Procedural Safety and Potential Vascular Complication of Endovascular Recanalization for Chronic Cervical Internal Carotid Artery Occlusion

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Background—Patients with chronic cervical internal carotid artery occlusion (ICAO) and cerebral ischemia may benefit from revascularization. The feasibility of endovascular recanalization for chronic ICAO has been reported recently, but its safety is still unproven. We report the follow-up results of 54 chronic ICAO patients who underwent endovascular recanalization, focusing on potential vascular complications and corresponding management.

Methods and Results—Endovascular recanalization for chronic ICAO was attempted in 54 consecutive patients (48 men; 69.2±9.8 years old) with either recurrent neurological deficit or objective ipsilateral hemisphere ischemia. Mean duration from occlusion documentation to the procedure was 237±327 days (range, 56 to 1424 days). Adverse events while in the hospital and during the 3-month follow-up were recorded. Successful recanalization was achieved in 35 of 54 patients (65%). Three-month cumulative stroke and death rate was 4% (2 of 54), including 1 in-hospital fatal nonipsilateral stroke and 1 in-hospital minor ipsilateral stroke secondary to systemic hypotension. Vascular complications developed in 3 of 54 patients (6%), including 1 late pseudoaneurysm formation 3 months after recanalization, 1 immediate carotid-cavernous fistula after recanalization, and 1 minor extravasation at carotid bifurcation after failed recanalization. However, no clinical sequela was noted with close follow-up and adequate management.

Conclusion—Certain immediate or delayed vascular complications may develop during or after the endovascular recanalization for chronic ICAO. Although periprocedural death and stroke rate is limited in our study, further study combining neuroimaging tools and cognitive function evaluation is mandatory to assess its utility and appropriateness in patients with chronic ICAO. (Circ Cardiovasc Intervent. 2008;1:119-125.)

Key Words: angioplasty ■ carotid arteries ■ occlusion ■ endovascular recanalization ■ stroke

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Endovascular recanalization for arterial occlusion is technically challenging. With the advance of devices and skills, however, arterial occlusions in various arterial territories can now be treated with high success rates.7-10 The feasibility of endovascular recanalization for chronic ICAO has been demonstrated recently,11 but potential complications including vascular trauma and thromboembolism may result in serious adverse events. We report the results of 54 consecutive patients who underwent endovascular recanalization for chronic ICAO, focusing on clinical events and vascular complications.
Table 1. Demographics (N=54)  

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>48 (89)</td>
</tr>
<tr>
<td>Age, years</td>
<td>69.2±9.8</td>
</tr>
<tr>
<td>Hypertension</td>
<td>43 (80)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>19 (35)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>29 (54)</td>
</tr>
<tr>
<td>Smoking</td>
<td>28 (52)</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Prior neck radiotherapy</td>
<td>3 (6)</td>
</tr>
<tr>
<td>Prior ipsilateral stroke</td>
<td>35 (65)</td>
</tr>
<tr>
<td>Ipsilateral transient ischemic attack</td>
<td>15 (28)</td>
</tr>
<tr>
<td>Amaurosis fugax</td>
<td>4 (7)</td>
</tr>
<tr>
<td>Contralateral ICA stenosis &gt;50%</td>
<td>19 (35)</td>
</tr>
<tr>
<td>Progression or recurrence of neurologic deficit after ICAO documentation</td>
<td>37 (69)</td>
</tr>
<tr>
<td>Hemispheric ischemia by perfusion CT</td>
<td>17 (31)</td>
</tr>
<tr>
<td>NASCET symptomatic at procedure</td>
<td>47 (87)</td>
</tr>
<tr>
<td>Duration from ICAO documentation to procedure, days</td>
<td>237±327</td>
</tr>
</tbody>
</table>

NASCET indicates North American Symptomatic Carotid Endarterectomy Trial.

