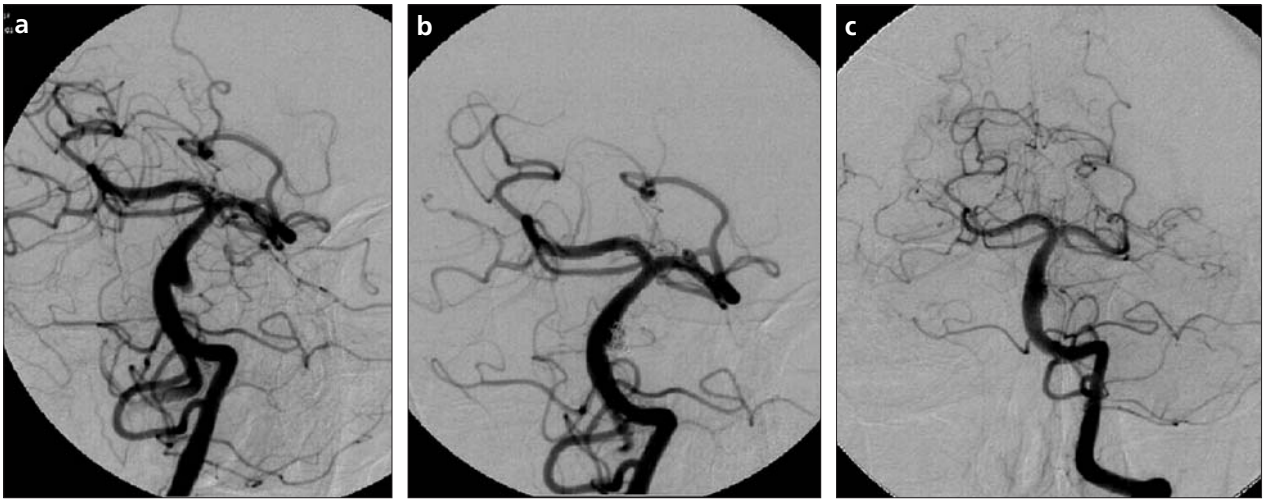

Figure 7. a–c. Patient 11 with dissecting aneurysm of the basilar trunk. Right-oblique vertebrobasilar angiograms before **(a)** and after **(b)** endovascular treatment with stent-assisted coil embolization. One-year follow-up angiogram **(c)** shows stable occlusion of the aneurysm except for a small bulging at the neck. Also note the residual dilatation of the entire basilar artery due to hemodynamic consequences of the healing process following long segment dissection.

tion. Thromboembolic occlusion of the distal PCA was another technical complication in patient 18, with a distal fusiform superior cerebellar artery (SCA) aneurysm. This occlusion readily disappeared after simple mechanical fragmentation of the thrombus by using a micro-guidewire. These two technical problems did not result in any apparent clinical consequences. There were four clinical complications (17%). Two of them were permanent, and two were transient. There was one death within 30 days after treatment due to re-bleeding of the partially thrombosed large dissecting aneurysm (Table 1).

Follow-up and re-treatment

All 22 patients were re-examined clinically at one month and six months postoperatively. There were two permanent neurological sequelae; patient 3 had hemianopsia, and patient 6 had hemiparesis which corresponded to Glasgow Outcome Scale (GOS) of 4 (moderate disability). On radiological follow-up (MRI in two patients, angiography in 10), there were two recurrences (Table 1).

One recurrence was in patient 8, who had a basilar trunk aneurysm treated previously with stent-assisted coil occlusion. The structure of the recurrent aneurysm rendered it impossible to occlude completely by using another stent and coil combination (prior to the era of the self-expanding stent). The patient underwent basilar artery (BA) test occlusion during bilateral carotid artery angiography. The aneurysm and parent artery at the aneurysm neck were occluded by coils because good posterior communicating arteries were observed bilaterally. The patient suffered left hemiparesis and facial paralysis after the procedure due to brain stem infarction. He had a GOS score of 4 (moderate disability) at the 6-month clinical follow-up.

The second recurrence was in patient 2, with a PCA P2 segment dissecting aneurysm treated initially with coil packing, which resulted in PVO. Follow-up angiography at one year showed reopening of both the parent vessel and a small segment of the previous aneurysm. This small aneurysm could be occluded completely with preservation of the parent vessel.

