Endovascular Treatment of Acute Thrombotic Occlusion of the Cervical Internal Carotid Artery Associated with Embolic Occlusion of the Middle Cerebral Artery: Case Report

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A 68-YEAR-OLD MAN with acute, total thrombotic occlusion of the right cervical internal carotid artery, associated with embolic occlusion of the ipsilateral middle cerebral artery, was treated by the endovascular approach, i.e., intra-arterial fibrinolysis and balloon angioplasty. Endovascular treatment for a totally occluded internal carotid artery is indicated in the very early stage where there is no thrombus or a short thrombus that can be evacuated. Fibrinolysis of the associated embolus in the middle cerebral artery, if any, can be done in the same session, enabling an early restoration of the blood flow. (Neurosurgery 34:359–364, 1994)

Key words: Fibrinolysis, Internal carotid artery, Middle cerebral artery, Percutaneous transluminal angioplasty, Thrombosis

Total occlusion of the cervical internal carotid artery (ICA) may be asymptomatic, may present with transient ischemic attack or completed stroke, and may sometimes be fatal (2, 4, 5, 8, 15). Cervical ICA occlusion associated with middle cerebral artery (MCA) embolic occlusion usually results in a poor outcome (10, 16). Possible surgical treatments for ICA occlusion and a MCA embolus are thromboendarterectomy (TEA), superficial temporal artery-MCA anastomosis (28), and embolectomy (16).

Using the endovascular approach, physicians can now perform percutaneous intra-arterial fibrinolysis (3, 21, 29) and angioplasty (1, 6, 11, 12, 17, 25, 26) in the carotid system. We report a patient with total occlusion of the cervical ICA associated with embolic MCA occlusion that was successfully treated by an endovascular approach.

CASE REPORT

This 68-year-old man suddenly developed left hemiparesis and was transferred to our hospital 90 minutes later. Twelve years before this event, he suffered a hypertensive left intracerebral hemorrhage, which was treated by surgical evacuation in another hospital. He recovered completely from this hemorrhage, except for right homonymous hemianopsia, and has been otherwise healthy and able to care for himself.

At admission, he was awake and oriented. He had mild impairment of his mental status, severe left hemiparesis (one-fifth muscle strength), and right homonymous hemianopsia caused by the previous intracranial hemorrhage.

A plain skull x-ray revealed a previous craniotomy in the left temporal region, and computed tomography disclosed cerebral atrophy of the left temporal lobe,

but no hemorrhage or any other abnormality on the right side. Acute right cerebral ischemia was suspected, and transfemoral cerebral angiography was performed immediately after the computed tomographic examination.

An internal left carotid injection showed a normal left anterior circulation and the right anterior cerebral artery filled through the anterior communicating artery, but only the distal portion of the right MCA was faintly visualized through the leptomeningeal anastomosis in the very late phase, and the M2 portion of the right MCA was not demonstrated. An injection of the right common carotid artery demonstrated the total occlusion of the cervical ICA and retrograde filling of the cavernous and petrous portions of the ICA through ophthalmic anastomosis and embolic occlusion of the right MCA at the proximal M1 segment (Fig. 1). The downward trickling of the contrast material toward the petrous carotid artery suggested fresh occlusion.

Endovascular treatment was attempted because of the short duration from the onset, the associated embolic occlusion of the MCA, the short length of the thrombus in the ICA, the relatively high position of the cervical ICA bifurcation, and severe right hemispheric ischemia. Systemic heparinization with 5000 U of heparin was administered before intervention. A guide wire (0.038 inch) could easily pass through the occluded segment of the ICA at two-thirds of the cervical vertebral level. A diagnostic catheter (6.5 French) could also pass through the occlusion site over the guide wire without any difficulty. When the tip of the catheter was placed distal to the occlusion site, soft, fresh thrombus was removed by gently sucking with a syringe. Then, 3 million IU (5.2 mg) of tissue plasminogen activator (t-PA) diluted with 10 ml of saline was slowly injected over 5 minutes to fibrinolyze the embolus in the MCA and to prevent further distal embolization from the cervical ICA (Fig. 2).

