Arterial Occlusion Following Anastomosis of the Superficial Temporal Artery to Middle Cerebral Artery

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SUMMARY Symptoms of cerebral ischemia following superficial temporal artery (STA) to middle cerebral artery (MCA) anastomosis are uncommon and are usually related to impaired flow through the anastomosis or occlusion of the cortical receptor artery. In this report, 3 patients are described who developed symptoms of cerebral ischemia after surgery despite a widely patent anastomosis. In each patient, postoperative angiography revealed occlusion of a previously demonstrated high-grade stenosis of the intracranial internal carotid artery (ICA) or MCA. Occlusion of the stenotic artery was probably related to decreased velocity of blood flow through the narrowed vessel resulting from alterations in flow patterns following anastomosis.

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THREE PATIENTS underwent superficial temporal artery (STA) to middle cerebral artery (MCA) anastomosis for symptomatic high-grade atherosclerotic stenosis of a major intracranial artery. All patients developed focal neurologic deficits in the immediate postoperative period. Angiography revealed occlusion of the bypassed stenosis with a patent anastomosis in each patient.

Case #1

A 56-year-old right-handed man presented with several transient episodes of aphasia and right arm weakness occurring over a 72-hour period. The patient had a long prior history of vascular disease, having sustained 2 remote myocardial infarctions. There was also a history of chronic hypertension, hyperlipidemia, and peptic ulcer disease. In May, 1977, a right vertebral artery to common carotid artery implant procedure had been performed for episodes of vertebral-basilar insufficiency. Carotid angiography at that time revealed moderate irregularity of both internal carotid artery siphons with retrograde filling of the basilar artery via the left posterior communicating artery. The left vertebral artery was occluded, and the right vertebral artery origin was tightly stenotic.

Three months after the vertebral artery implant, the patient developed transient episodes of aphasia and right arm weakness consistent with left cerebral hemisphere ischemia. Antiplatelet therapy with aspirin, and later dipyridamole, was begun. Despite antiplatelet therapy, he continued to experience frequent attacks of right-sided numbness. From May 15 through May 18, 1979, he had at least 10 attacks of aphasia and/or right arm weakness, which led to his admission to the Cleveland Clinic.

The admission examination revealed minimal speech hesitancy, right side hyperreflexia, and a right Babinski response. There was a loud systolic/diastolic right ocular bruit, and a softer left ocular systolic bruit.

The patient was stabilized on a continuous heparin infusion and repeat carotid angiography was performed on May 17, 1979. This revealed moderate stenosis of the right internal carotid artery (ICA) siphon. The left ICA siphon was markedly stenotic (approximately 90%), indicating definite progression from the 1977 angiogram. The vertebral artery implant was patent but the implant did not fill any vessels intracranially.

On May 24, 1979, a left STA-MCA anastomosis was performed. The operative procedure was uneventful, but an episode of laryngospasm at the time of extubation required respiratory assistance. On the first postoperative day the patient was alert with fluent speech and no new motor deficits. That evening he was noted to have fluctuating right arm weakness and slurred speech. Several episodes of hypotension occurred, and respiratory distress with hyperventilation necessitated intubation. Neurologic examination on May 31 revealed a semi-comatose patient with no spontaneous speech. There was some appropriate movement of the left side to painful stimuli, but the right side was flaccid. An electroencephalogram showed diffuse slowing with left hemisphere emphasis. A subsequent electroencephalogram revealed decreased background activity in the left hemisphere and continuous left temporal delta activity. A computer tomographic brain scan performed on June 1 demonstrated a vague area of decreased attenuation in the left parietal region thought to be an infarct. Angiography performed on June 1 showed that the intracranial segment of the left ICA was occluded in the vicinity of the previous tight stenosis. The anastomosis was patent and filled the intracranial circulation in both an anterograde and retrograde fashion up to the point of occlusion. Subsequently the patient has made an incomplete recovery.

Case #2

A 59-year-old right-handed man with a prior history of myocardial infarction and coronary revascularization experienced the sudden onset of
aphasia and right hemiparesis in July, 1978. A computer tomographic brain scan revealed a left frontal infarction. Angiography elsewhere demonstrated an ulcerated stenosis at the origin of the left ICA and moderate stenosis at the origin of the left MCA. The patient subsequently underwent an uneventful left carotid endarterectomy in August, 1978. He did well until March 2, 1979, when he again suddenly developed aphasia and right hemiparesis, which cleared within a few days except for mild residual right-side weakness. Carotid angiography performed on April 9, 1979, revealed a normal left ICA origin. However, there was now severe stenosis (greater than 90%) of the left MCA origin. The patient was referred to the Cleveland Clinic for a possible left STA-MCA anastomosis. The admission examination revealed a mild spastic right hemiparesis and mild speech non-fluency. There were no eye or neck bruits, and the superficial temporal artery pulses were full and equal.

On April 13, 1979, the patient underwent a left STA-MCA anastomosis. The initial postoperative neurologic examination was unchanged. On April 19, 1979, angiography was performed to document patency of the anastomosis. This study revealed complete occlusion of the left ICA just distal to the origin of the anterior choroidal artery. The anastomosis was patent and filled most branches of the left MCA.

