Endovascular treatment of blood blister–like aneurysms of the internal carotid artery

JAE HYO PARK, M.D.,1 IN SUNG PARK M.D.,2 DAE HEE HAN, M.D.,1 SEONG HYUN KIM, M.D.,3 CHANG WAN OH, M.D.,3 JEONG-EUN KIM, M.D.,4 HYUN JIB KIM, M.D.,2 MOON HEE HAN, M.D.,4 AND O-KI KWON, M.D.3

Departments of 1Neurosurgery and 4Radiology, Seoul National University College of Medicine; 2Clinical Neuroscience Center, Bundang Seoul National University Hospital; and 3Department of Neurosurgery Gyeong Sang National University Hospital, Seoul, Republic of Korea

Object. Because of its thin wall, an aneurysm arising from the posterior wall of the internal carotid artery (ICA), the so-called blood blister–like aneurysm (BBA), is difficult to manage surgically and is often associated with high morbidity and mortality rates. The authors treated these aneurysms endovascularly. In this paper, they present angiographic and clinical results obtained in patients with ICA BBAs treated endovascularly.

Methods. In seven patients with ICA BBAs who presented with subarachnoid hemorrhage, a total number of 12 endovascular treatments were performed, including seven endosaccular coil embolizations (four conventional, two stent-assisted and one balloon-assisted procedure) in four patients and five endovascular ICA trapping procedures in five patients. Repeated endovascular treatments were undertaken in four patients. In two patients, the endovascular treatment was performed after failure of surgical treatment (one case of rebleeding after clip placement and one aneurysmal regrowth after wrapping). A balloon occlusion test (BOT) was performed in all patients prior to ICA trapping.

All four patients treated by endosaccular coil embolization showed aneurysmal regrowth. Neither stents nor balloons helpfully prevented aneurysmal regrowth. Of these four patients, two experienced rebleeding. These two patients remained vegetative at the last follow-up examination. After the BOT, ICA trapping was performed with coils and balloons without complication in five patients; excellent outcomes were achieved in all cases but one in which the patient had been in poor neurological condition due to rebleeding after surgical clip therapy.

Conclusions. All ICA BBAs that were treated by endosaccular coil embolization exhibited regrowth of the aneurysm. Some of the lesions rebled. The majority of patients who underwent ICA trapping experienced excellent outcomes. Based on the authors’ experiences, they suggest that ICA trapping including the lesion segment should be considered as a first option for definitive treatment if a BOT reveals satisfactory results. Regarding trapping methods, endovascular treatment may be preferred because of its convenience and safety.

Key Words • blood blister–like aneurysm • internal carotid artery • endovascular treatment • aneurysm trapping

The term “blood blister–like aneurysm” has been used for aneurysms protruding from the anterior wall of the ICA and has also been described as dorsal wall, anterior wall, and superior wall aneurysms.5,9 These ICA BBAs are characterized by highly fragile walls and poorly defined necks, making surgical exploration and clip placement hazardous.1,5,9,10,13 Because of their fragility, and despite variously proposed neurosurgical strategies, such as clip placement, wrapping, clip placement on wrapped material, and ICA trapping,1,3,8,10,13 the surgical morbidity and mortality rates and the risk of aneurysm regrowth and rebleeding even after surgery remains high.1,3,5,9,10,14

For several years we have undertaken endovascular treatments, such as endosaccular coil embolization, with or without stents or balloons, and segmental ICA trapping with detachable coils and balloons. In this article, we present our experiences and treatment outcomes.

Clinical Material and Methods

Patient Population

We reviewed all records of seven patients with ICA BBAs who presented with SAH and who underwent endovascular treatment between May 2003 and August 2005 at our institute. Twelve cases involving unruptured aneurysms at the same location were excluded. Angiographically all seven aneurysms showed typical features of ICA BBAs; they appeared as slightly elevated hemispheric blebs that
Blood blister–like ICA aneurysm

originated on the dorsal surface (superior surface) of the ICA with no relation to an arterial branch. Among these, rapid growth to saccular configurations was confirmed by short-term follow-up angiography in five aneurysms. The other two aneurysms were treated on the day of first angiographic examination that revealed small blebs on the ICA. Six of the seven patients were female. The mean age was 35.4 years (range 24–45 years). All patients presented with SAH, and the Hunt and Hess grade was II in six patients and IV in one patient. Endovascular treatments included seven endosaccular coil embolizations (four conventional, two stent-assisted, and one balloon-assisted procedure) in four patients and five endovascular procedures in which the ICA was trapped in five patients. Balloon occlusion test was performed in all patients prior to ICA trapping. The postoperative conditions were evaluated at the time of discharge and last follow up. Table 1 provides a summary of the patients’ characteristics.

