

Treatment of Acute Traumatic Internal Carotid Artery Occlusion with Extracranial-to-Intracranial Arterial Bypass: Case Report

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Ligation of the cervical internal carotid artery resulted in an acute neurological deficit in the dominant hemisphere of a 35-year-old man who suffered a penetrating injury to the neck. Regional cerebral hypoperfusion was suspected because the ischemic symptoms occurred while the patient was fully heparinized. Immediate institution of a barbiturate coma, volume expansion, and placement of a high-flow extracranial-intracranial arterial bypass graft led to rapid recovery of hemispheric function. High-flow extracranial-intracranial bypass grafts appear to be indicated for the treatment of symptomatic cerebral ischemia in selected cases of acute ligation or occlusion of the extracranial carotid artery. (*Neurosurgery* 25:90-92, 1989)

Key words: Acute cerebral ischemia, Cerebral infarction, Extracranial-intracranial arterial bypass, Penetrating vascular injury

INTRODUCTION

The results of the International Cooperative Extracranial-Intracranial (EC-IC) Bypass Study (8, 11) have created great uncertainty about the indications for this procedure in patients with ischemic cerebrovascular disease. EC-IC bypass improves hemispheric blood flow (20, 23) and perfusion (18) and therefore may still be useful in the setting of therapeutic vascular occlusion for the treatment of giant intracranial aneurysms (14, 25), symptomatic regional cerebral hypoperfusion due to *moya-moya* disease (15) or cerebral vasospasm (22), and acute (2, 15) or multiple (18, 20) arterial occlusions. Acute and progressive hemispheric cerebral ischemia were clinical conditions not specifically addressed by the International Cooperative EC-IC Bypass Study (1, 9). This report describes a case in which acute ischemia in the dominant hemisphere after an emergency ligation of the cervical internal carotid artery (ICA) was successfully treated with a high-flow EC-IC bypass graft.

CASE REPORT

This 35-year-old, right-handed man was stabbed in the left side of the neck at the angle of the jaw. When the paramedics arrived, he was unconscious, had no recordable blood pressure or pulse, and was bleeding from the stab wound. Intravenous volume expansion and cardiopulmonary resuscitation were initiated, the wound was manually compressed, and the patient was placed in a pneumatic antishock garment.

Upon arrival in the emergency room, the patient was combative, moving all four extremities, and had a pulse of 100 beats/minute. His systolic blood pressure remained below 60 mm Hg. He was intubated and ventilated with 100% oxygen; volume expansion was continued with crystalloid solutions. Continued pulsatile bleeding from the injury site was controlled by digital compression. Within 14 minutes after his arrival, the patient was transferred to the operating room with a systolic blood pressure of 90 mm Hg.

Surgical exploration showed that the ICA was transected near the base of the skull. Heparin (10,000 IU) was adminis-

tered intravenously, and a primary repair was attempted unsuccessfully. The artery was ligated proximal and distal to the injury. In the recovery room, the patient was initially alert and moved all four extremities well. Within 30 minutes, however, progressive weakness developed on the right side. Systemic heparinization was continued intravenously (1000 IU/hr), and the blood pressure was elevated by intravenous infusions of blood and crystalloids. Within 3 hours, the patient was markedly hemiparetic but remained alert and moved his left side well.

The neurosurgical service was consulted. On examination, the patient was alert but followed few commands; because he was intubated, his speech could not be tested. His right arm was flaccid, and he had only 2/5 strength in his right leg. Progressive left hemispheric dysfunction due to thromboemboli from the distal portion of the ligated ICA was considered unlikely because systemic heparinization had been continued from the time of the operation. An ischemic deficit due to hemispheric hypoperfusion was postulated.

The patient was immediately placed into a barbiturate coma with intravenously administered pentobarbital (10 mg/kg), and a Swan-Ganz catheter was introduced to monitor pulmonary artery wedge pressure. Dopamine was administered intravenously to maintain the blood pressure. An emergency computed tomogram of the brain, performed to rule out intracerebral hemorrhage, showed no abnormalities. The patient was returned to the operating room. Under continued barbiturate anesthesia (thiopental, 0.5 mg/kg/hr), the left superficial temporal artery was microsurgically anastomosed to a large M2 branch of the middle cerebral artery (MCA) just beyond the M1-M2 junction. Before revascularization, the M1 and M2 vessels had little or no pulsation. After flow through the graft was established, approximately 6 hours after the right hemiparesis was discovered, strong pulsations were detected in these branches, and systemic heparinization was discontinued.

Postoperatively, the patient initially continued to have right arm weakness, but his right leg was stronger. The hemiparesis improved rapidly. At discharge, 6 days after the operation, he could walk and had good strength in his right arm, but still

had moderate paresis of the right hand. His speech and comprehension were intact. At the 6-month follow-up examination, the neurological deficit was stable, and a Doppler ultrasonogram showed that the graft was patent.