When the catheter was repositioned proximal to the occlusion, recanalization was obtained, and severe stenosis of

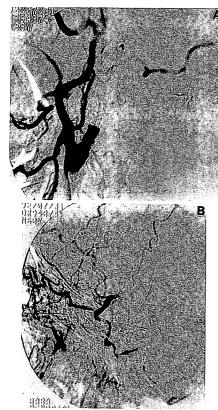


FIGURE 1. A and B (both lateral projections): Right common carotid injection demonstrates the complete occlusion of the cervical ICA near the bifurcation (A) and faint visualization of the cavernous and petrous portions of the ICA (arrow) through the ophthalmic anastomosis (B).

more than 80% of the ICA diameter could be identified and presented the cause of the thrombosis (Fig. 3). Proximal to the stenosis, fresh thrombus was observed, which was further removed through the diagnostic catheter. Although another 3 million U (5.2 mg) of t-PA was injected into the ICA proximal to the stenosis in the same manner, reocclusion of the ICA soon occurred. Next. the double-lumen Accent balloon catheter (2 cm in length and 6 mm in inflated balloon diameter; Cook Company, Bloomington, IN) was navigated to the occlusion site, and the balloon was inflated twice at 5 atm for 30 seconds and 1 minute, respectively, with an interval of 5 minutes. This resulted in the complete recanalization of the ICA.

A right carotid injection demonstrated recanalization of the right ICA, with

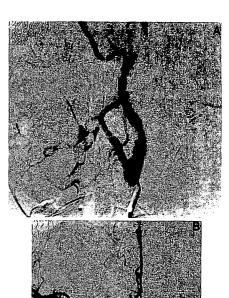


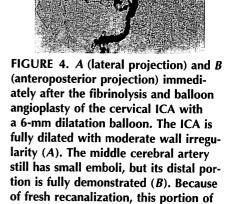
FIGURE 2. Right internal carotid injection (anteroposterior projection) after vigorous removal of the clot through the diagnostic catheter. The catheter tip (open arrow) is navigated beyond the occlusion site while the proximal ICA is still completely occluded. Closed arrows indicate the emboli in the middle cerebral artery.



FIGURE 3. Right common carotid injection (lateral projection) demonstrates recanalization of the ICA, but the stenosis of more than 80% is still noted. *Arrow* indicates the initial occlusion site. Proximal to this, the fresh clot is observed.

moderate irregularity of the arterial wall and recanalization of the right MCA (*Fig.* 4). There still were small emboli at the MCA bifurcation, but the distal MCA was fully visualized. Because the patient's condition improved markedly and because we were afraid of hemor-





the MCA is dilated and the flow here is

slower than usual.

rhagic complications with an additional infusion of t-PA, no further attempt was made to lyse the MCA emboli with a microcatheter. Immediately after this intervention, the patient's mental slowness improved and the right hemiparesis also improved to four-fifths muscle strength. Systemic heparinization with 24,000 IU/d of heparin was administered for 4 days. During this period, activated partial thromboplastin time was maintained at double the normal value. Ticlopidine, 200 mg/d, was administered immediately after intervention.

Control right carotid angiography, performed 3 days later through the femoral sheath that was left in place, disclosed the patent cervical ICA with some

irregularity of the wall and fully recanalized MCA without any embolus (Fig. 5). Computed tomography revealed a small low-density area in the right putamen, which disappeared in a month. No hemorrhagic episode occurred. Ten days later, the patient could walk without assistance and was discharged from the hospital 1 month postictus. At the time of discharge, his neurological condition had returned to the baseline prehospitalization status. He continued to receive ticlopidine, 200 mg/d. At the last follow-up, 5 months after discharge, his neurological examination was stable.

DISCUSSION

The natural history of total occlusion of the cervical ICA results in an initial mortality of 3 to 25%. Among the survivors, the subsequent stroke rate ranges from 6 to 25%, and the subsequent rate of transient ischemic attack ranges from 5 to 51% (2, 4, 5, 8, 15). The wide range of these figures might be partially the result of the different populations studied. Neurological symptoms vary from asymptomatic (silent) and transient ischemic attack to completed stroke with degrees of severity and death. Although in the chronic stage, the recurrence of cerebral ischemia on the occluded side is reported to be significantly less frequent (4, 8), a contradictory result (more frequent ischemia on the ipsilateral side) is also reported (2, 5).