Endosaccular Coil Embolization

In four patients (Cases 1–4), a total of seven endosaccular coil embolizations were performed. All procedures were conducted after induction of general anesthesia. A stent-assisted procedure (Neuroform stent, Boston Scientific) was performed in two patients (Cases 2 and 3) and a balloon-assisted procedure (Hyperglide system, EV3 Inc.) was used in one patient (Case 4).

Endosaccular coil embolization was mainly performed during the early period of our experience (Cases 1 and 2). In Case 3, rebleeding and regrowth of the aneurysm occurred 5 weeks after clip surgery, but ICA trapping was impossible because of the absence of an ipsilateral A1 segment. In addition, because of the patient’s poor neurological condition, bypass surgery could not then be attempted. Therefore, stent-assisted endosaccular coil embolization was performed for temporary protection. In Case 4, it was also undertaken for temporary protection because angiograms obtained during the BOT showed asymmetry due to vasospasm at the ipsilateral A1 segment. Three weeks later, repeated BOT was performed after vasospasm improvement. The patient underwent segmental ICA trapping successfully.

Balloon Occlusion Test

In conscious patients, BOT was conducted after application of a local anesthetic. First, cerebral angiography was performed via one femoral artery and the presence of the bilateral A1 segments was confirmed. If either the ipsilateral or contralateral A1 segment was not visualized, BOT was not attempted. If critical branches such as AChA or large PCoA were involved in the lesion segment intended for occlusion, BOT was performed depending on the clinical situation.

For BOT, an 8-F arterial sheath was inserted at the contralateral femoral artery, and an 8-F guiding catheter (Guidersofttip, Boston Scientific) was placed at the ipsilateral ICA. Through this guiding catheter, a nondetachable balloon (Sentry, Boston Scientific) was introduced and positioned at the petrous or cavernous ICA segment. While inflating the balloon, the collateral flow through the ACoA was checked by obtaining a contralateral ICA angiogram. If the angiogram demonstrated definitive asymmetry, no clinical test was performed. If the collateral flow was adequate and symmetrical, balloon inflation was maintained and the clinical test was performed for a minimum of 10 minutes. During the test, and while keeping the balloon inflated, the 8-F guiding catheter was slightly retrieved to the common carotid artery level, and an angiogram, which was virtually an ECA angiogram because the ICA was now occluded by the balloon, was acquired to check the ophthalm-

<table>
<thead>
<tr>
<th>Case No. (treatment)</th>
<th>Age (yrs), Sex</th>
<th>Hunt &amp; Hess Grade</th>
<th>Treatment</th>
<th>Regrowth†</th>
<th>Rebleeding</th>
<th>Vasospasm</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 1st</td>
<td>31, F</td>
<td>II</td>
<td>observation</td>
<td>yes (11 days)</td>
<td>during the 1st CE</td>
<td>yes</td>
<td>VS</td>
</tr>
<tr>
<td>2nd</td>
<td></td>
<td></td>
<td>CE</td>
<td>yes (11 days)</td>
<td>11 days after 1st CE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td></td>
<td></td>
<td>CE</td>
<td>yes (12 mos)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 1st</td>
<td>41, F</td>
<td>IV</td>
<td>observation</td>
<td>yes (12 days)</td>
<td>2 days after 1st bleeding</td>
<td>yes</td>
<td>VS</td>
</tr>
<tr>
<td>2nd</td>
<td></td>
<td></td>
<td>CE</td>
<td>yes (6 days)</td>
<td>6 days after 1st embolization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td></td>
<td></td>
<td>CE (stent)</td>
<td>FU</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 1st</td>
<td>37, F</td>
<td>II</td>
<td>clip</td>
<td>yes (5 wks)</td>
<td>5 wks after clip placement</td>
<td>no</td>
<td>VS</td>
</tr>
<tr>
<td>2nd</td>
<td></td>
<td></td>
<td>CE (stent)</td>
<td>yes (2 mos)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td></td>
<td></td>
<td>CE</td>
<td>FU</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4th</td>
<td>45, F</td>
<td>II</td>
<td>wrapping</td>
<td>yes (20 days)</td>
<td>during wrapping</td>
<td>no</td>
<td>GR</td>
</tr>
<tr>
<td>1st</td>
<td></td>
<td></td>
<td>CE (balloon)</td>
<td>yes (3 wks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2nd</td>
<td></td>
<td></td>
<td>ET</td>
<td>no (7 mos)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td></td>
<td></td>
<td>ET</td>
<td>no (10 mos)</td>
<td></td>
<td>yes</td>
<td>GR</td>
</tr>
<tr>
<td>5</td>
<td>32, F</td>
<td>II</td>
<td>ET</td>
<td>no (10 mos)</td>
<td></td>
<td>no</td>
<td>GR</td>
</tr>
<tr>
<td>6</td>
<td>24, M</td>
<td>II</td>
<td>ET</td>
<td>no (15 mos)</td>
<td></td>
<td>no</td>
<td>GR</td>
</tr>
<tr>
<td>7</td>
<td>38, F</td>
<td>II</td>
<td>ET</td>
<td>no (10 mos)</td>
<td>during ET</td>
<td>no</td>
<td>GR</td>
</tr>
</tbody>
</table>

