Microcatheter Navigation and Thrombolysis in Acute Symptomatic Cervical Internal Carotid Occlusion

BACKGROUND AND PURPOSE: The treatment of acute stroke distal to an occluded cervical internal carotid artery (ICA) presents a challenge. We report our results of endovascular therapy in 7 patients presenting with acute symptomatic cervical ICA occlusion.

PATIENTS AND TECHNIQUES: Among patients presenting with acute stroke at our institution from June 2001 to June 2005, we retrospectively identified 7 patients who underwent endovascular therapy of acute cervical ICA occlusion. The techniques used for vessel recanalization were analyzed. Postprocedure CT scans were reviewed for hemorrhage. The clinical outcomes were assessed by using the modified Rankin scale (mRS) with good outcomes assigned scores of ≤2.

RESULTS: All 7 patients revealed cervical ICA occlusion, with additional intracranial thrombus in 6 of the 7 patients. In all patients, a guiding catheter was placed in the ipsilateral common carotid artery proximal to the occlusion and a microcatheter advanced through the ICA clot to deliver intra-arterial (IA) tissue plasminogen activator (in 6 patients, the microcatheter was also advanced intracranially for thrombolysis). Successful recanalization of the occluded ICA was achieved in 6 patients. In 3 patients, balloon angioplasty and stent placement of the cervical ICA was also performed. Follow-up CT in 6 patients showed small basal ganglia infarcts in 4, patchy parietal infarcts in one, and frontal lobe hematoma in one patient. At 1 month after the procedure, 5 patients had good clinical outcomes (mRS of 0 in 4 patients and 1 in one patient).

CONCLUSION: Performance of IA thrombolysis by passing a microcatheter through an acutely occluded internal carotid artery may be an effective therapy in acute stroke.

The National Institute of Neurological Disorders and Stroke trial showed the benefit of intravenous (IV) tissue plasminogen activator (tPA) in patients presenting within 3 hours of onset of symptoms of acute stroke. Recent studies have revealed promising results for intra-arterial (IA) thrombolyis especially in patients with significant clot burden that may be refractory to IV tPA. At our institution, patients presenting with symptoms of acute stroke within 3 to 6 hours of onset who have thrombus in the M1 or M2 segments of the middle cerebral artery (MCA) are considered for IA thrombolysis. A unique subset of patients presenting with acute stroke may have an occluded MCA distal to an occluded internal carotid artery (ICA) just distal to the common carotid artery (CCA) bifurcation. Acute ICA occlusions may be embolic, related to atherosclerotic disease, or caused by dissections. These patients may have a poor response to IV tPA. The natural history of patients presenting with signs and symptoms of acute stroke distal to an acutely occluded cervical ICA is poor. There is a 16% to 55% likelihood of death from complications of infarction, 40% to 69% will be severely disabled, and only 2% to 12% will make a good recovery. There are various surgical options in acute symptomatic ICA occlusions, which include carotid endarterectomy (CEA), embolectomy, and surgical bypass. Patients with profound neurologic deficits or large infarcts, however, are at high risk for hemorrhagic transformation after CEA, thus making this a strong exclusion criterion in recent studies evaluating CEA. In addition, the results of urgent surgical recanalization may not be ideal: 26.5% normal neurologic outcome after emergency CEA in a study by Meyer et al.

Catheter navigation through an acutely occluded ICA for the purpose of performing IA thrombolysis has been considered an obstacle and is an issue of debate. Several small case series have described successful IA thrombolysis in the territory of an acutely occluded cervical ICA. Traversing the acutely occluded ICA segment combined with IA tPA may, however, uncover an underlying severe stenosis at the carotid bulb. The management of this residual stenosis or occlusion at the bulb presents an interesting challenge. Some authors have advocated acute angioplasty and stent placement at the time of thrombolysis. We report our results of urgent IA thrombolysis of acute MCA thrombus by navigating a microcatheter through an acutely occluded ICA and discuss the management of the underlying proximal ICA stenosis or occlusion in 7 patients presenting with acute stroke.

