Prevention of Retrograde Blood Flow Into Large or Giant Internal Carotid Artery Aneurysms by Endovascular Coil Embolization with High-Flow Bypass: Surgical Technique and Long-Term Results

Nori Nakajima1, Shinji Nagahiro1, Junichiro Satomi1, Yoshiteru Tada1, Kohei Nakajima2, Shu Sogabe1, Mami Hanaoka3, Shunji Matsubara3, Masaaki Uno2, Koichi Satoh2

BACKGROUND: Recanalization has been reported in large or giant aneurysms of the internal carotid artery (ICA) addressed by high-flow bypass and endovascular treatment. Aneurysmal recanalization may be attributable to retrograde blood flow into the aneurysm through the ICA branches, such as the ophthalmic artery or the meningohypophyseal trunk, or through the surgically created bypass. We modified the endovascular treatment of aneurysms to prevent retrograde flow and evaluated the long-term efficacy of our method.

METHODS: We used a hybrid operative/endovascular technique to treat 5 patients with large or giant aneurysms arising from the C2—C4 segment of the ICA who presented with visual symptoms due to the mass effect of the aneurysm. To prevent retrograde flow into the aneurysm, our modified endovascular treatment involves coil embolization of the aneurysmal orifice and the ICA, including the origin of the ophthalmic artery and meningohypophyseal trunk, and placement of a high-flow bypass using a radial artery graft.

RESULTS: During the 5- to 12-year follow-up period, 4 aneurysms disappeared, and the other decreased in size. There were no subarachnoid hemorrhages. All bypass grafts remained patent. Visual preservation was achieved in 2 patients; 1 patient manifested visual improvement. Although 2 patients experienced transient neurological deficits we encountered no permanent complications in this series. The final modified Rankin scale of the 5 patients was 0 or 1.

CONCLUSIONS: Prevention of retrograde flow into the aneurysm by coil embolization with high-flow bypass is a safe and effective method. It prevents the recanalization of large or giant ICA aneurysms.

Key words
- Coil embolization
- Giant aneurysm
- High-flow bypass
- Internal carotid artery
- Retrograde blood flow

Abbreviations and Acronyms
BTO: Balloon test occlusion
CCA: Common carotid artery
ECA: Extracranial artery
EC—IC: Extracranial-to-intracranial
ICA: Internal carotid artery
ISUIA: International Study of Unruptured Intracranial Aneurysms
M2: Middle cerebral artery second portion
MCA: Middle cerebral artery
MHT: Meningohypophyseal trunk
MRA: Magnetic resonance angiography
MRI: Magnetic resonance imaging
OphA: Ophthalmic artery
RA: Radial artery
STA: Superficial temporal artery

INTRODUCTION
Various treatment options have been reported for patients with large or giant complex aneurysms arising from the C2—C4 segment of the internal carotid artery (ICA). However, because of their large size, difficult location, wide atheromatous neck, and calcified dome, some aneurysms are unclippable and the intracranial approach is contraindicated. Endovascular techniques to address cerebral aneurysms are available and stent-assisted coil embolization and special stents including covered and flow-diverting stents, such as pipeline stents, are useful for treating giant ICA aneurysms [5, 21]. Becske et al. [5] showed that at 1-year follow-up, 87% of their large or giant ICA aneurysms were completely occluded. However, unstable scaffolding, intraparenchymal hemorrhage unrelated to rupture of the target aneurysms, and thromboembolic complications requiring prolonged dual antiplatelet therapy remain an unsolved problem [20]. Although ligation of the cervical ICA with or without extracranial-to-intracranial (EC—IC) bypass is available to address unclippable aneurysms, their recanalization and rupture have been reported [10, 12, 13, 15, 17]. Proximal ICA occlusion does not exclude aneurysms from the flow stream and retrograde flow into the aneurysm may be related to aneurysmal recanalization. There are two collateral pathways into aneurysms, the intrinsic collateral routes between the extracranial artery (ECA) and the ICA through ICA branches, such as the ophthalmic artery (OphA) or the meningohypophyseal trunk (MHT), and retrograde flow from the surgically created bypass graft.