* Balloon = balloon-assisted technique; CE = endosaccular coil embolization; ET = endovascular ICA trapping; FU = no follow up or follow-up period was too short to conclude; GR = good recovery; stent = stent-assisted technique; VS = vegetative state.
† Value indicates the time after treatment that regrowth was detected.
mic flow. The patient was asked whether he or she experienced any changes in visual acuity. In our series, no objective vision test was used. If there was a clear visualization of ophthalmic flow or choroidal blush from the ECA collateral structures and no change in visual acuity was reported, we considered that the ICA, including its OphA origin, could be sacrificed. When the OphA was not visualized via the ECA collateral structures, only a short segment of the ICA was occluded with coils while saving the OphA (Case 6).

The origin of the PCoA was commonly involved in the lesion segment to be trapped. Because of its location, the PCoA could not be considered as a source of effective collateral blood flow. If the PCoA is large enough or of the fetal type and included in the lesion segment, then therapeutic strategies other than trapping should be considered. Regardless of its size, if the PCoA was apart from the lesion segment, BOT was performed under the assumption that the artery could be preserved during ICA trapping.

In cases not amenable to a local anesthetic, BOT was performed after induction of general anesthesia. In these cases, a decision was made based on angiographic analysis alone. Collateral blood supply and bilateral angiographic symmetry, especially venous-phase symmetry, were evaluated. In patients with good collateral blood flow or venous-phase symmetry, the BOT is considered to be successful.

In our cases, the BOT was successful six times in five patients (twice in Case 4 with a 3-week interval). Five BOTs were performed after application of a local anesthetic and one after induction of general anesthesia. The clinical test was successful in all patients in whom angiography revealed adequate and symmetrical collateral vessels. In one patient (Case 4) the test failed because of vasospasm at the ipsilateral A1 segment. Heparin was not given during the BOT. Perfusion studies such as SPECT were not conducted.

Occlusion of the ICA: Internal Trapping With Coils and Balloons

After a successful BOT, the ICA was trapped. Through the 8-F guiding catheter, already placed in the ICA for the BOT, two 10-series microcatheters, such as Excelsior 10 (Boston Scientific) and Prowler-10 (Cordis Endovascular), or one 18-series microcatheter (ProwlerPlus, Cordis Endovascular) and one 10-series microcatheter were introduced while the nondetachable balloon, which was also used for the BOT, was already positioned at the petrous or cavernous ICA.

To perform segmental ICA trapping effectively and safely, the coils should be precisely placed to save adjacent branches and properly packed to prevent recanalization. For this purpose, we used two microcatheters for coil delivery and a balloon for proximal flow control. First, one microcatheter was placed at the ICA–aneurysm segment, while the other was placed in the distal ICA region after passing the aneurysm segment. If difficulties were expected while crossing the aneurysm, the second microcatheter was positioned in the same region as the first. The balloon, already positioned at the petrous or cavernous ICA, was then inflated to control proximal ICA flow. Proximal balloon occlusion was necessary for two reasons. First, technically it was very difficult or often impossible to place coils precisely and stably within the ICA lumen due to strong ICA blood flow. Second, because the aneurysm segment had very fragile walls, deploying coils within this segment itself could be hazardous. Thus, to prevent and reduce the consequences of premature rupture during compact coil packing, controlling the ICA flow was very important.

