Stent Placement for the Treatment of Intracranial Vertebral Artery Dissecting Aneurysms

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Summary: Intracranial vertebral artery (VA) dissecting aneurysms are commonly treated with endovascular parent artery occlusion. However, this procedure cannot be applied to patients with aplastic/hypoplastic contralateral VA or with posterior inferior cerebellar artery (PICA) involved in the dissecting segment. Recently, endovascular treatment using stents for VA dissecting aneurysms has been reported. In this study, we investigated the safety, efficacy, and optimal application of this treatment.

Thirteen patients were treated using stents to preserve the patency of the VA, PICA, and anterior spinal artery (ASA). In 6 patients, PICA or ASA was involved in the dissecting segment. Seven patients presented with subarachnoid hemorrhage (SAH), 4 with ischemic symptoms, 1 with headache without SAH, and 1 with incidental discovery. Eleven patients were treated with stent-assisted coil embolization and 2 with stenting only.

Stents were successfully deployed in all patients. Of the 11 patients treated with stent-assisted coiling, complete obliteration of the aneurysm was achieved in 5 patients and residual dome filling was present in 5. In the remaining patient, aneurysm rupture occurred during the insertion of the coils, and therefore parent artery occlusion was performed. In 2 patients treated with stenting only, complete obliteration was confirmed by follow-up angiography. Growth of the aneurysmal dilatation occurred in 1 patient, but subsequent SAH was not observed in any patient.

Endovascular treatment using stents and coils appears safe and effective, and provides an effective alternative to treating patients with aplasia/hypoplasia of the contralateral VA and some patients with involvement of the origin of PICA or ASA in the dissecting segment.

Key words: vertebral artery, dissecting aneurysm, stent


Introduction

Intracranial vertebral artery (VA) dissecting aneurysms are commonly treated by parent artery occlusion (PAO) including the aneurysmal dilatation through the endovascular approach. However, this procedure cannot be applied to patients with aplastic/hypoplastic contralateral VA or with posterior inferior cerebellar artery (PICA) involved in the dissecting segment. Moreover, delayed thrombosis of the VA stump may cause occlusion of the anterior spinal artery (ASA) or the perforating arteries to the medulla. Recently, endovascular treatment using stents for VA dissecting aneurysms has been reported. Here, we retro-
Table 1  Summary of the patients treated by stenting only or stent-assisted coiling