A patient with total occlusion of the cervical ICA was once thought not to be a candidate for TEA. Instead, carotid endarterectomy for the contralateral carotid stenosis (18) and ipsilateral superficial temporal artery-MCA anastomosis (28) had been recommended. In the late 1970s, TEA for occlusion of the cervical ICA with or without a Fogarty balloon catheter was reported, but it was not widely accepted (5, 19, 21). Shucart and Garrido (21) reported that this procedure was indicated when angiography disclosed a retrograde flow into the cavernous portion of the ICA. Hugenholtz and Elgie (10) classified the collateral supply distal to the occlusion into five grades: Grade 1, nonvisualization of the entire intracranial ICA; Grade 2, visualization of the ICA proximal to the posterior communicating artery, distal to the ophthalmic artery; Grade 3, visualization of the ICA proximal to the ophthalmic artery, distal to the meningohypophyseal artery; Grade 4, visualization of the cavernous ICA to the point of superimposition of the ICA and the floor of the sella turcica on lateral projection film; and Grade 5, visualization of the ICA into the carotid canal, but not beyond the base of the skull. They concluded that patients with Grade 1 to 3 collateral supply should not be explored unless occlusion had occurred very recently and that patients with Grade 4 to 5 collateral supply were good candidates for recanalization, regardless of the duration of occlusion.

Recently, TEA for acute occlusion of the ICA has been reported by several authors (9, 13, 14, 16, 24). Most of the patients treated in the acute stage were those awaiting definitive carotid surgery, and those who sustained peri-angiography occlusion or postoperative occlusion of the carotid artery (9, 14, 16, 19). This enabled the acute surgical intervention to be performed and resulted in a relatively good outcome. According to Grillo et al. (8), reopening of the occluded carotid artery is indicated for selected patients with acute stroke. Early intervention (short duration of carotid occlusion) results in a better patency rate

Thompson et al. (24) reported 100 cases of total ICA occlusion treated by TEA with a mortality rate of 7.4% and stated that the best anatomical results were obtained in patients who underwent surgery within 6 to 12 hours from the onset of symptoms. They concluded that patients with acute, profound, rapidly progressing strokes should not undergo surgery because of the prohibitive rate of mortality. Lindberg (14) reported emergency TEA of six patients within 2 hours after the onset of deterioration. Although carotid flow was restored in all cases and in three patients the neurological deficits disappeared, in the remaining three patients, there was no change in their clinical status. Most au-

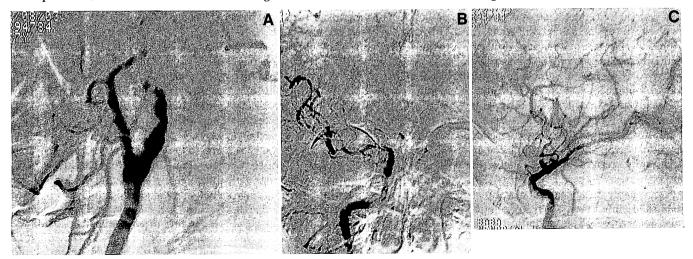


FIGURE 5. A (lateral projection), B (anteroposterior projection), and C (lateral projection). The right common carotid injection 3 days later shows the patent ICA with mild wall irregularity, essentially the same feature in the postangioplasty state (A). Intracranially, neither embolus nor arteriovenous shunting is shown. The circulation time of the right hemisphere is normal (B and C).

thors advise against emergency TEA for patients with severe neurological deficits. Meyer et al. (16), however, reported fairly good results (a 38% rate of good recovery among 31 patients) for such patients after careful patient selection and suggested that good prognostic factors for patient selection are an absence of an associated MCA embolus and the presence of good collateral flow.