The operator introduced a detachable coil through the proximally located microcatheter and then deployed it partially or fully to make an initial frame. The distally placed microcatheter was retrieved and positioned at the desired location within the coil frame. Proper positioning of this microcatheter may be critical for compact coil packing, and this can be safely done by retrieving the microcatheter. Positioning microcatheters by advancing (not by retrieving) them may be very difficult or sometimes impossible because the inflated balloon at the proximal ICA holds the microcatheters tightly and because the first coil interferes with direct visualization of the second microcatheter tip. Even a slight deflation of the balloon may lead to an unexpected movement of inserted coils.

Additional smaller detachable coils were inserted and deployed for compact coil packing. Coils were delivered, deployed, and detached through the second microcatheter. Not until the entire coil frame became stable was the first coil detached. Sometimes fibered nondetachable coils were added for proximal ICA occlusion but not within the lesion segment. After achieving stable and compact coil packing, the balloon was deflated and removed. If ophthalmic collateral vessels were intact from the ECA, the ICA segment, including OphA origin, was occluded, and the cervical ICA was also occluded with detachable balloons (Gold Valve Balloon, Nycomed) or coils.

In our series, ICA internal trapping was accomplished in five patients (Cases 3–7). The PCoAs originated from the opposite side of the aneurysms in three patients (Cases 3–5), and these arteries were very small. In these patients, the PCoAs were occluded together with the lesions, whereas in the other two patients (Cases 6 and 7) the distance from the origin of PCoA to the distal margin of the aneurysm segment was approximately 2 mm, and ICA trapping was performed while saving the arteries.

The distances between the origin of the AChA and the distal aneurysm margin were 0 mm (Case 3), 1 mm (Case 5), 2 mm (Case 4), and 4 mm (Cases 6 and 7). In Case 3, the AChA was occluded with the ICA because the patient had already suffered hemiplegia due to previous hemorrhage. In the other cases (Cases 4–7), the AChAs were all saved. In Case 6, OphA collateral flow from the ECA system was not evident, and thus only the diseased segmental ICA was trapped with coils while saving the OphA.

Single-photon emission computed tomography, before and after Diamox administration, was performed in all patients who underwent ICA trapping at the time of discharge from the hospital; none of these patients experienced a perfusion deficit. For these patients, daily aspirin (100 mg) was prescribed and the patient was given an education of lifestyle modifications to prevent degenerative vascular diseases such as atherosclerosis, diabetes, and hypertension. The necessity for regular follow up for the early detection of potential cerebral perfusion problem was also stressed.
**Blood blister–like ICA aneurysm**

**Results**

**Endovascular Treatment**

Four patients (Cases 1–4) underwent endosaccular coil embolization. The total number of endosaccular embolizations was seven because three procedures were necessary due to regrowth (Cases 1–3). Complications were procedure-induced ruptures in three cases (Cases 1–3) and acute in-stent thrombosis in one case (Case 3), which was relieved by the intraarterial injection of ReoPro. Angiographically, complete occlusion was achieved in three procedures (the second embolization in Cases 1–3), whereas very small neck remnants remained after four procedures. Follow-up angiography was performed after five endosaccular embolizations. All follow-up angiograms showed aneurysm regrowth, which included all cases treated with stent- and balloon-assisted embolization. The aneurysms that recurred were almost twice the original size in Cases 1 and 3 and approximately 1.5-fold the size in Cases 2 and 4. In all cases of recurrent disease, the aneurysm necks were elongated and coil masses were moved away from the neck. Rebleeding occurred in two patients (Cases 1 and 2). In terms of treatment for the regrowth, repeated endosaccular coil embolization was performed in Cases 1 and 2 because of poor collateral structures and poor neurological condition, which prevented ICA trapping and bypass surgery. In Case 3, elective bypass surgery and endovascular ICA trapping were performed 3 months later. In Case 4, the ICA was trapped with coils after BOT on the day of follow-up angiography.

Endovascular ICA trapping was accomplished in five patients (Cases 3–7). It was performed as a first treatment in three patients (Cases 5–7). In Case 4, it was performed after confirming regrowth of the aneurysm 3 weeks after endosaccular embolization. Vasospasm developed in three cases (Cases 1, 2, and 5). In Cases 1 and 2, it resulted in permanent neurological deficits despite intraarterial papaverine infusion, triple-H (hypertension, hypervolemia, and hemodilution) therapy, and pentothal coma therapy. In Case 5, the patient had undergone ICA trapping several days before mild but symptomatic vasospasm developed. Papaverine angioplasty was successfully conducted across the ACoA. The patient fully recovered.