Methods

Patients

All patients were ≥18 years of age. ICAO was documented by using neck duplex ultrasound study, computed tomography (CT) angiography, or magnetic resonance angiography. After documentation of the occlusion, patients were followed, at least for 2 months, for recurrence of ipsilateral neurological deficit or objective ipsilateral hemisphere ischemia by perfusion CT with Diamox stress. From October 2002 to August 2007, endovascular intervention was attempted in 54 patients (48 males; age, 69.2±9.8 years; range, 48 to 86 years) with unilateral chronic ICAO with recurrent symptoms or objective ischemia. Mean duration from occlusion documentation to the procedure was 237±327 days (range, 56 to 1424 days). The clinical characteristics of the patients are summarized in Table 1. Forty-seven patients (87%) were found to be symptomatic according to North American Symptomatic Carotid Endarterectomy Trial criteria at the time of the procedure. Three patients (6%) had a history of neck radiotherapy for nasopharyngeal cancer. Nineteen patients (35%) had contralateral internal carotid artery (ICA) stenosis >50%, and 12 of these underwent carotid artery stenting according to the established indications before the index procedure. According to criteria of the North American Symptomatic Carotid Endarterectomy Trial, 35 patients (65%) were at high surgical risk, and 37 patients (69%) experienced recurrent ipsilateral hemispheric neurological symptoms after the documentation of the occlusion, and objective ischemia was demonstrated by perfusion CT in 17 patients (31%).

Procedure

Diagnostic cerebral angiography was performed with femoral route. ICAO was defined as (1) the discontinuation of ICA lumen ≥5 mm in length, (2) Thrombolysis in Myocardial Infarction (TIMI) grade 0 antegrade flow distal to the occlusion, and (3) established collateral filling to the ipsilateral intracranial ICA territory via anterior communicating artery, posterior communicating artery, or ipsilateral ophthalmic artery (OA). About 100 mg of aspirin and 75 mg of clopidogrel per day were given for ≥7 days before the procedure. Heparin bolus was administered to maintain the activated clotting time between 200 and 250 seconds during intervention. Interventional technique was performed as described previously. Wire crossing success was defined as the passage of guidewire across the occluded segment with the tip in distal true lumen confirmed by multiple angiographic projections. Technical success was defined as final residual diameter stenosis ≤20% with TIMI grade 3 distal antegrade flow after intervention. All patients were sent to the intensive care unit for overnight hemodynamic and neurological monitoring, where systolic blood pressure was carefully maintained within 100 to 140 mm Hg. Aspirin and clopidogrel were continued for ≥3 months after successful intervention.

Data Collection and Follow-Up

All clinical, angiographic, and procedural data were retrospectively collected from the medical chart and recorded on standard forms by a physician. Quantitative measurement of angiography was done offline with calibration of the system using the known diameter of the catheters. Diameters of the common carotid artery and cervical ICA and occlusion length were measured after the establishment of antegrade flow in the ICA. Final residual diameter of the stenosis was determined using the method of the North American Symptomatic Carotid Endarterectomy Trial, with the distal nontapering portions of the postintervention ICA after nitroglycerin as the reference segment.

All patients were examined, including the National Institutes of Health Stroke Scale assessment, by an independent neurologist before and 1 day after the intervention. Follow-up clinical and ultrasound examination were scheduled at 1, 3, 6, and 12 months after the intervention. Clinical events, including any stroke, transient ischemic attack, or death, were documented in the chart. Transient ischemic attack was defined as a focal neurological deficit that resolved completely within 24 hours. A minor stroke was defined as a nondisabling deficit that persisted for >24 hours but resolved completely within 1 week. A major stroke was classified as a persistent neurological deficit that lasted >7 days. Myocardial infarction was defined as creatine kinase >2 times higher than the upper limit of normal, with a positive muscle-brain fraction.

Statistical Analysis

Continuous data were presented as mean±SD, and categorical variables were presented in numbers and percentage. Differences of National Institutes of Health Stroke Scale scores among baseline, 1-, and 3-month follow-up were determined by 1-way repeated-measures ANOVA.

Results

Angiographic and Procedural Data

Angiographic characteristics and procedural data are shown in Table 2. Wire crossing was successful in 37 patients (69%).

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TIMI grade 3 antegrade flow was not achieved in 2 cases after predilatation, and further stenting was aborted. Hence, the technical success rate was 65% (35 of 54). Embolic protection devices were used in 73% (27 of 37) after wire crossing, and 2 embolic protection devices were used sequentially in 1 patient because of huge thrombus burden causing full filter and flow obstruction after predilatation. Embolic protection devices were not used in the other 10 cases because of small distal vessel diameter. Two or more stents were needed to cover the occluded segment in 13 of 37 patients (35%), including the 2 cases with wire crossing success but without final TIMI grade 3 flow. External carotid artery was jailed by stent in 34 of 37 patients (92%). In the 35 technical success lesions, the final residual diameter stenosis was 9±7%.