All told, there were three permanent (12.7%) and two transient (8.5%) neurological sequelae, and one patient died (4.2%) within 30 days after treatment.

Discussion

Intracranial dissection is the cause of SAH in well below 10% of patients with ruptured aneurysm (3); however, the true incidence of SAH caused by a dissecting aneurysm is not known and is probably underestimated (1). This is mainly because there is still non-uniformity in the nomenclature. One of two recent reports has designated these aneurysms as "dissecting related fusiform aneurysm" (2), while the other has termed the same entity "peripheral aneurysm", never mentioning dissection (4).

In general, more than 80% to 90% of all intracranial dissecting aneurysms were located in the posterior circulation, and at least three-fourths to four-fifths of these were located in the V4 segment of the vertebral artery (1–3). The diagnosis of dissecting aneurysm is based primarily on angiographic findings. In a Japanese nationwide study, the most common angiographic finding in the hemorrhagic dissecting aneurysm group was fusiform dilatation (42%), followed by the "pearl and string" sign (34%), and then by narrowing (15%) (3). Angiographic results of our patients were comparable to those presented in this Japanese study.

In general, the natural history of untreated ruptured dissecting intracranial aneurysm is thought to be unfavorable. Aoki and Sakai (5) found a re-bleeding rate of 30% in a review of 60 reported cases of ruptured vertebral artery dissection. This re-bleeding rate was much higher in the series of Mizutani et al. (6), who described 71% re-bleeding in patients with vertebrobasilar dissecting aneurysms. Several recent reports concur with these reports of relatively high rates of re-bleeding and mortality from such aneurysms (7). Comparing those morbidity rates to those of unsecured dissecting intracranial aneurysm, our results have further supported the assertion that the endovascular technique is feasible with acceptable morbidity-mortality rates in ruptured dissecting aneurysms of non-vertebral arteries of the posterior circulation.

Distally located or branch artery dissecting aneurysms can be difficult to treat with embolization techniques that spare the parent artery. In these cases, PVO during aneurysm embolization may be inevitable. Today, PVO remains the definitive treatment of dissecting intradural vertebral artery aneurysm in the presence of a patent contralateral vertebral artery. PVO of non-vertebral arteries of the posterior circulation, on the other hand, may be problematic. Beneficial (i.e., elimination of ruptured aneurysm) and harmful (e.g., possible infarction distal to occlusion site) effects of PVO must be weighed in every case. In addition to the risk of distal infarction, brainstem ischemia due to the occlusion of proximal perforators may be another cause of neurologic morbidity in case of PVO involving the P1 or P2 segments of the PCA, and the anterior medullary segment of the posterior inferior cerebellar artery (PICA).

In our series, clinical symptoms of perforator ischemia occurred in only one of the four instances of P1 segment occlusion (patient 6); no perforator ischemia was detected in the remaining two P2 occlusions. One proximal PICA occlusion at the anterior medullary segment resulted in brainstem infarction (patient 21) in our series, while the remaining three occlusions distal to the caudal loop did not. The feasibility and efficacy of endovascular PVO of various non-vertebral arteries of the posterior circulation has been demonstrated by several teams (1, 4, 8–22).

In our group of patients, fusiform aneurysms and wide-necked aneurysms arising from small caliber distal branches were treated by PVO. The aim of embolization in these cases is to occlude the aneurysm first, and subsequently to occlude the parent artery as close to the aneurysmal neck as possible. Thus, the proximal portion of the parent vessel, as well as the perforating arteries arising from these segments, remains patent. Retrograde filling of the distal segments is expected from the pial collateral circulation.

We reviewed the results from the most recent series focusing on aneurysms of the non-vertebral posterior circulation that were treated by PVO. We divided these aneurysms into 4 groups: PCA, SCA, anterior inferior cerebellar artery (AICA), and PICA aneurysms. Table 2 shows a summary of the literature review including our 14 patients. There was 32.6% neurological deficit in PCA territory. The majority of the deficits (28.5%) were permanent, including hemiparesis (6.3%) and hemianopsia, or visual loss (22.2%). All neurologic deficits in the PICA and SCA territory were transient cerebellar symptoms with rates of 21.7% and 27.7%, respectively. Although the number was relatively low (10 cases), there were five (50.0%) PVO-related neurologic deficits in AICA territory, two (20.0%) of which were permanent, including paralysis in the distribution of cranial nerves 7 and 8.