To avoid recanalization due to retrograde flow into the aneurysms, in our 5 patients we performed endovascular coil occlusion of the aneurysmal orifice and the ICA, including the origin of the OphA and MHT. We placed a bypass (radial artery [RA]) to second portion [M2] of the middle cerebral artery or the meningohypophyseal trunk, and placed the ICA through the surgically created bypass graft.
artery) before endovascular coil occlusion. We report our operative techniques and the long-term outcomes in these 5 patients.

METHODS

Patients
Between January 1996 and June 2011, 18 consecutive patients with large or giant ICA aneurysms were treated in our department. The treatment strategy for each patient was decided by neurovascular surgeons and endovascular specialists. Of the 18 patients, 5 underwent coil embolization of the aneurysmal orifice and the ICA including the origin of the OphA and MHT and placement of a high-flow bypass using an RA graft.

The 5 patients presented with large or giant ICA aneurysms (C3–C4 cavernous aneurysms, n = 3; C2–C3 paracranial aneurysms, n = 2) and dysfunction of cranial nerve II (n = 2), III (n = 2), and/or VI (n = 3) due to compression by the aneurysms. One patient (case 2) had a past history of subarachnoid hemorrhage due to rupture of a contralateral ICA aneurysm that had been clipped earlier. The aneurysms were giant in 4 patients and large in 1; their sizes ranged from 15–43 mm.

All patients underwent ICA balloon test occlusion (BTO) to identify those at risk for acute ischemia related to the temporary occlusion of the middle cerebral artery (MCA) during the high-flow bypass procedure. Although all patients were BTO-tolerant, had our series including BTO-intolerant patients, we would have placed a double-insurance bypass (i.e., the superficial temporal artery [STA]–MCA bypass before the introduction of the high-flow bypass to reduce the risk of ischemic complications) (9). A high-flow bypass using an RA graft was placed in all patients to prevent hemodynamic ischemia in the ipsilateral hemisphere and the development of de novo aneurysms.

Surgical Technique
Schematic drawings of our operative techniques and steps are shown in Figures 1 and 2. Neurophysiological monitoring to identify possible cerebral ischemic damage during temporary clipping included somatosensory-evoked and motor-evoked potentials. Craniotomy, exposure of the carotid artery, and harvesting of the RA were performed simultaneously by 3 teams of neurosurgeons. A 15- to 17-cm long segment of the RA was harvested between the wrist and the elbow. Before use, the harvested graft was distended in heparinized saline to avoid twisting and kinking. The common carotid artery (CCA), ICA, and ECA were exposed by making a cervical incision along the anterior aspect of the sternocleidomastoid muscle. Pterional craniotomy was performed with wide dissection of the Sylvian fissure.

Next, the harvested RA graft was passed from the cervical- to the cranial tomentum side through an 18-gauge chest tube in a tunnel created under the mandible and zygomatic arch (Figure 1). Distal anastomosis between the distal end of the RA and M2 was performed first in an end-to-side fashion with 9-0 monofilament nylon sutures. The proximal anastomosis was placed in an end-to-side manner to the ECA using 8-0 nylon continuous sutures. To retain patency of the bypass graft and to eliminate turbulent flow in the aneurysm, we then immediately proceeded to the endovascular procedure using a portable digital subtraction angiography instrument in the operating room. The exposed CCA was punctured directly and a 7F sheath ([Brite tip], Cordis, Miami, Florida, USA) was inserted from the CCA to the ICA (Figure 1). We injected radiopaque dye through the 7F sheath after temporary clipping of the cervical ICA to confirm bypass patency and to visualize the OphA and the aneurysmal supply by collaterals from the ECA (Figure 2A). Then we inserted a 7F balloon catheter ([PATLIVE], Clinical Supply, Gifu, Japan) into the ICA through the sheath. A microcatheter was inserted into the aneurysm under fluoroscopic guidance. After balloon dilation of the ICA proximal to the aneurysm, we then proceeded to the endovascular procedure.
prevent distal migration of coils used for subsequent embolization. After identifying the aneurysmal orifice, we used several coils for tight embolization of the aneurysmal orifice and the ICA including the origin of the OphA and the MHT (Figures 1 and 2). The cervical ICA was ligated with a silk thread in all patients. During the endovascular procedure, 3000 U of heparin were administered intravenously, followed by 1000 U at hourly intervals. Final external carotid angiography was performed to confirm bypass patency and no visualization of the aneurysm. The total operative time ranged from 6–8 hours.