<table>
<thead>
<tr>
<th>No.</th>
<th>Age(yr)</th>
<th>Sex</th>
<th>Onset</th>
<th>Shape and size of the aneurysm (diameter × length)</th>
<th>Location of the aneurysm</th>
<th>Reasons for stenting</th>
<th>Interval between onset and treatment</th>
<th>Procedures (size of the stents)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>M</td>
<td>SAH (grade 3)</td>
<td>aneurysmal dilatation (7 × 10 mm)</td>
<td>distal to PICA</td>
<td>hypoplasia of the contralateral VA</td>
<td>5 days</td>
<td>stent-assisted coiling (S670 3.5 × 12 mm)</td>
</tr>
<tr>
<td>2</td>
<td>48</td>
<td>M</td>
<td>Wallenberg</td>
<td>aneurysmal dilatation with intimal flap (4 × 5 mm)</td>
<td>distal to PICA</td>
<td>preservation of the ASA</td>
<td>46 days</td>
<td>single stenting (S670 3.0 × 9 mm)</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>M</td>
<td>SAH (grade 3)</td>
<td>aneurysmal dilatation (11 × 8 mm)</td>
<td>distal to PICA</td>
<td>narrowing of the contralateral VA due to dissection</td>
<td>25 days</td>
<td>stent-assisted coiling (S670 3.5 × 12 mm)</td>
</tr>
<tr>
<td>4</td>
<td>65</td>
<td>F</td>
<td>SAH (grade 4)</td>
<td>aneurysmal dilatation (8 × 4 mm)</td>
<td>distal to PICA</td>
<td>hypoplasia of the contralateral VA</td>
<td>15 days</td>
<td>stent-assisted coiling (S670 3.5 × 12 mm)</td>
</tr>
<tr>
<td>5</td>
<td>64</td>
<td>M</td>
<td>dysarthria</td>
<td>aneurysmal dilatation with intimal flap (5 × 5 mm)</td>
<td>distal to PICA</td>
<td>preservation of the ASA</td>
<td>10 days</td>
<td>single stenting (S670 3.5 × 12 mm)</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>M</td>
<td>vertigo</td>
<td>aneurysmal dilatation with intimal flap (6 × 6 mm)</td>
<td>involving PICA</td>
<td>preservation of the PICA</td>
<td>21 days</td>
<td>stent-assisted coiling (S670 3.5 × 12 mm)</td>
</tr>
<tr>
<td>7</td>
<td>56</td>
<td>M</td>
<td>Wallenberg</td>
<td>aneurysmal dilatation with intimal flap (7 × 14 mm)</td>
<td>distal to PICA</td>
<td>PICA end of the contralateral VA</td>
<td>35 days</td>
<td>overlapping stenting (Driver 4.0 × 24 mm, Driver 4.0 × 18 mm)</td>
</tr>
<tr>
<td>8</td>
<td>41</td>
<td>M</td>
<td>SAH (grade 1)</td>
<td>aneurysmal dilatation (8 × 9 mm)</td>
<td>distal to PICA</td>
<td>preservation of the ASA</td>
<td>28 days</td>
<td>stent-assisted coiling (Driver 4.0 × 24 mm)</td>
</tr>
<tr>
<td>9</td>
<td>53</td>
<td>M</td>
<td>SAH (grade 5)</td>
<td>aneurysmal dilatation with proximal VA narrowing (11 × 13 mm)</td>
<td>involving PICA</td>
<td>preservation of the PICA</td>
<td>4 days</td>
<td>stent-assisted coiling (Driver 4.0 × 24 mm)</td>
</tr>
<tr>
<td>10</td>
<td>73</td>
<td>F</td>
<td>SAH (grade 1)</td>
<td>aneurysmal dilatation (10 × 10 mm)</td>
<td>involving PICA</td>
<td>preservation of the PICA and ASA</td>
<td>2 days</td>
<td>stent-assisted coiling (Driver 3.5 × 18 mm)</td>
</tr>
<tr>
<td>11</td>
<td>65</td>
<td>M</td>
<td>headache</td>
<td>aneurysmal dilatation with proximal VA narrowing (7 × 7 mm)</td>
<td>proximal to PICA</td>
<td>hypoplasia of the contralateral VA</td>
<td>19 months</td>
<td>stent-assisted coiling (Driver 2.5 × 14 mm)</td>
</tr>
<tr>
<td>12</td>
<td>65</td>
<td>M</td>
<td>SAH (grade 3)</td>
<td>aneurysmal dilatation (6 × 6 mm)</td>
<td>distal to PICA</td>
<td>aplasia of the contralateral VA</td>
<td>0 days</td>
<td>stent-assisted coiling (Driver 3.0 × 15 mm)</td>
</tr>
<tr>
<td>13</td>
<td>64</td>
<td>F</td>
<td>incidental</td>
<td>aneurysmal dilatation with proximal VA narrowing (10 × 10 mm)</td>
<td>no PICA</td>
<td>hypoplasia of the contralateral VA</td>
<td>unknown</td>
<td>stent-assisted coiling (Driver 2.75 × 14 mm)</td>
</tr>
</tbody>
</table>

WFNS, World Federation of Neurosurgical Societies; SAH, subarachnoid hemorrhage; VA, vertebral artery; PICA, posterior inferior cerebellar artery; ASA, anterior spinal artery.

respectively review 10 patients treated with stents, and investigate the safety, efficacy, and optimal application of this treatment.

**Patients and methods**

Seventy-one patients with VA dissecting aneurysms underwent endovascular treatment from October 1993. In this period, none underwent direct surgery. Clinical presentation was subarachnoid hemorrhage (SAH) in 52 patients, ischemic symptoms in 13, headache and/or neck pain in 5, and incidental discovery in 1. All ruptured VA dissecting aneurysms were indicated for endovascular treatment. Unruptured VA dissecting aneurysms with relatively large or growing aneurysmal dilatations were indicated for endovascular treatment. Selection of the treatment modality for VA dissecting aneurysms in our hospital was as follows: PAO was a primary treatment modality, and stent-assisted coil embolization or stenting only was indicated for patients who were at high risk of developing ischemic complications after PAO. Fifty-eight patients were treated by PAO (proximal PAO in 7 and PAO including the aneurysm in 51). Thirteen patients (7 patients presenting with SAH, 4 with ischemic symptoms, 1 with headache without SAH, and 1 with incidental discovery) were treated with stents, and were included in this study (Table 1).