Patients and Methods
All patients presenting with symptoms of acute stroke at our institution within 6 hours of onset have a complete neurologic examination and are evaluated with nonenhanced CT scan of the head to rule out hemorrhage. In the absence of intracranial hemorrhage, CT angiogram (CTA) and CT perfusion (CTP) studies are also routinely performed to look for presence of thrombus in the neck/intracranial vasculature and to evaluate tissue at risk, respectively. On CTA, the arterial system from the aortic arch to the circle of Willis is evaluated by using 100 to 120 mL Iohexol (Omnipaque 300; GE Healthcare, Princeton, NJ) injected at a rate of 3 mL/s and a 20- to 22-second delay. CTP is performed covering a 2-cm-thick area at the level of the
basal ganglia chosen by the radiologist present on-site and involves administration of 40 to 50 mL of additional contrast. All patients with contrast allergy, known chronic renal failure, or elevated creatinine are routinely excluded from CTA and CTP studies at our institution. All scans in the present study population were acquired on a multidetector CT (GE Lightspeed Plus, Milwaukee, Wis). The CTP data are analyzed off-line on the GE Advantage Workstation and cerebral blood volume (CBV), cerebral blood flow (CBF), and mean transit time (MTT) maps are generated. Areas of significantly low CBV are presumed to represent dead tissue (the color coding is set up transit time (MTT) maps are generated. Areas of significantly low CBV are presumed to represent dead tissue (the color coding is set up

Note: CTA indicates computed tomographic angiography; CTP, computed tomographic perfusion; NIHSS, National Institutes of Health Stroke Scale; RICA, right internal carotid artery; LMCA, left middle cerebral artery; LMCA, left middle cerebral artery.

Table 1: Clinical and imaging features of 7 patients with occluded internal carotid artery and acute stroke

<table>
<thead>
<tr>
<th>Patient No./Age (y)/Sex</th>
<th>Site of Occlusion (CTA)</th>
<th>Presence of Penumbra (CTP)</th>
<th>NIHSS at Presentation</th>
<th>NIHSS at Follow-up</th>
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<tbody>
<tr>
<td>1/54/M</td>
<td>LICA and LMCA: M1</td>
<td>Yes</td>
<td>22</td>
<td>15 (1 mo)</td>
</tr>
<tr>
<td>2/51/M</td>
<td>LICA and LMCA: M1 and M2</td>
<td>Yes</td>
<td>15</td>
<td>1 (14 mo)</td>
</tr>
<tr>
<td>3/62/F</td>
<td>RICA and RMCA: M1</td>
<td>Yes</td>
<td>21</td>
<td>0 (6 mo)</td>
</tr>
<tr>
<td>4/78/F</td>
<td>LICA and LMCA: M1</td>
<td>Yes</td>
<td>11</td>
<td>0 (5 mo)</td>
</tr>
<tr>
<td>5/76/F</td>
<td>RICA*</td>
<td>Yes*</td>
<td>17</td>
<td>1.1 (40 mo)</td>
</tr>
<tr>
<td>6/28/F</td>
<td>LICA and LMCA: M1</td>
<td>No perfusion study</td>
<td>17</td>
<td>0 (40 mo)</td>
</tr>
<tr>
<td>7/24/M</td>
<td>RICA and RMCA: M1</td>
<td>No perfusion study</td>
<td>12</td>
<td>0 (1 mo)</td>
</tr>
</tbody>
</table>

Note: CTA indicates computed tomographic angiography, CTP, computed tomographic perfusion, NIHSS, National Institutes of Health Stroke Scale, RICA, right internal carotid artery; LICA, left internal carotid artery; RMCA, right middle cerebral artery; LMCA, left middle cerebral artery.