RESULTS
A summary of our treatment outcomes is presented in Table 1. All patients tolerated BTO without developing neurological deficits. There were no serious perioperative complications. One patient (case 2) suffered transient mild hemiparesis and another (case 4) manifested aphasia, but their symptoms resolved completely by the first postoperative day. In 2 patients postoperative diffusion-weighted imaging showed a small high-intensity spot in the ipsilateral cerebral hemisphere; follow-up T₁-weighted imaging detected no infarction. In case 2 we observed transient symptom aggravation 2 weeks after surgery. This was probably due to abrupt aneurysmal thrombosis that resulted in the transient expansion of the aneurysm mass seen on magnetic resonance imaging (MRI) scans. The preoperative symptoms gradually disappeared completely in 2 patients (cases 1 and 4) and improved in 1 (case 2). The visual symptoms were unchanged in 2 patients (cases 3 and 5); preoperative damage of their cranial nerves appeared to have been severe.

The mean follow-up period was 7 years (range, 5–12 years). The grafts remained patent in all patients. Cerebral angiography or magnetic resonance angiography (MRA) confirmed aneurysmal obliteration in all patients and no de novo aneurysms were detected. The aneurysmal mass disappeared almost completely in 4 patients and was markedly smaller in the other (Table 1). In all patients the coils placed in the aneurysm looked like small residual lesions. All had good outcomes; the modified Rankin scale was 0 in 2 patients and 1 in 3 patients with visual symptoms.

Illustrative Cases
Case 1. A 43-year-old woman presented with a 2-month history of headache and double vision. She manifested mild right abducens nerve paresis. MRI showed a mass lesion on the inner side of the temporal lobe (Figure 3A). Angiography revealed a giant aneurysm at the cavernous segment of the right ICA (Figure 3B).

We placed an ECA–M2 bypass with an RA graft on the right side. Selective angiography of the ECA after temporary occlusion of the ICA showed patency of the RA graft. The OphA and ICA were supplied by collaterals from the ECA in the early phase (Figure 3C). Filling of the aneurysm lumen through the surgically created bypass graft and the OphA was confirmed in the late phase (Figure 3D). To cut off the aneurysm from any retrograde blood flow we performed coil embolization of the aneurysmal orifice and the ICA including the origin of the OphA and the MHT (Figure 3E). The fundus of the aneurysm was loosely packed with coils to prevent distal coil migration. Angiography confirmed no filling of the aneurysm and patency of the bypass graft. The OphA was fed by ECA networks (Figure 3F).

Her postoperative course was uneventful and her preoperative symptoms subsided gradually. Angiography of the ECA performed 7 days after the operation revealed no filling of the aneurysm and patency of the bypass graft (Figure 3G). On T₁-weighted images acquired 5 years after the operation there was complete disappearance of the aneurysmal mass. Coils placed in the aneurysm were discernible (Figure 3H). MRA confirmed bypass patency (Figure 3I) and the patient continues to do well with no neurological deficits.