The outcome of emergency carotid endarectomy seems to depend on the following: 1) duration of symptoms; 2) individual collateral blood flow; 3) evidence of distal migration of the thrombus; and 4) cerebral tissue vulnerability. We think that it is possible to reestablish the blood flow in the carotid artery in the ultra-acute stage by means of an endovascular technique, which can be performed immediately after angiography. This enables an earlier restoration of the carotid flow than does surgical treatment. Endovascular treatment also enables careful neurological monitoring throughout the procedure and prompt detection of any adverse change in the patient's clinical status.

The endovascular approach now enables percutaneous local fibrinolysis (3, 22, 29) and angioplasty (1, 6, 11, 12, 17, 25, 26) of the carotid system after diagnostic angiography, although most angioplasty is done for carotid stenosis as an elective procedure. Tsai et al. (26) are credited with performing the first percutaneous transluminal angioplasty in a 60-yearold woman for right, total ICA occlusion, with symptoms of intermittent attacks of aphasia and mild weakness in both sides. Percutaneous transluminal angioplasty was successful with a good outcome. Courthéoux et al. (3) reported a 52-year-old man with total thrombotic occlusion of the cervical ICA treated with fibrinolysis with streptokinase and angioplasty on Day 1. Although the angiographic results were excellent, the outcome was "subtotal recovery." This was probably because of an already established infarction. They concluded that local fibrinolysis is indicated for postoperative ICA thrombosis and carotid stenosis with floating clots extending to the cranial base, whereas it is contraindicated for belated cases and patients with altered consciousness and/or involvement of the lenticulostriate arteries. Théron (23) reported a 54-year-old patient with left cervical ICA thrombosis with MCA occlusion. After thrombolysis with urokinase, he performed "minimal ICA angioplasty." The outcome was moderate disability, and he stated that incomplete angioplasty is enough to keep the ICA open.

ICA occlusion associated with MCA embolic occlusion indicates poor outcome (10, 16). It is impossible to know whether MCA embolus is a natural event or whether it is induced by manipulation. Direct TEA might have less risk of dislodging the thrombus than endovascular treatment. However, during this surgery, dislodging the thrombus, if there is one, cannot be recognized. Distal embolization is a potential risk of using a Fogarty balloon catheter. Meyer et al. (16) reported 6 of 14 intraoperative angiograms were positive for MCA embolus, which necessitated an emergency embolectomy immediately after the

The major risk of percutaneous transluminal angioplasty for ICA occlusion is a dislodging of the thrombus into the intracranial circulation. To eliminate or to significantly reduce this complication, two techniques are used: 1) evacuating the fragile thrombus by negative pressure through the diagnostic catheter, using a syringe; and 2) local use of t-PA for fibrinolysis. Although there is no widely accepted dose of t-PA for this kind of procedure, we used a total dosage of 6 million IU (10.4 mg) of t-PA in this case. This dose is based on accumulated experience in using local fibrinolysis for acute occlusion of the coronary arteries.

The shorter the length of the thrombus in the occluded ICA, the easier it is to remove it. It should be noted that some of the patients with total ICA occlusion had no thrombosis in the ICA distally (10, 13). These patients are the best candidates for emergency angioplasty. Hafner and Tew (9) think that retrograde filling of the ipsilateral ICA to the petrous or cavernous portion is a good sign of TEA operability. This finding is classified as Grade 3 to 5, according to Hegenholtz (10). This can be adopted for the endovascular treatment. Should dislodging of the thrombus into the intracranial vessels occur, t-PA through the microcatheter near or in the thrombus might lyse it. For endovascular treatment, it is indispensable to select the patient with a shorter thrombus in the ICA. Thus, patients with Grade 4 to 5 collateral supply are good candidates.

It is not always possible to perform TEA immediately after the angiography. High risk of cardiac complication (myocardial infarction) (8, 15, 19) might warrant the endovascular approach in selected patients instead of TEA under general anesthesia. Preoperatively, it cannot be recognized how hard or soft the thrombus is, whether or not it can be evacuated through the catheter, or where and how severe the atherosclerotic stenosis of the ICA is. We have noticed in several patients with total ICA occlusion that the guide wire could pass through and be advanced distal to the occlusion site without any resistance. Fresh thrombus distal to the atherosclerotic stenosis might be soft enough to be removed through the catheter in the very early stage. The site of severe stenosis is usually located near the cervical carotid bifurcation. As a preoperative procedure for emergency TEA, it is possible to inject t-PA through a microcatheter during angiography within or distal to the thrombus to facilitate the removal of thrombus and to prevent distal embolization during surgical intervention.