Spontaneous BA dissection that does not involve the vertebral arteries is an uncommon, yet increasingly recognized, cause of SAH. In fact, an extensive review of the world literature reported in 1997 by Amin-Hanjani et al. (23) revealed that fewer than 20 such cases have ever been reported. Of these patients, however, 19% experienced fatal re-bleeding and nearly 60% either died or became severely disabled within days to months after the hemorrhage. These data reveal the devastating natural history of these lesions.

Management of BA dissecting aneurysms is challenging. Vessel dissection may result in simple saccular aneurysm with variable neck width (small and wide); complex saccular aneurysm with small, wide, or no neck; or nonsaccular fusiform aneurysm (circumferential dissection) without a neck. Because simple coil occlusion is not possible in many patients, additional endovascular strategies, techniques, and devices are needed. Endovascular options currently available are intrasaccular coiling, concomitant occlusion of the aneurysm and parent vessel with coils, proximal occlusion of the parent vessel with coils or balloons to obtain flow reversal, stent-assisted coiling, and stenting alone across the aneurysm neck.

Detailed reports of endovascular treatment of dissecting BA aneurysms are scarce; most of the literature consists of individual cases within a larger group of aneurysms. For those aneurysms difficult to treat with selective endosaccular coil embolization, and for aneurysms for which it is impossible to preserve the BA even by using balloon- or stent-assisted techniques, endovascular occlusion of both the aneurysm and BA is a definitive although worrisome option. Most recently, Wenderoth et al. (24) and Hassan et al. (25) have published their case series describing successful endovascular treatment of aneurysms with BA trunk occlusion. Hassan et al. (25) have stated that transient neurological deficits were a constant finding after occlusion of the BA, but final outcomes were good in both series (24, 25).

Even when there is good collateral circulation, occlusion of the BA may also result in neurological deficits due to the occlusion of small brainstem perforators. It is believed that occlusion of the lower BA or vertebrobasilar junction is usually better tolerated than is occlusion of the mid or upper BA, due to the fact that the lower BA has fewer brainstem perforators. Proximal occlusion of the both vertebral arteries (flow reversal) or occlusion of one vertebral artery only (flow diminishing) is another alternative if there is good collateral circulation, although this is not

Table 2. Reported (1, 4, 8–22) neurological complications in patients with aneurysms of the non-vertebral posterior circulation treated by parent vessel occlusion (including our 14 patients)

	Neurological complications		
Vessel (number)	Transient (%)	Permanent (%)	Total (%)
PCA (49)	2 (4.0)	14 (28.5)	16 (32.6)
SCA (18)	5 (27.7)	-	5 (27.7)
PICA (23)	5 (21.7)	-	5 (21.7)
AICA (10)	3 (30.0)	2 (20.0)	5 (50.0)
Total (100)	15 (15.0)	16 (16.0)	31 (31.0)

PCA, posterior cerebral artery; SCA, superior cerebellar artery; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery.

definitive therapy for these aneurysms. Recent attention has focused on neuroendovascular arterial reconstruction of complex and dissecting vertebrobasilar aneurysms using intracranial stents, both alone (26) and in concert with coils.

The most important disadvantage of stenting is the necessity to use pre- and postembolization medication (i.e., dual anti-aggregants). It is generally accepted that stent should not be used in acute SAH due to the risk of anti-aggregation. The role of stent technology in the treatment of these lesions in either the acute or chronic stage after SAH is a promising and evolving new solution; however, further data regarding the long-term efficacy of stent technology are required before the uniform application of this modality.

In conclusion, as is true for other intracranial dissecting aneurysms, early intervention is recommended in non-vertebral arteries of the posterior circulation to prevent re-bleeding in dissecting aneurysms previously revealed by SAH. When occlusion of the dissecting site of the parent vessel was performed with coils, embolization of the aneurysm was a safe and efficient treatment with acceptable risk of ischemic complications in peripherally located and/or small-vessel aneurysms. In case of BA dissecting aneurysm, in combination with coils, a self-expanding intracranial stent covering the dissection may offer an opportunity to preserve the patency of the parent vessel while the aneurysm is occluded. However, our stent series is too small to draw final conclusions, which must await further study.

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