Case 4. A 98-year-old woman presented with a 6-month history of visual disturbance in her left eye; visual acuity on the left was markedly decreased (0.05). MRI, MRA, and angiography showed a giant aneurysm arising at the C2–C3 segment of the left ICA (Figures 4A–C). Endovascular coiling occlusion of the aneurysmal orifice and the ICA including the origin of the OphA and the MHT and placement of a high-flow bypass was as in case 1. Figure 4D is a preembolization angiogram. The aneurysmal orifice and the ICA including the origin of the OphA and the MHT were tightly occluded with coils (Figure 4E). Subsequent intraoperative selective angiography of the ECA revealed a patent bypass (Figure 4F), late filling of the OphA by ECA networks, and no filling of the aneurysm (Figure 4G). Her postoperative transient aphasia resolved quickly and her visual disturbance...
improved gradually and normalized several months after the operation. MRI and MRA studies acquired 5 years after surgery demonstrated complete disappearance of the giant aneurysm and patency of the RA graft (Figures 4H, I). Her modified Rankin scale was 0, and her vision was normal 8 years after surgery.

**DISCUSSION**

We treated 5 patients with large or giant ICA aneurysms at C2–C4 by dense coil occlusion of the aneurysmal orifice and the ICA including the origin of the OphA and the MHT to prevent retrograde flow into the aneurysms. All patients received a high-flow bypass. The long-term treatment outcomes were excellent and surprisingly, 4 of 5 aneurysms almost completely disappeared.

Ligation of the cervical ICA with or without EC–IC bypass is the classic treatment for unclippable ICA aneurysms [11, 24]. Although ligation is easier than our endovascular approach and most aneurysms can be expected to be thrombosed after cervical ICA ligation, recanalization and rupture after EC–IC bypass combined with ICA occlusion have been documented [10, 12, 13, 15, 17]. Matsuda et al. [15] reported a patient with an unruptured giant aneurysm of the ICA who suffered an aneurysmal rupture 34 days after ICA occlusion and STA–MCA bypass. This suggests that immediate complete exclusion of aneurysms from the circulation is preferable to proximal ICA occlusion. We and other investigators [1, 20] posit that postoperative recanalization and rupture are due to retrograde filling of the aneurysm by the bypass graft or by ICA branches in the ECA network such as the OphA or the MHT (Figure 2). In our case 1, intraoperative selective angiography of the ECA obtained after temporary occlusion of the cervical ICA revealed late filling of the aneurysm through the surgically created bypass and the OphA. Therefore we performed dense coil occlusion of the aneurysmal orifice and the ICA including the origin of the OphA and the MHT to eliminate retrograde flow into the aneurysm. This cut off the blood flow into the aneurysm and resulted in its transformation into a blind sac and complete thrombosis.

Surgical or endovascular trapping may also be considered to stop the flow into ICA aneurysms. However, distal clipping can be difficult if there is not enough space for clip

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**Table 1. Characteristics of Patients at Admission and Surgical Results**

<table>
<thead>
<tr>
<th>Case Number</th>
<th>Age/Sex</th>
<th>Side and Location</th>
<th>Size (mm)</th>
<th>Symptoms</th>
<th>Perioperative Complications</th>
<th>Follow-up (years)</th>
<th>mRS</th>
<th>Postoperative Visual Symptoms</th>
<th>Perioperative Visual Symptoms</th>
<th>Postoperative Bypass Graft</th>
<th>Postoperative Aneurysm Filling Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40</td>
<td>Rt. C3–C4, cavernous</td>
<td>43</td>
<td>Double vision (VII)</td>
<td>No</td>
<td>7</td>
<td>Disappeared</td>
<td>None</td>
<td>Disappeared</td>
<td>No</td>
<td>Disappeared</td>
</tr>
<tr>
<td>2</td>
<td>62</td>
<td>Lt. C3-C4, cavernous</td>
<td>25</td>
<td>Double vision (II, VI)</td>
<td>Transient worsening</td>
<td>5</td>
<td>Disappeared</td>
<td>Patient</td>
<td>Disappeared</td>
<td>No</td>
<td>Disappeared</td>
</tr>
<tr>
<td>3</td>
<td>68</td>
<td>Lt. C3–C4, cavernous</td>
<td>15</td>
<td>Double vision (II, VII)</td>
<td>None</td>
<td>12</td>
<td>Disappeared</td>
<td>Patient</td>
<td>Disappeared</td>
<td>No</td>
<td>Disappeared</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>Lt. C2–C3, petrosal</td>
<td>29</td>
<td>Visual disturbance (III)</td>
<td>Transient aphasia</td>
<td>8</td>
<td>Disappeared</td>
<td>Patient</td>
<td>Disappeared</td>
<td>No</td>
<td>Disappeared</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>Lt. C2–C3, petrosal</td>
<td>29</td>
<td>Visual loss (V, almost blind)</td>
<td>None</td>
<td>6</td>
<td>Disappeared</td>
<td>Patient</td>
<td>Disappeared</td>
<td>No</td>
<td>Disappeared</td>
</tr>
</tbody>
</table>