Another complication of restoring the carotid flow is intracranial hemorrhage (7, 27). Most of the hemorrhage occurred in patients who did not undergo surgery in the acute stage and whose infarction was already complete. Early intervention, whether endovascular or surgical, might eliminate this type of complication.

For postintervention management, our patient was treated with systemic heparinization, 24,000 U of heparin for 4 days, and was also given ticlopidine, 200 mg/d orally. The dose of heparin, which maintained activated partial thromboplastin time at roughly double the normal value, and the duration of heparinization are based on accumulated experience in percutaneous transluminal coronary angioplasty. The antiaggregation effect of ticlopidine starts at 24 to 48 hours, and its maximal effect is reached after 3 to 6 days of administration (20).

We think that, at present, endovascular treatment for totally occluded ICA is

indicated in the very early stage, with no thrombus or a short thrombus that can be evacuated. Fibrinolysis of the associated MCA embolus, if any, can be done in the same session, enabling an early restoration of blood flow.

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COMMENTS

This article describes the endovascular treatment of an embolus to the middle cerebral artery and a thrombotic occlusion of the internal carotid artery and demonstrates that this disease process, which has no good surgical therapy and a poor natural history, can be well treated by endovascular approaches. There may be some advantage to superselecting the middle cerebral artery for thrombolytic therapy. The dose of the thrombolytic agent can possibly be lowered by this more direct administration of the drug. In addition, there may be some benefit to mechanical disruption of the clot by the microcatheter, such as the Tracker. I would agree that the early post-thrombolytic result of the fibrinolytic therapy is not perfect in the middle cerebral artery, but in my own experience, I have found that if there is good anterograde flow in the artery, then complete clearing of the vessel may not be necessary, as the authors indicate. The follow-up angiogram confirms that there has been further fibrinolytic action with clearing of the clot. Their management of the internal carotid artery thrombosis is novel, and I think is a significant step in the overall management of this disease. I have treated a patient with a similar problem that I thought was related to a dissection. After opening the supraclinoid internal carotid artery and the middle and anterior cerebral arteries and thereby the Circle of Willis, I went on to occlude the internal carotid artery because I thought there was a dissection in the artery, which could be a source for further embolic problems. However, after reading this report, I wonder if I should have attempted angioplasty on the carotid artery, as these authors described.

The authors have reported this dual therapy in the treatment of cerebrovascular occlusive disease. Rapid advances are being made in the treatment of stroke, and I think it is no longer a disease that should necessarily be left to its own natural history or be treated with systemic thrombolytic or anticoagulants alone. The endovascular approach to this type of problem is gaining popularity among skilled neurointerventionalists.

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This is a case report that illustrates the beginning of a new era in the manage-

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ment of occlusive vascular disease. A major revolution will occur with our capabilities to recanalize cerebral circulation. Newer catheters of the conventional type, variable stiffness, microcatheters, dilatation, balloon catheters, as well as our increased familiarity with chemical recanalization with thrombolytic therapy have the potential of making a major impact in the management of this devastating disease. Although the use of thrombolytic therapy in the ca-

rotid arteries was attempted in the 1960s, sometimes with lethal results, the newer availability of better technology and the experience gained from coronary and peripheral recanalizations will permit us to correct the deficiencies of the 1960s. This case illustrates the multiple modalities possible, such as thrombectomy by suction, angioplasty with a balloon, the ability to cross the clot with a guide wire, the use of intrathrombi chemical lysis, as well as the possibility of thrombolysis in

distal cerebral arteries. This represents an exciting era. Our experience with nine cerebral recanalizations with a combination of angioplasty and thrombolysis demonstrates the feasibility of this technique.

In this early period, protocols and doses are still not well defined. Therefore, the experience of the authors is an important parameter to consider.

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