These 13 patients had aneurysms located “distal to the PICA” in 8, “proximal to the PICA” in 1 and “involving the PICA” in 3. In 4 patients, ASA was involved in the dissecting segment. The angiographic features of the lesions were aneurysmal dilatation in 6, aneurysmal dilatation with intimal flap in 4, and aneurysmal dilatation with proximal VA narrowing in 3. Stenting only or stent-assisted coil embolization was indicated for various reasons: aplasia/hypoplasia of the contralateral VA, bilat-
eral dissections, and preservation of the PICA/ASA. Eccentrically projecting large aneurysms were chosen for stent-assisted coil embolization and circumferentially dilated small aneurysms for stenting only. Aspirin 100 mg and ticlopidine 200 mg (or clopigrel 75 mg) were administered orally before treatment and were continued for 3 months.

In patients with SAH, the duration between rupture and treatment was 0 to 25 days (mean 8.1 days). Endovascular treatment was performed under general anesthesia. Two guiding catheters were introduced into the VA for stent-assisted coil embolization; one for placing the stent and the other for inserting the coils. Balloon expandable S670 and Driver coronary stents (Medtronic AVE, Santa Rosa, CA) were used in all patients. The stent size was selected to equal the diameter of the parent artery and to sufficiently cover the orifice of the aneurysm. The stent was positioned across the lesion and was released by inflating the balloon with nominal pressure. The balloon was kept in the stent lumen to provide backup against aneurysm rupture during the procedures. A microcatheter was then introduced into the aneurysm through the stent strut (Patients 1, 3, 4, 10, and 12), or was introduced into the aneurysm before the deployment of the stent (Patients 5, 6, 8, 9, 11, and 13), to allow insertion of the coils if rupture of the aneurysm or parent artery occurs during deployment of the stent. The aneurysm was then embolized using coils. If necessary, the balloon was inflated to prevent the coils protruding into the stent lumen.

**Patient 7 (Fig. 1)**

A 56-year-old man suffered sudden onset of headache, vertigo, and hypesthesia of the left side of the face and left upper extremity. CT revealed no SAH. Magnetic resonance (MR) imaging showed a high intensity area in the left posterolateral portion of the medulla, but MR angiography showed no aneurysm. Repeat MR angiography obtained 1 week later showed left VA dissecting aneurysm. Left vertebral angiography demonstrated an aneurysmal dilatation with intimal flap of the left VA and the PICA end of the contralateral VA. Follow-up angiography performed 2 weeks later showed no change of the aneurysm. After informed consent, he underwent
endovascular treatment 35 days after the onset. He received oral aspirin (100 mg) and clopidogrel (75 mg) for 3 days before the treatment. A Driver stent (4.0 × 24 mm) was deployed in the lesion, and a second Driver stent (4.0 × 18 mm) was placed inside the first stent. Immediate angiography showed no change in the intraneurysm flow. He was discharged home without additional neurological deficits. Antiplatelet therapy was continued for 2 weeks after the stenting. Complete obliteration of the aneurysm was confirmed by CT angiography 1 month later and by angiography 4 months later.

Patient 8 (Fig. 2)