DISCUSSION

Diaz et al. (3, 5) and others (7, 16) have shown experimentally that cerebral revascularization can reverse an ischemic deficit caused by occlusion of a major vessel if blood flow can be reestablished within approximately 4 hours. Thereafter, revascularization may result in no improvement or in worsened function, increased cerebral edema, infarction, and hemorrhage (3, 5, 24). The duration of ischemia that can be tolerated may be increased by protecting the brain with mannitol (21, 28), hemodilution (31, 32), or barbiturates (3, 12, 13, 17, 24, 27, 30, 33).

Barbiturates suppress the electroencephalogram (3, 12, 13, 24, 27, 30) and decrease the cerebral metabolic oxygen rate (3, 12, 13, 27, 30), the formation of intracellular free radicals (3, 27, 30), and mitochondrial auto-oxidation (3). Barbiturates appear to reduce stroke size and improve the functional recovery of cerebral tissue after as long as 6 hours of ischemia (12, 13, 24, 27, 30). Barbiturate anesthesia has been used successfully during experimental (12, 13, 24, 27) and clinical (13, 17, 27, 30, 33) cerebral revascularization. Barbiturates also protect the brain during temporary vascular occlusion in the course of aneurysm surgery (27) and carotid endarterectomy (30). Intravenous administration of these compounds often causes systemic hypotension (3, 29), necessitating the use of inotropic agents, such as dopamine, and judicious volume management, guided by monitoring of central venous or pulmonary artery wedge pressure, to support the blood pressure (29).

Gratz et al. (10) reported that cerebral revascularization for evolving stroke caused severe cerebral edema and, in a few cases, hemorrhage. These problems have not been detected in more recent series (2, 4). Diaz et al. (4) used emergency EC-IC bypass to revascularize the brain in eight patients with crescendo transient ischemic attacks, three with evolving stroke, and four with acute stable deficits; the latter four patients came to surgery 4, 6, 9, and 12 hours after the onset of the deficit. In 10 of these 15 patients, the clinical deficits resolved within 10 hours after blood flow was reestablished. Five patients, including two with established stroke, had residual postoperative deficits, which were mild in three of them. Andrews et al. (2) performed emergency EC-IC bypass in 15 patients with evolving stroke. Preoperative angiographic examination showed ipsilateral ICA occlusion in nine patients, ICA stenosis in three, MCA stenosis in two, and MCA occlusion in one. Three patients had an ipsilateral temporoparietal lucency on preoperative computed tomographic scans. Postoperatively, nine patients (60%) had rapid reversal of their neurological deficits; four remained stable, and two continued to deteriorate. No patient had intracerebral hemorrhage or malignant edema postoperatively.

One criticism of EC-IC bypass procedures has been that conventional grafts of the superficial temporal artery to a branch of the MCA on the cortical surface supply only about 25 to 50 ml of blood flow per minute, which might not be expected to provide an adequate collateral blood supply after acute ICA occlusion. As an alternative, Diaz et al. (6) and others have anastomosed the superficial temporal artery or saphenous vein interposition grafts (6, 19, 26) to a main limb or branch of the MCA within the Sylvian fissure. Such grafts may provide higher flow rate and volume (6, 19) than con-

ventional grafts. In our patient, the superficial temporal artery was relatively large in caliber and was therefore anastomosed to a proximal M2 branch of the MCA to maximize graft outflow into the ICA distribution.

The rapid recovery of hemispheric function postoperatively in our patient supports the assumption that the initial hemispheric deficit resulted from ischemia due to hypoperfusion. It remains possible that the ischemia was instead caused by thromboemboli that developed at the time of cervical carotid ligation, despite systemic heparinization, and that the clinical recovery was simply the natural history of stroke in this particular patient. Nevertheless, the neurological deficit began to resolve immediately after completion of the EC-IC bypass. It would therefore appear that in selected cases of acute extracranial carotid ligation or occlusion, symptomatic cerebral ischemia may be treated by quickly reestablishing blood flow with a high-flow EC-IC bypass.

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The Growth of Cerebral Cavernous Angiomas

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The natural history of cavernous angiomas is poorly understood, and their growth has rarely been documented. We report three cases of cavernous angiomas that grew to large size in 6 years, 2 years, and 2 months, respectively. An initial computed tomographic scan disclosed no abnormalities in one patient and demonstrated two "minimal" lesions in the other two. The mechanisms of growth of these lesions are discussed; subsequent hemorrhages and capsule formation played a major role in their enlargement. (*Neurosurgery* 25:92-97, 1989)

Key words: Cavernous angioma, Arteriovenous malformations, Intracerebral hematomas, Magnetic resonance imaging, Computed tomography

INTRODUCTION

The clinical manifestations of cavernous angiomas include seizures, intraparenchymal bleeding, and progressive neurological deficit (17). Their biological behavior is not completely understood, however (21). In particular, little is known about the progression of their growth and changes in size with time, since only a few cases have been followed up prospectively. In the last 13 years, we have treated 40 patients with cerebral cavernous angiomas. Progressive tumor growth was docu-

mented in 3 patients and constitutes the origin of this report. These cases seem to demonstrate a surprisingly dynamic character of this type of lesion.

CASE REPORTS

Case 1

In 1982, this 32-year-old woman complained of sudden headache and vomiting, which were treated at home by her personal physician; these symptoms subsided almost com-