In-Hospital and 3-Month Results

Table 3 shows the cumulative incidence of adverse events during hospitalization and at 3-month follow-up. Four hours after the procedure, 1 patient had fatal nonipsilateral stroke due to an acute basilar artery occlusion with massive posterior infarction after successful recanalization of chronic left ICAO. Emergent intra-arterial thrombolysis failed to achieve reperfusion, and the patient died because of massive brain stem hemorrhage. Another patient experienced minor ipsilateral stroke 2 days after the intervention, which presented as central facial palsy after a hypotension episode in the ward secondary to volume depletion. The symptom lasted for >24 hours but resolved completely before discharge. Except 1 fatal case, there were no new major adverse events during the 3-month follow-up in all 53 patients. Also, there was no statistical difference in the National Institutes of Health Stroke Scale among baseline, 1-, and 3-month follow-up (1.23±2.30, 1.09±2.26, and 1.45±2.61, respectively; P=0.14). In-hospital and 3-month cumulative incidence of stroke and death was 4% (2 of 54). In-stent reocclusion was found in 2 of 35 technically successful cases. Reintervention was not performed because no recurrent symptom could be documented.

Vascular Complication

Case 1: Late Carotid Pseudoaneurysm Formation
A 77-year-old man with chronic right ICAO was documented by carotid duplex with reversed OA flow direction. Angiography confirmed the occlusion (Figure 1A), also revealing collateral filling from ipsilateral OA (Figure 1B). The occlusion was successfully recanalized without any apparent vascular injury (Figure 1C and 1D). The OA flow direction returned to normal (by ultrasound) after the procedure. However, he reported dizziness 3 months after the procedure, and OA flow direction was reversed again. Surprisingly, CT angiography showed no in-stent restenosis but showed a large pseudoaneurysm with significant true lumen compression distal to the stent (Figure 1E). Perfusion CT showed decreased blood flow (Figure 2A) and delayed mean transit time in the right hemisphere (Figure 2B). He was admitted, and a bare-metal stent was deployed across the neck of the pseudoaneurysm (Figure 1F). Follow-up CT angiography 1 month later documented stent patency and significant reduction of the aneurysmal sac (Figure 1G). Perfusion CT also demonstrated normalized blood flow (Figure 2C) and transit time (Figure 2D).

Case 2: Iatrogenic Carotid-Cavernous Fistula
A 75-year-old man with right ICA stenosis and chronic left ICAO documented by magnetic resonance angiography was first treated with right ICA stenting. Perfusion CT 6 months later revealed residual left-hemisphere ischemia, and endovascular intervention for the left chronic ICAO was attempted. After successful wiring across the occluded segment and subsequent small balloon dilatation, a small carotid-cavernous fistula (CCF) was noted in the cavernous portion of distal ICA (Figure 3A), possibly due to wire perforation. There was no ocular symptoms or neurological deficit accompanied, but the procedure was suspended and the patient
was closely monitored. He remained asymptomatic, and brain CT showed no abnormality. Repeat cerebral angiogram obtained 3 days later showed no residual CCF flow, and staged stenting over the recanalized ICA was performed uneventfully (Figure 3B).

Case 3: Cervical Extravasation
Extravasation in proximal cervical left ICA was noted during a wiring attempt for chronic ICAO in a 59-year-old man (Figure 4). Local pain and tenderness were reported without hemodynamic consequence or airway compromise. The procedure was aborted, but no significant neck hematoma or any other clinical event was developed during the following 2 days of hospital stay.

Discussion
The feasibility of endovascular recanalization of chronic ICAO has been shown recently. The present results in our series indicated that, despite the potential of catastrophic vascular complication, the actual periprocedural major adverse event rate was limited. The short-term outcomes and complications compared favorably with those of surgical treatment for chronic ICAO and were similar to those of stenting and endarterectomy for ICA stenosis.

Instead of embolism, compromised cerebral perfusion may be the most important etiology of ipsilateral ischemic events in patients with chronic ICAO. In the Harvard Stroke Registry, 20% (95 of 471) of the stroke patients experienced deterioration after the initial event, and 33% had a large artery occlusion. It was postulated that decreased perfusion or decreased potential for developing collateral most likely caused advanced ischemic symptoms in patients. Grubb et al used positron-emission tomography scanning to establish the importance of hemodynamic factors in predicting outcomes among these patients. Medical management alone may be inadequate, and revascularization may be beneficial in patients with impaired cerebral perfusion due to chronic ICAO. In our series, all patients were enrolled because of recurrent neurological deficit or objective hemisphere ischemia after documentation of the occlusion, and in theory, they should benefit from revascularization.
Once the ICA is occluded, downstream thrombus formation will begin and propagate to the first branch of the ICA with significant collateral inflow, usually the OA. The trailing end of this thrombotic column of blood is a potential source of emboli to the middle cerebral artery.\textsuperscript{20} Therefore, a major concern in recanalizing chronic ICAO is the possibility of causing distal embolism from the stump. However, in a small series of acute carotid occlusion reported by Spearman et al\textsuperscript{21}, the passage of catheters through fresh thrombi and revascularization of the occlusion did not cause apparent embolism. In chronic ICAO, the thrombotic content in the stump is further organized, and the possibility of releasing embolic debris during device manipulation should be minimal. In addition, the antegrade flow after guidewire crossing and gentle undersized predilatation is usually sluggish, with low risk of carrying emboli downstream. Deployment of a distal embolic protection device will therefore protect the subsequent stenting and postdilatation, which are actually the most emboli-prone steps of the procedure. In fact, only 1 minor stroke occurred in our study, which resulted from a hypotensive episode 2 days after the intervention, and there was no periprocedural ischemic event related to embolism.