A 41-year-old man presented with sudden onset of headache and was referred to another hospital. CT revealed SAH. Left vertebral angiography showed a VA dissecting aneurysm. Conservative treatment was chosen because the ASA originated from the aneurysm. Three weeks later, he suffered onset of severe headache and became transiently unconscious. CT revealed rebleeding, and left vertebral angiography demonstrated enlargement of the aneurysm. He was transferred to our hospital. Mild ataxia and hemisensory disturbance were observed. MR imaging showed a high intensity area in the left posterolateral medulla and cerebellum. Endovascular treatment was performed 6 days after rebleeding. Three-dimensional (3-D) rotational angiography showed that the aneurysm projected eccentrically. The ASA was involved in the dissecting segment, but originated from the relatively intact VA. Therefore, we
selected stent-assisted coil embolization of the aneurysmal dilatation. Two guiding catheters (6F and 5F) were introduced into the left VA. A microcatheter was introduced into the aneurysm, and a Driver stent (4.0 × 24 mm) was deployed to sufficiently cover the lesion. The stent delivery balloon was exchanged for a compliant HyperGlide balloon (4.0 × 15 mm; MicroTherapeutics, Inc., Irvine, CA) which was positioned in the orifice of the aneurysm. The aneurysmal dilatation was then embolized using coils with balloon inflation to prevent the coils from protruding into the stent lumen. The parent artery and the ASA were preserved, but the aneurysm dome was slightly opacified. His ataxia recovered after rehabilitation, and he was discharged with independent ambulation. Follow-up angiography 3 months after the treatment showed complete obliteration of the aneurysm, despite enlargement of the aneurysm and distal displacement of the coils in the aneurysm. The origin of the ASA was occluded, but the ASA was retrogradely fed by the segmental arteries.

**Patient 10 (Fig. 3)**

A 73-year-old woman presented with SAH, and was transferred to our hospital 2 days later. Left vertebral angiogram showed a left VA dissecting aneurysm involving the left PICA and the ASA in the dissecting segment. Left internal carotid artery was filled from the posterior communicating artery because of the occlusion at its cervical portion. Contralateral VA was hypoplastic. Therefore, stent-assisted coil embolization was performed. Flow retention of the left VA due to stretching of the coil at the VA origin was observed when an 8F guiding catheter was placed in the left VA. After deployment of a Driver stent (3.5 × 18 mm), coil embolization of the aneurysm was performed with preservation of the PICA and ASA. After the treatment, she suffered ataxia and mild aphasia due to infarction in left cerebellum and angular cortex. Hypoperfusion of the left VA and internal carotid artery during the procedures was considered a cause of infarction. Follow-up angiography performed 7 months later showed growth of the aneurysmal dilatation, but subsequent SAH did not occur.

**Results**

Stents were successfully positioned across the lesion,
and deployed by nominal pressure in all patients (Table 2). However, the released stents were displaced backward during removal of the balloon in Patients 1 and 3.

Coil embolization of the aneurysmal dilatation was performed in 11 patients, and only stenting with a single stent in 1 and overlapping stents in 1.

Of the 11 patients treated with stent-assisted coiling, complete obliteration of the aneurysm was achieved in 5 patients and residual dome filling was present in 5. Aneurysm rupture occurred during the insertion of the coil, so PAO was performed in Patient 1. Ischemic complication occurred in 1 patient (Patient 10) during the procedures.

In 2 patients treated with stenting only, the aneurysmal dilatation remained patent immediately after the treatment, and complete obliteration was confirmed by follow-up angiography.

Follow-up angiography performed in all the patients (follow-up period 2–12 months, mean 4.5 months) showed complete obliteration in 9, residual dome filling in 1, residural neck filling in 2, and growth of the aneurysmal dilatation in 1 patient. Ischemic events and subsequent SAH were not observed in any patient (follow-up period 6–91 months, mean 40.6 months). Modified Rankin scale of the 13 patients was 0–1 in 10, 3 in 1 and 5 in 2 (aneurysm rupture during the procedure in Patient 1 and brain damage due to the initial SAH in Patient 9).

### Discussion

**Problems of parent artery occlusion**

PAO for VA dissecting aneurysms remains the treatment of choice. However, this procedure cannot be applied to patients with aplasia/hypoplasia of the contralateral VA and with involvement of the origin of PICA or ASA in the dissecting segment. Moreover, ischemic events due to delayed thrombosis of the VA stump may occur.

If posterior communicating artery is well-developed, PAO may be indicated for the patients with aplasia/hypoplasia of the contralateral VA. However, when sufficient outflow (PICA) is absent, delayed thrombosis of basilar artery can cause brain stem infarction. Therefore, PAO is difficult to be applied to “distal to the PICA type” VA dissecting aneurysms.