Clinically, the patients in Cases 1 and 2 who had undergone endosaccular embolization remained in a vegetative state due to rebleeding and vasospasm. The patient in Case 3 also remained in a vegetative state due to rebleeding that occurred 5 weeks after clip therapy. All patients who underwent endovascular ICA trapping, except the patient in Case 3, experienced excellent outcomes and returned to normal life. During the mean 1-year follow-up period of Cases 4 through 7, SPECT was performed every 6 months and all studies showed the absence of a perfusion deficit.

**Illustrative Cases**

**Case 1**

**History and Examination.** This 31-year-old woman presented with sudden severe headache. A CT scan showed diffuse SAH. Her neurological status was unremarkable except for signs of meningeal irritation. Angiography revealed multiple thrombotic filling defects at the supraclinoid portion of the right ICA but without definite aneurysm dilation (Fig. 1A). The patient was transferred to our hospital 11 days after the first SAH. Repeated angiography demonstrated a saccular aneurysm at the supraclinoid portion of the right ICA (Fig. 1B) and severe and diffuse vasospasm of the middle cerebral artery and the anterior cerebral artery. Surgical treatment was abandoned because of severe vasospasm. The angiographically documented configuration of the aneurysm was thought to be amenable to endosaccular coil embolization.

**Operation.** After induction of general anesthesia, coil embolization was performed, but during the second coil deployment contrast medium leakage suggesting an aneurysmal rupture was observed. Fortunately the leakage stopped soon after further coil placement. The aneurysm was nearly completely obliterated (Fig 1C). Following coil embolization, intraarterial papaverine (150 mg) was administered through a microcatheter to treat the vasospasm. The patient recovered well.

**Postoperative Course.** One day after the embolization, the patient developed left hemiparesis and repeated angiography confirmed severe vasospasm. An intraarterial papaverine (300-mg) infusion was then successfully performed. After treatment, the patient fully recovered until the 11th day, when a sudden severe headache developed. A CT scan showed SAH on the right sylvian fissure, and an angiogram demonstrated regrowth of the aneurysm over the previously embolized site (Fig. 1D). Repeated endosaccular embolization obliterated the aneurysm completely and eventfully (Fig. 1E), but postembolization CT scanning revealed a large right frontal intracerebral hematoma, prompting us to perform hematoma evacuation and decompressive craniotomy. The patient tolerated this procedure but suffered a neurologically severe deficit. Twelve-month follow-up angiography showed regrowth of the aneurysm in the region of the obliterated neck, but no further treatment has been performed (Fig. 1F).

**Case 3**

**History and Examination.** This 37-year-old woman experienced a sudden severe headache 4 days before admission to the hospital. A neurological exam revealed normal findings. A CT scan revealed SAH, and angiography displayed a blood blister–like lesion on the right distal ICA. The absence of the right A segment made internal trapping impossible (Fig. 2A). Surgical clipping of both the aneurysm and parts of the ICA was chosen.

**Operation.** On the day of the patient’s angiographic examination, a right frontotemporal craniotomy was performed. The sylvian fissure was opened widely and the right frontal lobe was carefully retracted to expose the right ICA. Clots with fibrous materials that surrounded the BBA were not fully dissected to prevent rupture. A curved clip was applied parallel to the ICA so that its blades could catch the arterial wall beyond the lesion. After clipping, the ICA and the clip were wrapped with many pieces of gauze and commercial thrombin material.