Hyperperfusion is another concern related to recanalization of chronic ICAO. Spontaneous cerebral hemorrhage after
carotid endarterectomy has been reported in many large studies,22–24 and cerebral hyperperfusion may play an important role. Cerebrovascular autoregulation in regions with chronic ischemia may be defective, and a sudden increase of perfusion after removal of a high-grade ICA stenosis may lead to brain swelling or even hemorrhage. Preoperative perfusion imaging can be used to identify patients at risk for hyperperfusion after CEA,25 and blood pressure control is crucial in preventing hyperperfusion.20–25 In theory, patients with successfully recanalized ICA may be at risk for hyperperfusion, but with strict periprocedural blood pressure control, none of our patients experienced hyperperfusion.

The tapered-tip, stiff, 0.014-inch coronary angioplasty wire should be handled with extreme dexterity to minimize iatrogenic arterial injury. Complete vessel perforation may cause catastrophic bleeding, but even minor intimal injury may lead to dissection or pseudoaneurysm formation. Delayed iatrogenic pseudoaneurysm has been reported in ICA, vertebral artery, and superior mesenteric artery after angiography or endovascular intervention,26,27 but its natural history and optimal management remains undefined. Touze et al28 reported 71 cases of spontaneous carotid dissection, of which aneurysms developed in 35, managed with antiplatelet or anticoagulant therapy. Felber et al29 and Kadkhodayan et al30 both reported endovascular repair of carotid dissection and aneurysm, using stent graft or bare-metal stent. In our study, we managed the single case of delayed pseudoaneurysm with endovascular bare-metal stenting. Conservative medical treatment was considered inadequate, as the aneurysmal sac increased in size, causing true lumen compression, which led to recurrent symptoms and subjective ischemia. Stent graft was not chosen because of the device rigidity and thrombogenicity. A bare-metal stent deployed across the aneurysm inlet eliminated flow into the sac and restored laminar flow to the hemisphere effectively.

CCF has been reported to be associated with dural sinus thrombosis,31 intracranial surgery,32 or trauma,33 but it is rarely seen after endovascular intervention.34 The clinical presentation and prognosis of CCF is dependent on the arteriovenous shunt volume, the direction of venous drainage, and the patency of the draining veins. The chance of spontaneous resolution is high if the CCF drains directly into a dural sinus with normal antegrade flow. Elective intervention is seldom indicated if asymptomatic. In the single case of CCF in our study, the clinical course was benign. The CCF was obliterated spontaneously 3 days later, and left ICAO recanalization was uneventfully completed with staged stent deployment.

In conclusion, potential vascular trauma may develop after endovascular recanalization for chronic ICAO, but the risk of
periprocedural vascular events, including cerebral embolism and hyperperfusion syndrome, was not increased under close post-procedural monitoring and proper management. Future prospective study using objective neuroimaging tools and cognitive function evaluations is mandatory to establish the indication and benefit of this highly demanding endovascular intervention.

Disclosures

None.

References

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Clinical Perspective

Patients with chronic internal carotid artery occlusion and compromised cerebral perfusion may benefit from recanalization. Surgical bypass failed to reduce stroke, and carotid endarterectomy trials have never enrolled this patient population. The feasibility of endovascular recanalization of internal carotid artery occlusion has been shown, with an acceptable overall success rate of ~60% to 70%. Despite the possibility of iatrogenic vascular trauma, as described in this article, the risk of periprocedural clinical events was not increased under close post-procedural monitoring and proper management. Prospective study combining neuroimaging and neurocognitive functional evaluation is mandatory in the future to establish the clinical efficacy and indication of endovascular recanalization of internal carotid artery occlusion.
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