‡ No perfusion imaging performed since the intervention “pre-dated routine perfusion imaging.”
* Seen on MR angiogram.
† Seen on MR perfusion study.
§ No perfusion imaging performed since the intervention “pre-dated routine perfusion imaging.”

Results

The clinical characteristics and imaging features of the 7 patients are summarized in Table 1. There were 3 men and 4 women, with a mean age of 52.4 ± 20.2 years (range, 24–78 years). The mean time of presentation to the hospital in 5 of the 7 patients was 117 minutes (range, 75–150 minutes) within onset of symptoms of acute stroke. Two patients were inpatients and were investigated with MR imaging scans within 120 and 160 minutes of onset of acute stroke symptoms. All patients presented with significant hemiplegia (power, 2/5 or less) at the time of the initial imaging. There was occlusion of the cervical ICA in all 7 patients and also presence of ipsilateral MCA thrombus in 6 of the 7 patients. One patient presented with an acute occlusion of the ICA at its origin 2 hours after
CEA. One patient had bilateral posttraumatic cervical ICA pseudoaneurysms with an acute dissection occluding the right cervical ICA and presence of thrombus in the ipsilateral M1 segment. Percuffusion studies were performed in 5 of the 7 patients (4 on CTP and one on MR perfusion) and tissue at risk was demonstrated in all 5 scans.

In all patients, a guiding catheter was placed in the ipsilateral CCA proximal to the occlusion and a microcatheter advanced through the clot in the ICA to deliver IA tPA (in 6 patients, the microcatheter was advanced to the face of the intracranial thrombus for intracranial thrombolysis). We were able to achieve successful recanalization of the occluded ICA in 6 of the 7 patients. In one patient, after the microcatheter was advanced through the occluded ICA into the face of the thrombus in the MCA and local tPA was infused, there was successful resolution of the MCA thrombus but persistent ipsilateral ICA occlusion (seen on check angiograms performed through the guiding catheter). It was decided to leave the ICA occluded in this patient because of adequate collateral flow across the anterior communicating artery (AcomA) demonstrated on contralateral carotid angiogram. In 3 patients, balloon angioplasty and stent placement of the cervical ICA was also performed after successful thrombolysis. In the patient who had been treated with CEA a few hours earlier, it was decided to stop after successful ICA thrombolysis alone because of concerns about arterial rupture.

Immediate postprocedural CT scans revealed no hemorrhage in 6 patients and a small frontal lobe hematoma in one patient. In CT scans performed before discharge from the hospital in 6 patients, 4 patients showed small basal ganglia infarcts, one showed patchy parietal infarcts, and one showed evolving frontal lobe hematoma.

At 1 month after the procedure, 5 patients had good outcomes (mRS of 0 in 4 patients and 1 in one patient), one had an mRS of 3, and one had an mRS of 4. There were no deaths. Details of the endovascular treatment and outcomes are summarized in Table 2.

Case Illustration 1

A 62-year-old woman presented to the emergency department 2 hours after onset of left complete hemiplegia (power, 0/5), pronounced left facial droop, and marked dysarthria. Noncontrast CT scan of the head showed subtle hypodensification in the right basal ganglia with no hemorrhage. CTA revealed complete occlusion of the right ICA with intracranial thrombus in the distal right M1 segment of the MCA. CTP demonstrated a small area of decreased CBV in the right basal ganglia and a larger area of CBF and prolonged MTT signaling significant penumbra. En route to angiography, 0.6 mg/kg (two thirds the normal IV dose) of tPA was administered intravenously. Diagnostic angiogram of the left ICA revealed good flow across the AcomA but no M1 filling (Fig 1A). The right ICA was completely occluded on right CCA injection. Through a 6F guiding catheter placed in the right distal CCA, a microcatheter was advanced over a 0.014-in microwire through the occluded proximal ICA. Injection through the microcatheter distal to the bifurcation demonstrated thrombus extending to the petrous ICA, although the supraclinoid ICA and A1 segments were patent. The microcatheter was advanced into the right M1 clot, and a total of 9 mg of tPA (diluted 1:1 with heparinized normal saline) was administered into the thrombus for a period of 20 minutes. Repeat injections revealed complete resolution of thrombus and restoration of antegrade flow in the MCA (Fig 1B). Injection into the left ICA revealed cross-filling through the AcomA into the right MCA branches (Fig 1C). The patient started to improve immediately and regained antigravity strength in her left extremities. Her right ICA was left occluded, because we felt that collateral flow across the AcomA was sufficient to perfuse the right brain. Her postprocedure CT demonstrated a small basal ganglia stroke with no hemorrhage. In a follow-up visit 6 months after thrombolysis, the patient had made a full neurologic recovery.