Cranial nerve signs: II, optic nerve; III, oculomotor nerve; VI, abducens nerve.

mRS, modified Rankin scale.
insertion between the giant aneurysm and ICA branches distal to the aneurysm. Murayama et al. (16) obtained complete endovascular occlusion of a large ICA aneurysm and its parent artery with the STA–MCA bypass. We, on the other hand, first loosely embolized the aneurysms with a few anchor coils to prevent distal coil migration and to avoid dysfunction of cranial nerves II–VI due to the mass effects exerted by tightly packed coils. Cranial nerve dysfunction improved in 3 of our 5 patients and no patients manifested worse visual symptoms postoperatively. Van Rooij and Sluzewski (22) reported that 31 unruptured large or giant ICA aneurysms with cranial neuropathy were treated with ICA occlusion and that the symptom improvement was as good as in our study.

Ishishita et al. (11), who routinely placed a high-flow bypass in patients with large or giant ICA aneurysms, reported that their procedure was safe with excellent

Figure 3. Case 1. (A) Preoperative T₁-weighted image demonstrated an intracavernous aneurysm. (B) Preoperative internal carotid angiogram (anteroposterior view) demonstrating a giant aneurysm of the right internal carotid artery (ICA). (C–F) Selective angiography of the external carotid artery (ECA) ob-
tained during the operation. The pre-embolization angiogram (lateral view) obtained in the early (C) and late phase (D) after ECA–radial artery (RA)-middle cerebral artery second portion bypass revealed patency of the bypass (thin arrows) and filling of the aneurysmal lumen (thick arrows) and the cavernous portion of the ICA through the RA graft, and the ophthalmic artery (OphA) (arrowheads) by the external networks. The intraoperative angiogram (E) showed loose packing of the fundus of the aneurysm, dense coil embolization of the aneurysmal orifice, and the ICA including the origin of the OphA and the meningohypophyseal trunk (large arrow), and the patent bypass (small arrows). Intraoperative angiography (F) obtained after coil occlusion showed flow in the OphA (arrowheads). (G) Angiogram acquired 7 days after the operation revealed patency of the bypass graft (arrows) and no filling of the aneurysm. (H) A T₁-weighted image obtained 5 years after the operation showed complete disappearance of the intracavernous mass. The inserted coils can be seen (arrow). (I) Magnetic resonance angiography confirmed patency of the bypass graft (arrows).
long-term graft patency. Although all of our patients were BTO-tolerant, we placed an EC-IC bypass to prevent ischemic events and the development of de novo aneurysms. Although BTO has been reported to be useful for assessing the tolerance for ICA sacrifice, even in BTO-tolerant patients, the early ischemic rate was 2.0% – 22.7% and up to 1.9% developed late ischemic complications (8, 14). Furthermore, de novo aneurysms developed on the contralateral side due to increased hemodynamic stress after ICA occlusion without distal revascularization (3, 4, 6). High-flow EC–IC bypass surgery using an RA graft can alleviate hemodynamic ischemia after ICA occlusion and future hemodynamic stress on the contralateral side. Our long-term follow-up revealed patency of the bypass graft and no de novo aneurysm formation. The high-flow bypass prevented hemodynamic ischemic events, not only in the perioperative period, but also in the long-term period.