In our series treated by PAO, ischemic complications occurred in 9 of 58 patients (15.5%). Eight patients presented with Wallenberg syndrome, and 1 patient died of respiratory disturbance due to occlusion of the ASA.\(^{10}\) The ASA is formed by the paired anterior ventral spinal arteries originating from the VA and has anastomoses with the anterior radicular arteries.\(^{10}\) Therefore, occlusion of the anterior ventral spinal artery on one side rarely causes ischemic symptoms. However, catastrophic complications can occur if the ASA originates only in the affected VA and is involved in the VA stump formed by PAO.\(^{5}\) Our recent study showed the VA stump and ASA did not thrombose in most cases as

### Table 2  Results of the treatment

<table>
<thead>
<tr>
<th>No.</th>
<th>Immediate angiographic results</th>
<th>Complications</th>
<th>Follow-up angiographic findings (mo.)</th>
<th>Subsequent rupture (mo.)</th>
<th>Outcome (mRS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>parent artery occlusion</td>
<td>aneurysm rupture</td>
<td>complete obliteration (2)</td>
<td>none (91)</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>no change</td>
<td>none</td>
<td>complete obliteration (6)</td>
<td>none (77)</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>complete obliteration</td>
<td>stent displacement</td>
<td>complete obliteration (4)</td>
<td>none (77)</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>complete obliteration</td>
<td>none</td>
<td>complete obliteration (12)</td>
<td>none (73)</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>complete obliteration</td>
<td>none</td>
<td>complete obliteration (3)</td>
<td>none (70)</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>complete obliteration</td>
<td>none</td>
<td>complete obliteration (3)</td>
<td>none (62)</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>complete obliteration</td>
<td>none</td>
<td>complete obliteration (4)</td>
<td>none (16)</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>residual dome filling</td>
<td>none</td>
<td>complete obliteration (3)</td>
<td>none (14)</td>
<td>1</td>
</tr>
<tr>
<td>9</td>
<td>residual dome filling</td>
<td>none</td>
<td>residual dome filling (2)</td>
<td>none (14)</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>residual dome filling</td>
<td>cerebral infarction (angular cortex, cerebellum)</td>
<td>growth of the aneurysmal dilatation (7)</td>
<td>none (11)</td>
<td>3</td>
</tr>
<tr>
<td>11</td>
<td>residual dome filling</td>
<td>none</td>
<td>residual neck filling (3)</td>
<td>none (9)</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>residual dome filling</td>
<td>none</td>
<td>residual neck filling (3)</td>
<td>none (8)</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>complete obliteration</td>
<td>none</td>
<td>complete obliteration (6)</td>
<td>none (6)</td>
<td>0</td>
</tr>
</tbody>
</table>

mo., months; mRS, modified Rankin scale.
there was sufficient residual sump effect from the ASA if its origin was preserved. Therefore, we believe that preservation of the orifice of the ASA is most important.

**Indication for stenting only and stent-assisted coil embolization**

Stenting only and stent-assisted coil embolization, which can preserve the patency of the parent artery, PICA, and ASA, are both potential treatments in patients with high risk of developing ischemic complications after PAO. However, these procedures are inferior to PAO for the prevention of rupture, and also need antiplatelet therapy to prevent in-stent thrombosis. In the treatment of ruptured aneurysms, early thrombosis is essential because recurrent rupture occurs in the acute period after the initial bleeding. Therefore, these procedures, especially stenting only, are difficult to apply to patients with ruptured aneurysm.

Stent placement carries relatively low risk. Stenting across the lesion can obliterate the aneurysm by repairing the dissecting arterial wall, altering the flow dynamics within the aneurysm, and promoting neointimal proliferation. However, immediate obliteration of the aneurysm is rare and whether complete obliteration is achieved in the follow-up angiography is uncertain. The complete obliteration rate is higher with overlapping stents (67 to 100%) than with a single stent. If stenting only is used for the prevention of bleeding, overlapping stents are recommended. Accordingly, good candidates for stenting only are unruptured, circumferentially dilated aneurysms, and aneurysmal dilatations too small to accommodate coil embolization.