Table 2: Intervention and outcomes in the 7 patients with acute stroke due to occluded ICA

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>IA tPA (mg)</th>
<th>Stenting and angioplasty of ICA</th>
<th>Lysis of MCA Clot through occluded ICA</th>
<th>TIMI Flow after Thrombolysis</th>
<th>Post Rx CT</th>
<th>Outcome (mRS) at 30 days</th>
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<tr>
<td>1</td>
<td>20</td>
<td>Yes</td>
<td>Yes</td>
<td>2</td>
<td>Basal ganglia infarct</td>
<td>4</td>
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<tr>
<td>2</td>
<td>10</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
<td>Patchy parietal infarcts</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>No</td>
<td>Yes</td>
<td>3</td>
<td>Basal ganglia infarct</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0*</td>
<td>Yes</td>
<td>No</td>
<td>3</td>
<td>Basal ganglia infarct</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>No</td>
<td>Yes</td>
<td>3</td>
<td>Basal ganglia infarct</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>No</td>
<td>No</td>
<td>3</td>
<td>Evolving frontal bleed</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>Yes†</td>
<td>Yes†</td>
<td>3</td>
<td>Small frontal lobe infarct</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: ICA indicates internal carotid artery; IA tPA, intra-arterial tissue plasminogen activator; MCA, middle cerebral artery; CT, computed tomography; mRS, modified Rankin Scale.
* This patient received 38 mg intravenous tPA prior to angiography.
† IA pseudoaneurysm was treated with 2 overlapping covered stents.
Discussion

Untreated, the prognosis of patients with acute symptomatic cervical ICA occlusion is poor.7 Before endovascular therapy, emergent CEA yielded modest results, with good outcomes in 38%, fair in 29%, and poor in 12% and a surgical mortality rate of 21%.7 There were 2 important prognostic factors: the presence of an associated MCA embolus and lack of collateral flow, both of which correlated with poor clinical outcomes. Studies have shown that intravenous thrombolysis is ineffective in recanalizing the occluded cervical ICA.8 The poor outcome of less direct thrombolysis in ICA occlusion also involving the MCA is probably due to poor drug delivery to the thrombus and lack of collateral flow from lenticulostriate arteries. The stasis in the ICA proximal to the occlusion delivers little thrombolytic agent to the MCA thrombus.8 Several studies have reported successful navigation of an occluded cervical ICA to thrombolize a distal MCA clot.8,10,14 The largest series was published by Wang et al10 and described 6 patients, 5 of whom had good or excellent outcomes. Favorable clinical outcome at 30 days was also achieved by Mori et al,15 who performed balloon angioplasty of the MCA and stented the ipsilateral carotid stenosis in a 66-year-old man presenting with acute embolic total occlusion of the left MCA. In our opinion, the primary goal was to address the acute MCA clot first, because this was suspected to be the etiology of the patient’s acute symptoms. Once the MCA was recanalized, the attention was then directed to the ICA occlusion. It can be difficult to distinguish angiographically between acute thromboembolic occlusions and dissections causing complete occlusion at the ICA origin. In our limited experience with treatment of acute carotid occlusions, we have encountered mainly presumed atherosclerotic occlusions (except for one young patient with a traumatic dissection) and used similar treatment techniques regardless of its presumed etiology.