Our approach has some risks and disadvantages. Although occlusion of the origin of the OphA may result in retinal ischemia and vision worsening, it usually does not elicit visual disturbances because the blood supply from the collaterals of the

Figure 4. Case 4. (A, B) Preoperative T2-weighted image (A) and magnetic resonance angiography (MRA) (B) showed a giant aneurysm in the chiasmal cistern. (C) Left carotid angiogram (anteroposterior view) demonstrated a giant aneurysm arising from the paraclinoid segment of the left internal carotid artery (ICA) (C2 portion). (D) Intraoperative internal carotid angiogram (lateral view) obtained after external carotid artery (ECA)- radial artery (RA)-middle cerebral artery second portion (M2) bypass showed filling of the ICA giant aneurysm. (E) Note the loose packing of the aneurysm and dense packing of the aneurysmal orifice and the ICA including the origin of the ophthalmic artery (OphA) and the meningo-hypophyseal trunk. (F, G) Selective angiography of the ECA performed after ECA-RA-M2 bypass and endovascular embolization in the early (F) and late phase (G). In the early phase (F, lateral view) the angiogram showed occlusion of the aneurysm and patency of the RA graft (arrow). In the late phase (G) the OphA was supplied by ECA networks (arrowheads). (H, I) Postoperative T2-weighted image (H) and magnetic resonance angiography (I) obtained 5 years after the operation showed disappearance of the aneurysm and patency of the bypass graft (arrow).
ECA is sufficient and the central retinal artery originates from the OphA after passing through the optic canal (7, 19). Ocular ischemia due to thromboembolism is more detrimental to visual acuity than occlusion of the OphA origin. Intraoperative angiography showed blood flow in the OphA through these collaterals and none of our patients suffered postoperative vision aggravation. In fact, preoperative visual disturbances were markedly improved in 2 patients although visual loss cannot be ruled out after occlusion of the OphA. Occlusion tests of the origin of the OphA and the visual-evoked potentials are useful for the selection of treatment strategies to preserve vision (2, 18).

Distal embolism may arise if aneurysmal thrombolysis occurs abruptly in the acute postoperative phase. In fact, 2 of our patients suffered transient postoperative hemiparesis and aphasia. Their symptoms subsequently resolved completely and no cerebral infarcts were observed despite the presence of small high-intensity spots on postoperative diffusion-weighted imaging studies. Appropriate intraoperative anticoagulation and postoperative antiplatelet therapy should be considered to prevent embolic complications in the perioperative period. Abrupt aneurysmal thrombolysis may also produce a temporary increase in the size of the aneurysm. One of our patients (case 2) manifested transient oculomotor palsy 2 weeks after surgery. It subsided gradually in the course of aneurysmal shrinkage.

The International Study of Unruptured Intracranial Aneurysms (ISUIA) reported that the clipping of large or giant ICA aneurysms resulted in a major morbidity rate of more than 20% (23). Although our study population was small, we achieved aneurysm obliteration and graft patency in all 5 patients, none of whom manifested permanent neurological complications in the course of long-term follow-up.

CONCLUSIONS

With meticulous attention to the surgical techniques, retrograde flow into the aneurysm can be prevented by coil embolization and the placement of a high-flow bypass. We suggest that our technique is a safe and effective treatment in patients with large or giant ICA aneurysms. The endovascular technique and the placement of a high-flow bypass involve fewer surgical manipulations for completing aneurysmal occlusion.

REFERENCES


Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

N. Nakajima and S. Nagahiro contributed equally to the project.

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