Stent-assisted coil embolization carries a higher risk of rupture during the procedure than PAO or stenting only. Furthermore, recurrence of the aneurysm or rebleeding may occur. Follow-up angiography of our Patient 8 showed coil migration into the enlarged aneurysm, but the aneurysm did not recanalize. We suppose that the stent prevented recanalization of the aneurysm by promoting neointimal formation. The eccentrically dilated aneurysms which are large enough to accept coils are good candidates for this procedure. This procedure may also be indicated for PICA- or ASA-involved aneurysms, if these arteries originate from the relatively intact and non-dilated arterial wall (Patients 8, 9 and 11). 3-D rotational angiography is useful for estimating the anatomical relationship between the aneurysm and the origins of the PICA and ASA.

**Technical consideration of stenting only and stent-assisted coil embolization**

Balloon expandable coronary stents were used in all our patients because self-expandable stents (Neuroform; Boston Scientific, Natick, MA; and Enterprise: Cordis Endovascular, Miami Lakes, FL) are not available in Japan. The stent size was selected to equal the diameter of the parent artery and to sufficiently cover the orifice of the aneurysm. If an over-sized stent is selected, rupture of the aneurysm or parent vessel may occur. In contrast, an under-sized stent may be displaced downward during removal of the balloon (Patients 1 and 3). Therefore, we selected a longer stent if available to anchor to the parent artery in later patients. Self-expandable stents, which can be anchored securely to the parent artery with less stress, are better than balloon expandable stents. However, coronary stents have low porosity compared with self-expandable stents, and may be effective if additional coiling is not planned because thrombosis of the aneurysm depends on the porosity of the stent struts.

When 2 guiding catheters could be placed in the VA, a microcatheter was introduced into the aneurysm before stent placement because navigation of a microcatheter into the aneurysm through the stent strut may be difficult in some patients and because the coils can be immediately inserted if aneurysm rupture occurs during deployment of the stent. Aneurysm rupture is also prone to occur during insertion of the coils because the stent restricts the movability of the microcatheter and so the coils directly compress the aneurysmal wall. The stent delivery balloon, which is sometimes exchanged for a more compliant HyperGlide balloon, is left inside the stent lumen and inflated if aneurysm rupture occurs. This balloon is also available for preventing coil protrusion and for confirming if the coils protrude into the parent artery through the stent strut.

**Conclusion**

PAO for VA dissecting aneurysms remains the treatment of choice. Endovascular treatment using stents and coils, which can preserve the patency of the parent artery, PICA, and ASA, provides a good method for treating patients with VA dissecting aneurysms with high risk of developing ischemic symptoms after PAO. Stenting only is indicated for unruptured, circumferentially dilated small aneurysms, and stent-assisted coil embolization for ruptured, eccentrically dilated large aneurysms.
References


要 旨

解離性椎骨動脈瘤に対するステントを用いた血管内治療

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目的：解離性椎骨動脈瘤の治療は、血管内手技による母血管閉塞が第一選択の治療法であるが、虚血性合併症が問題となる。今回われわれは、血管内治療を施行した71例のうち、両側解離、対側の椎骨動脈奇形、後下小脳動脈(PICA)や前脳幹動脈(ASA)の血行鈍化などの理由からステントを併用した血管内治療を行った13例を検討した。方法：くも膜下出血発症が6例、虚血発症が4例、incidentalが1例であった。6例で解離部にPICAまたはASAがinvolveされていた。ステント併用内腔内術11例、ステント留置のみ2例であった。結果：ステント併用内腔内術を施行した11例のうち、1例で内腔内術中に動脈瘤が破裂し、結果的に母血管閉塞となった。6例で動脈瘤の完全消失が得られ、4例でdome fillingであった。PICAまたはASAがinvolveされた6例では、ステントを併用し拡張部のみにコイルを留置することにより血行を温存できた。ステント留置のみの2例はfollow-upで拡張部の血流を確認した。虚血性合併症を1例に認めた。治療後、くも膜下出血をきたしたものはなかった。転帰はMRS 0-1が10例、3が1例、5が2例であった。結論：母血管閉塞により虚血性合併症の危険がある症例に対しては、ステントを併用した治療が有用と考えられる。PICAまたはASAがinvolveされた症例でもステントを併用し血行を温存できる場合がある。