Traversing an occluded vascular segment has been described in the coronary arteries, in bypass grafts, and in the extremities. In theory, there is a risk of dislodging more thrombi while crossing the stenosed segment. In our experience, this seems to be a real possibility, although the dislodged thrombi have fortunately responded to IA thrombolysis. The other concern with blindly probing an occluded artery is the risk of entering the false lumen of an ICA dissection. Fortunately, this has not happened in our experience. Thus, we advise gentle probing with a slightly curved microwire and avoidance of advancing the microwire against resistance to prevent iatrogenic dissection of an atherosclerotic lesion or entrance into the false lumen of a dissection. In the one patient presenting with ICA occlusion from a traumatic dissection, the true lumen was easily found by gentle probing with a soft, curved microwire.

After successful intracranial thrombolysis, one is faced with the management dilemma of the proximal ICA occlusion. If traversing the proximal ICA occlusion with the microcatheter and subsequent administration of thrombolytics has resulted in restoration of ICA flow, we have opted to angioplasty and stent the proximal ICA lesion because of concern for future thromboembolic events. The rationale for this approach lies in previously reported macroscopic or histopatho-

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**Fig 1.** A, Diagnostic angiogram of the left internal carotid artery (ICA) reveals good flow across the anterior communicating artery (AcomA) but poor filling of the right MCA territory because of a thrombus demonstrated on an earlier CT angiogram. B, Successful recanalization of right MCA is seen after administration of 9 mg of tissue plasminogen activator (tPA) into the middle cerebral artery (MCA). C, Diagnostic angiogram of the left ICA reveals cross-filling through the AcomA into the right MCA. There is probably dilution of contrast in the MCA by nonopacified blood from the ipsilateral posterior communicating artery because good patency of the MCA was demonstrated on the earlier microcatheter injection.
logic investigations, which have revealed that lesions of atherothrombotic ICA occlusion often have a thick atheroma plaque associated with fresh thrombus. Some plaques have demonstrated thrombi in areas showing intraplaque hemorrhage. This suggests to us that, if flow is restored in the ICA, the potential for thrombi to develop in areas of plaque hemorrhage exists and may be treated with angioplasty and stent placement. After intracranial thrombolysis, if there is demonstration of good collateral flow to the parenchyma despite a persistent ipsilateral ICA occlusion, we feel it is reasonable to leave the ICA occluded.

In conclusion, performance of IA thrombolysis by passing a microcatheter through an acutely occluded internal carotid artery may be an effective therapy in a setting of acute stroke.

References


Fig 2. A, Lateral angiogram of the left common carotid artery (CCA) in a 24-year-old man with acute left hemiplegia reveals a pseudoaneurysm (arrow) in the distal cervical internal carotid artery (ICA) with narrowing of the parent artery. B, The right CCA angiogram shows complete occlusion of the right ICA (arrow) just beyond the bulb. C, Microcatheter injection demonstrates a thrombus in the distal M1 segment (arrow) of the middle cerebral artery (MCA). D, Administration of 7.5 mg of intra-arterial tissue plasminogen activator (IA tPA) through the microcatheter, which was advanced into the face of the thrombus, resulted in lysis of thrombus and restoration of antegrade flow. E, Microcatheter injections demonstrate a pseudoaneurysm in the distal cervical right ICA (arrow). F and G, Repeat angiogram through the guiding catheter after successful treatment by using 2 overlapping covered stents reveals no filling of the pseudoaneurysm and normal flow in the MCA. The patient recovered complete power on the left side within minutes of the procedure. The left ICA dissection and pseudoaneurysm were managed conservatively and, at 2 months after the procedure, the patient had no neurologic deficits.