**Postoperative Course.** The patient tolerated the procedure well, but during the 5th week postsurgery she became comatose. A CT scan demonstrated right frontal intracerebral
hematoma and intraventricular hemorrhage. Angiography revealed slippage of the aneurysm clip and regrowth of the aneurysm (Fig. 2B). Because the patient’s neurological condition was too poor for bypass surgery and ICA trapping, endosaccular embolization was chosen, although at this time we well appreciated the risks of endosaccular embolization for these lesions. We decided to use a stent-assisted technique to reduce the risk of aneurysm regrowth. A Neuroform stent (Boston Scientific) was deployed, and the aneurysm was nearly completely occluded with detachable platinum coils (Fig. 2C); however, multiple thrombi developed around the stent, and the ICA blood flow began to diminish. ReoPro (6 mg) was given intraarterially and the thrombi resolved. The patient recovered from general anesthesia but remained in poor neurological condition. Follow-up angiography performed 2 months later revealed regrowth of the aneurysm (Fig. 2D). The surgical clip and coils had moved away from the neck. Bypass surgery and ICA trapping were chosen but due to the patient’s poor condition, the surgery was delayed. For temporary protection, repeated endosaccular coil embolization was performed. The aneurysm was nearly completely occluded after placement of detachable coils. Several days later, bypass surgery involving a radial artery graft was successfully performed, but surgical ICA trapping failed because the fibrous adhesion around the ICA was too tight to be dissected safely. One day after the bypass surgery, endovascular coil-based ICA trapping was successfully performed. The patient has remained with a severe neurological deficit for 8 months.

Discussion

Internal carotid artery BBAs are characterized by extremely fragile walls that make surgical exploration and clip therapy hazardous.\textsuperscript{1,5,9,10,13} Although surgery has been the preferred treatment, many surgeons appear to be reluctant to proceed because of the fragile nature of these lesions.\textsuperscript{9,15} Various surgical tactics and strategies have been reported including clip placement, wrapping, clip placement over the wrapping material, and ICA trapping.\textsuperscript{1,8,10,13} Regardless of surgical modality, however, all procedures pose a high risk intraoperatively (premature rupture and ICA laceration) and postoperatively (regrowth and rebleeding). In many reports the authors have demonstrated that clip surgery alone may provoke aneurysm avulsion and ICA laceration.\textsuperscript{5,9,14} Special clip deployment tactics such as placing the blade parallel to the axis of the ICA—to include the neck and parts of the ICA—also pose high risks of aneurysm avulsion and clip slippage.\textsuperscript{15} Moreover, parallel clipping is not always possible because of the difficult anatomical situation in the operating fields. As was shown in one of our cases, wrapping of the aneurysm does not seem to prevent regrowth and rebleeding.\textsuperscript{10} Placing a clip over wrapping material that envelops the entire circumference

Fig. 1. Case 1. A: Right ICA angiogram (lateral view) obtained on the day of symptom onset, showing multiple thrombotic filling defects at the supraclinoid ICA without definite aneurysm dilation. B: Repeated angiogram obtained on the 11th day after the initial angiogram, demonstrating a saccular aneurysm at the supraclinoid ICA. C: Right ICA angiogram (anteroposterior view) after endosaccular coil embolization. The aneurysm is nearly completely obliterated. D: Angiogram acquired after rebleeding showing regrowth of the aneurysm. E: Angiogram showing repeated endosaccular coil embolization that obliterated the aneurysm completely (anteroposterior [left] and lateral [right] views). F: A 3D angiogram obtained at 12 months after the last embolization showing aneurysmal regrowth.
of ICA has been reported, but rebleeding or regrowth rates remain high.\textsuperscript{1} The use of wrapping material such as gauze or sheets to envelop an ICA may not be straightforward. Moreover, as our cases show, the ICA segment of BBAs often includes the PCoA at the contralateral side and the AChAs are nearby. From the technical standpoint, even simple exposure of these lesions may be dangerous.\textsuperscript{1,5,9,10,13}

We also found that endosaccular coil embolization, a typical endovascular aneurysm treatment, cannot be recommended in cases of ICA BBAs. Even when their angiographically documented configurations indicate typical saccular aneurysm features, they cannot be recommended because the risk of procedural rupture is too high. Moreover, neither permanent nor short-term efficacy can be expected. In all of our patients who underwent endosaccular embolization, we observed regrowth and/or rebleeding of the lesion. In one case, rebleeding occurred 6 days after successful endosaccular coil embolization, and in this patient angiography revealed that the aneurysm regrew to twice its previous size.

Theoretically, the trapping of a vascular hemorrhagic segment may be a definite means of preventing recurrent bleeding as long as there is no risk of ischemia after sacrifice of the ruptured segment. In the neurosurgical field, trapping is commonly applied to the treatment of ruptured vertebral artery dissecting aneurysms when the contralateral vertebral artery is intact.\textsuperscript{1,2} The basic reason for trapping is that true lesions in vertebral artery dissections are not confined to aneurysm dilation but include the arterial segment itself. Although the pathogenesis of ICA BBAs remains uncertain, it is evident that these lesions are different from saccular aneurysms—that is, they have characteristics of pseudoaneurysms.\textsuperscript{1,5,9,10,13} As our series shows, treatment that focuses only on saccular dilation portions cannot prevent further bleeding or recurrence. We believe that trapping of the ICA segment and the aneurysm is a proper treatment when the collateral circulation is intact. We performed ICA trapping in patients in whom the BOT was tolerated, and this treatment resulted in an excellent outcome without regrowth or rehemorrhage of the aneurysm.