Immediately following angiography, the patient complained of a left visual field defect. He subsequently appeared confused. Examination revealed a marked fluent dysphasia with jargon speech and numerous paraphasic errors. There was increased right arm weakness. Ophthalmological evaluation revealed a large central scotoma in the left eye and funduscopic findings consistent with retinal ischemia. There was no evidence of recent infarction on a computer tomographic brain scan. The patient subsequently returned to his preoperative state, except for the scotoma in the left eye, by the time of discharge on April 27, 1979.

Case #3

A 60-year-old right-handed woman was admitted to the Montreal Neurological Hospital one month following a 4-day episode of right-hand weakness and right-side sensory loss. She had experienced 2 transient episodes suggestive of vertebral-basilar insufficiency 4 years prior to admission, at which time warfarin, and later aspirin, were begun. One year prior to admission she had experienced a 24-hour episode of dysphasia. Her past history also included well-controlled hypertension and peptic ulcer disease.

The admission neurologic examination revealed bilateral carotid bruits, more marked on the right. Rapid alternating movements were mildly decreased in the right hand. There was no evidence of a language disturbance, and the rest of the neurologic examination was unremarkable.

A computer tomographic brain scan demonstrated moderate generalized cortical atrophy. Carotid angiography performed on November 15, 1977, showed a moderate stenosis of the proximal left ICA and a more severe, focal stenosis of the supraclinoid arterial segment. There was occlusion of some of the temporal branches of the left MCA. Less marked atherosclerotic changes were also present in the right intracavernous ICA.

On December 1, 1977, the patient underwent a left STA-MCA anastomosis. The cerebral circulation was studied intraoperatively using fluorescein angiography and 133Xenon clearance. 1 The sodium fluorescein and 133Xenon were injected through a polyethylene catheter inserted percutaneously into the left common carotid artery prior to surgery. The studies were carried out before and after anastomosis of the STA to the angular branch of the left MCA. 133Xenon detectors were placed on the inferior frontal, supramarginal, angular, and superior temporal gyri.

Before anastomosis, fluorescein filling of the frontal cortical arteries was initially seen 1.7 seconds following injection. Later filling was restricted to MCA cortical branches in the frontal and parietal areas. This indicated the distribution of arterial blood entering the cerebral circulation through the stenotic left ICA. Mean regional cerebral blood flow (rCBF) was 31 ml/100gm/min. After anastomosis, fluorescein filling of the angular artery occurred 0.9 seconds after injection. Fluorescein entering the cerebral circulation through the STA filled the angular and posterior temporal arteries. Filling of the frontal and parietal arteries was slower than before anastomosis, that is, initial filling was seen 2.3 seconds after injection (fig. 1). Mean rCBF was 35 ml/100gm/min with most of the improvement occurring in the angular artery territory (18 ml/100gm/min pre-anastomosis, 33 ml/100gm/min post-anastomosis). No increase of rCBF occurred in the inferior frontal or supramarginal areas after anastomosis.

In the first few postoperative days the patient developed episodic dysphasia and weakness in the right arm and face, possibly related to moderate

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**Figure 1.** Intraoperative fluorescein angiogram in Case 3. Upper left — left parieto-temporal craniotomy before anastomosis. The arrow indicates the angular artery. Upper right — at 2.44 seconds following injection, fluorescein can be seen filling some of the frontal and parietal branches of the left MCA. Middle left — an anastomosis (arrow) of the left STA and angular artery has been performed. Middle right — at 1.10 seconds, fluorescein can be seen in the angular artery. Lower left — at 3.32 seconds, there is substantial filling of the cortical microcirculation supplied by the angular and posterior parietal branches. Filling of the frontal arteries is similar to that seen at 2.44 seconds before anastomosis. Lower right — filling of the cortical microcirculation was generalized at 4.74 seconds.
hypotension. The patient was also mildly anemic. These deficits cleared with restoration of the blood pressure and correction of the anemia. Postoperative angiography performed on December 12, 1977, revealed a fully patent STA-MCA anastomosis with significant back flow into the MCA circulation. The left ICA was occluded just distal to the origin of the ophthalmic artery (fig. 2).
FIGURE 2. Left carotid angiograms in Case 3. The pre-anastomosis angiogram (A) shows moderate stenosis of the internal carotid artery (ICA) origin, and marked stenosis (arrow) of the supraclinoid ICA. In an early view (B), the post-anastomosis angiogram demonstrates a widely patent STA-MCA anastomosis (arrow). In a later view (C), the post-anastomosis angiogram demonstrates occlusion of the ICA just distal to the origin of the ophthalmic artery (arrow).

Discussion

Recent reports suggest that STA-MCA anastomosis can reduce the frequency of brain infarction in a selected group of patients with cerebral vascular disease.\textsuperscript{2-4} Bypass surgery has been performed most frequently for ICA occlusion, but a number of procedures have been carried out in patients with high-grade stenosis of the distal ICA or proximal MCA. These lesions probably produce symptoms of cerebral ischemia on a hemodynamic basis rather than on an embolic basis. Regional cerebral blood flow studies appear to substantiate this hypothesis. Pre-anastomotic reduction of rCBF to 25 ml/100gm/min or less has been reported by Schmiedek et al.\textsuperscript{5} and Little et al.\textsuperscript{1,6} In patient 3, rCBF in the left angular gyrus region was 18 ml/100gm/min before anastomosis.

Peri-operative infarction is uncommon following bypass surgery, and is usually thought to be related to impaired flow through the anastomosis or occlusion of the cortical receptor artery. However, our 3 patients developed postoperative cerebral ischemia which appeared to result from secondary occlusion of the bypassed ICA or MCA stenosis. The anastomoses in our patients were widely patent. In patient 3, fluorescein angiography demonstrated delayed filling of frontal and parietal arteries after anastomosis when compared with the study before anastomosis. This suggested reduction in velocity of flow through the stenotic ICA which supplied those areas. Apparently shunting of blood through the external carotid artery and STA-MCA anastomosis produced a small but critical drop in flow through the stenotic ICA or MCA resulting in relative stasis and subsequent thrombotic occlusion. Although the anastomosis was patent, initial flow through it was not sufficient to prevent cerebral infarction or transient cerebral ischemia. Arterial bypass grafts dilate with time and the blood flow through them increases correspondingly.\textsuperscript{7} As with any acute vascular occlusion, if collateral flow channels, in this case the STA-MCA anastomosis, are inadequate or incompletely developed infarction is more likely to occur.

Factors which influence cerebral perfusion pressure are important in maintaining blood flow through a tightly stenotic vessel. To minimize the risk of occlusion of a bypassed stenotic artery, careful attention should be paid to postoperative factors which might interfere with cerebral perfusion pressure. In 2 of our patients, reduction in the systemic arterial pressure during the postoperative period probably contributed to the thrombotic occlusion of the bypassed artery. Avoidance of hypotensive episodes in the early postoperative period by the adequate maintenance of intravascular volume appears to be of critical importance in these patients. Although potentially hazardous, antiplatelet therapy or anticoagulation during the immediate postoperative period could be considered for patients in whom the risk of secondary occlusion is thought to be high.

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**Spontaneous Bilateral Recanalization in Bilateral Internal Carotid Artery Occlusion**

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**SUMMARY** The authors report a young woman with bilateral internal carotid artery occlusion shown by carotid angiography. There was spontaneous bilateral recanalization demonstrated radiographically. The possible causes, e.g., the intake of oral contraceptive drugs, a spontaneous intimal dissection and other etiological features are discussed and the literature is reviewed.

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**BILATERAL OCCLUSION** of the internal carotid arteries is often found in adults of middle and older age with risk factors such as atherosclerosis, hypertension, cardiovascular disease, diabetes mellitus and lipid metabolism abnormalities.1-7

Unilateral and bilateral occlusive disease of the internal carotid arteries has been reported also in young adults, with or without these risk factors.8-12

Spontaneous recanalization of one of the obstructed vessels has been described in both age groups,13-16 although it has been predominantly observed with occlusions of the middle cerebral artery.17-18 Radiographic documentation of recanalization is rarely obtained because of the lack of an indication for another neuroradiological investigation, such as angiography, in these patients with a stable clinical state.

Internal carotid artery occlusions in young women using oral contraceptive medication was described first by Lorentz18 in 1962 and later by Wolf19 and de Gennes.20-22 Cole,23 Mandel,24 Mumenthaler,25 Pesendorfer,26 and others.27-30 Only one patient with bilateral thrombosis of the internal carotid arteries, a young woman using oral contraceptives, has been reported (Mandel22 in 1969). In this patient recanalization did not occur and she died in a decerebrated state. We report a patient with bilateral internal carotid artery occlusion in whom there was bilateral spontaneous recanalization proven radiologically.

**Case Report**

The patient was a 33-year-old white American woman. She was admitted to the neurology department of the Bernese University on April 3, 1971, because of sudden loss of speech, nausea and right facial palsy. There was no loss of consciousness. Examination confirmed a right-sided facial palsy of the upper motor neuron type, some buccolingual apraxia, a discrete right hemiparesis without any sensory loss and an almost complete motor aphasia. Subsequent carotid angiography on both sides revealed an occlusion of the left internal carotid artery located 1 cm distally to the bifurcation of the common carotid artery and an occlusion of the right internal carotid artery at the level of the atlas.

There was collateral circulation via the opthalmic arteries on both sides (fig. 1 A, B). Her blood pressure showed 140/80 mm Hg at several recordings. Her red and white blood count showed erythrocytes within normal range, leucocytes 4000/mm³ with mild shift to the left. Hemoglobin was 15.3 g % hematocrit 46 %, thrombocytes 196000/mm³, Quick 100%, partial thromboplastin time 46 sec, thrombin time 10.3 sec, fibrinogen 320 mg %. Factors I, V, VII and X were within normal range. Electrolytes and the blood lipid contents were: total cholesterol 159 mg/100 ml, triglycerides 55 mg/100 ml, phosphatids 167 mg/100
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