**Endovascular ICA Trapping**

Technically, the endovascular occlusion of focal ICA segments is not easy. Unlike aneurysm clips, the precise and stable placement of endovascular devices such as coils and balloons is difficult. As we found, the distance between AChAs and the lesion segment to be trapped may be less than 1 to 2 mm. In addition, compact occlusion is also essential to prevent recurrence. For compact and precise trapping, balloons may be preferred to coils, but ICA BBAs are very fragile and usually do not have uniform configurations that accommodate balloons. Balloon inflation at these segments would be highly dangerous. While avoiding these dangerous lesions, proximal ICA occlusion by endovascular or surgical means may be a treatment option, but this does not eliminate rebleeding risk posed by retrograde flow.

We believe that our trapping technique that involves two microcatheters and a balloon is both unique and effective. It is performed through a single 8-F guiding catheter. Coil deployment through two microcatheters (one for frame making and one for compact packing) allows precise coil placement and compact coil packing. Moreover, by blocking the proximal ICA flow during coil packing, a nondetachable balloon at the proximal ICA allows secure and stable coil placement, and because the balloon used for the BOT is actually reused, there is no additional cost. Using this technique, we were able to occlude ICA segments precisely and compactly, as planned. Compared with surgical trapping\textsuperscript{10} or combined endovascular–surgical trapping,\textsuperscript{6} our endovascular trapping technique has the following advantages: it can be performed immediately after angiography and it poses fewer risks associated with surgery such as premature rupture.

For ICA trapping, the cardinal arteries such as the OphA, PCoA, and AChA require consideration. As mentioned earlier, because of their locations, PCoAs should often be occluded together with the lesions. Therefore, if a fetal-type or large PCoA is included in the lesion segment, other therapeutic strategies should first be considered.

Regarding the OphA, if an orbital choroidal blush or OphA via the ECA collateral circulation is visualized on angiograms during the BOT, the ICA is trapped with the origin of the OphA. Additionally, the proximal ICA should...
also be occluded with detachable balloons or coils to achieve long-term stable occlusion. If no ophthalmic collateral vessel is visualized, however, short segmental ICA trapping should be undertaken while saving the origin of the OphA. Accordingly, the proximal ICA cannot be occluded to preserve ophthalmic flow, and therefore, close follow-up monitoring is essential to detect recanalization of the trapped ICA segment. We have one such case in which recanalization was detected at the 6-month follow-up examination.

In practice, the AChA is the most important branch to be saved. The origin of the AChA is often too close to the lesion to be occluded. In our series, this distance ranged from 0 to 4 mm. We found that the trapping technique involving the two microcatheters and proximal balloon occlusion is helpful in achieving precise coil placement and compact packing while saving the artery. If the AChA originates from the lesion segment, however, alternative treatments such as clip placement or wrapping should be contemplated.

**Balloon Occlusion Test**

We believe that the preoperative assessment of collateral potential is essential when managing ICA BBAs, even prior to surgical treatment. During surgery, ICA trapping may become the only option. Ogawa et al. have described five cases that involved surgical trapping. In three cases, the trapping was done incidentally to treat intraoperative bleeding. The authors did not mention a preoperative ICA occlusion test. Among the three cases, two patients experienced a good recovery, whereas the other died of cerebral infarction.

Our BOT protocol was designed to be performed practically. If patients are alert, it is conducted after the application of a local anesthetic. No perfusion study such as SPECT was performed because it takes time and patients have to be moved for the examination. In the present study, the ICAs were trapped according to angiographic and clinical findings. When patients were unconscious or uncooperative, the BOT was performed after general anesthesia was induced. In these cases, of course, the decisions were made only on the basis of angiographic findings.

**Risk of Cerebral Ischemia After ICA Trapping**

The major postoperative problems associated with ICA trapping are the short- and long-term risks of cerebral ischemia. During the early period, vasospasm is a major concern, and thus to reduce risks, aggressive medical management to prevent vasospasm should be undertaken immediately after ICA occlusion. After ICA trapping, endovascular treatment for vasospasm is also technically challenging. Because the ipsilateral carotid artery is occluded, the microcatheter should be placed through the contralateral ICA. If the ACoA is large enough, the microcatheter tip for intraarterial infusion of antispasm drugs can be placed at the A1 or M segment across the ACoA, but if its diameter is too small to take the microcatheter, crossing the ACoA may result in further flow reduction. In such cases, the microcatheter should be placed at the contralateral A1 segment, but at this location most infused drugs go to the distal anterior cerebral artery and not to the contralateral A1 segment and middle cerebral artery (the locus of the vasospasm), and therefore, vasodilation may not be properly achieved.

In our series of patients, symptomatic vasospasm developed in approximately 50% of our cases. It was more severe, diffuse, and frequent in cases involving repeated bleeding that developed during the waiting period without definitive treatment or after incomplete treatment. If vasospasm developed, it was often impossible to apply any definitive treatment, such as ICA trapping or combined bypass–trapping, because of the high risk of ischemia. Our experience suggests that ICA BBAs should be treated with definitive methods at the earliest opportunity because an aggravated clinical condition caused by rebleeding and vasospasm can deprive one of a chance to save the patient.

Long-term cerebral ischemic risk is one of the major potential problems of ICA occlusion. Many authors have concluded that there is an increased risk for delayed ipsilateral cerebral ischemia after ICA occlusion in aneurysm treatment. For the early detection and preventive treatment of cerebral perfusion deficits, regular follow-up examination with perfusion studies, such as SPECT, are mandatory. De novo aneurysm formation, especially an ACoA aneurysm caused by hemodynamic stress after carotid artery occlusion is also a potential problem, and for early detection regular follow-up magnetic resonance angiography is also recommended.

**Alternative Treatments**

Endovascular ICA trapping cannot be indicated in all patients with ICA BBAs. Failure of the occlusion test may be a definitive contraindication. Depending on clinical situations, other conditions may be contraindications—that is, when a cardinal branch such as the AChA, PCoA, or OphA is included in the lesion segment or when severe vasospasm has already occurred, implying that the sacrifice of one ICA is associated with a high risk of cerebral ischemia. For these patients, alternative treatment strategies should be considered, including clip placement, wrapping, bypass surgery and trapping, and endosaccular coil embolization. We believe, however, that the trapping of the lesion segments, regardless of trapping techniques (surgical, endovascular, or combined), would be the only method of preventing ICA BBA recurrence during a long-term period. Therefore, close follow-up monitoring is mandatory after alternative treatments. According to clinical situations, preparations for ICA trapping, such as bypass surgery, should be undertaken during follow up because trapping may be eventually required.

**Conclusions**

Based on our results, we suggest the following. First, endosaccular coil embolization cannot be the definitive treatment for ICA BBAs; we observed regrowth/rebleeding in all cases. Moreover, the duration of regrowth/rebleeding was short, possibly less than 1 week. Stent or balloon assistance does not appear to prevent regrowth. Second, ICA trapping including the lesion segment should be considered a first option for definitive treatment if the BOT is successful in the patient. If this is chosen, we recommend endovascular trapping rather than surgery because of its convenience, rapidity, and most importantly its safety. Third,
practically speaking, our technique of using two microcatheters and balloons could be found helpful for the precise, stable, and compact segmental occlusion of lesions. Fourth, when the BOT is not tolerated, alternative treatments should be considered including surgical, endovascular, and combined treatments. Among them, endoscopic coil embolization may prevent rebleeding for a while, but only for a short period. Therefore, during this period, other definitive treatments should be prepared. Finally, after ICA trapping, aggressive vasospasm prophylaxis is required. Long-term follow-up monitoring of cerebral perfusion status is also mandatory.

References


Manuscript submitted April 4, 2006. Accepted September 29, 2006. This study was supported by a grant of the Korea Health 21 R&D Project, Ministry of Health & Welfare, Republic of Korea (grant no. A06-0171-B51004-06N1-00040B).

Address reprint requests to: O-Ki Kwon, M.D., Department of Neurosurgery, Seoul National University Bundang Hospital, 300 Gumi-dong, Bundang-gu, Seongnam-si, Gyeonggi-do, 463-707, Republic of Korea. email: kwonoki@korea.com.