Traumatic Dissection of the Internal Carotid Artery Treated by ECIC Anastomosis

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SUMMARY A patient with traumatic dissection of the left internal carotid artery (ICA) was treated by extracranial-intracranial (ECIC) anastomosis. Anastomosis was performed because the near total occlusion of the left ICA was associated with a cerebral transient ischemic attack. At surgery, an embolus was found in a cortical branch of the middle cerebral artery and was removed at the time of the ECIC. Traumatic dissection of the ICA is briefly reviewed.

INTERNAL CAROTID ARTERY (ICA) dissection following blunt trauma to the head and neck is a well described, but uncommon entity. The patient, usually a young male involved in a motor vehicle accident, presents with hemispheric symptoms after a lucid interval of hours to days. Ultimate outcome and treatment are highly variable. We describe a patient with traumatic ICA dissection, who was treated by extracranial-intracranial (ECIC) anastomosis and a middle cerebral artery embolectomy.

S.B., a 20-year-old, right-handed male was admitted to the Neurosurgery Service at the University of North Carolina because of suspected left internal carotid artery occlusion. He was in good health until August 19, 1978, when he was involved in a motor vehicle accident. He was riding in the passenger’s seat during a low speed, front end collision. On the collision he struck his head and neck on the steering wheel and dashboard. Following an unconscious interval of 15-20 minutes, he recovered in a local hospital. No focal neurologic deficits were recorded, but he did complain of persistent headache and left neck pain. A dynamic radionuclide brain scan demonstrated decreased flow on the left side, and he was transferred to the University of North Carolina for further evaluation. His medical history was complicated by a Wolfe-Parkinson-White syndrome which had occasionally caused supraventricular tachycardia.

Examination revealed an alert, healthy, young male with minimal tenderness of the left neck anteriorly, but no apparent trauma. There were no bruits and the neurological examination was normal. Admission laboratory studies and plain skull and cervical spine radiographs were normal. Non-invasive vascular studies were then carried out. Carotid phonoangiography (CPA) did not demonstrate a bruit on either side, while Kartchner-McRae occuloplethysmography (OPG) showed a differential pulse skewed to the left. These indicated a left carotid stenosis of 85-100%. The Gee OPG showed a significant decrease in the left ophthalmic artery pressure relative to the right and a significantly decreased ophthalmic artery/brachial artery pressure ratio. These findings prompted a transfemoral bilateral carotid arteriogram, which revealed an 80% left ICA stenosis secondary to dissection, beginning 3 centimeters distal to the bifurcation of the common carotid artery and extending to the base of the skull (fig. 1). As the patient remained asymptomatic, he was discharged the following morning. That evening (10 days post trauma), he experienced a syncopal spell lasting a few minutes following which he could not speak to, but apparently understood his father.

He was readmitted to the Neurosurgery Service approximately one hour later, at which time he was again without neurological deficit. Repeat carotid arteriography demonstrated 99% occlusion of the left ICA (fig. 2). There was enlargement of the superficial temporal artery (STA) compared with previous studies and cross filling of the left ICA from the right ICA.

We believed that his symptoms were of ischemic origin due to hypoperfusion secondary to the nearly total obstruction of the left ICA. Two days later a left superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis was performed. At surgery, an embolus was found in a separate cortical branch of the ICA (fig. 2) and was removed directly through an arteriotomy. The patient's postoperative course was marked by transient hypoxemia, but there were no further neurological symptoms. He was discharged 8 days after surgery, with a normal neurological examination. He was readmitted a month later for follow up angiography. The neurological examination was normal, and the angiogram demonstrated approximately 60% stenosis of the ICA with good function of the STA-MCA anastomosis (fig. 3). Further angiography 7 months later, revealed only minimal stenosis 5% (fig. 4) of the ICA but continued function of the anastomosis. His neurological examination remained normal.

Discussion

Dissection of the ICA caused by non-penetrating trauma has become a well described, but uncommon clinical syndrome. Approximately 130 patients have been described in the literature, the first in 1872. The syndrome was initially recognized as a postmortem finding; with the increasing use of angiography in patients with head trauma more have been
diagnosed antemortem, although only rarely before the angiogram. The “classical” presentation is that of a young male involved in a motor vehicle accident in which he has sustained head and neck trauma of varying severity. After a short symptom-free interval, hemispheric symptoms develop with relative sparing of the patient’s level of consciousness, a cardinal feature in the diagnosis.

As with many syndromes, however, most patients are variants on the general theme. The age range is wide — 4 to 72 years — and women certainly may sustain head and neck trauma. Approximately 75% of patients have a history of head injury,10 25% of them being considered “serious.”3,10 Only half of all patients with traumatic ICA dissection have evidence of neck trauma.10 The symptom-free period may vary from under an hour to 2 weeks, but the majority occur in less than 24 hours.5–10 A small number of patients present with transient ischemic attacks (TIA).4,10 Symptoms vary in severity from isolated transient aphasia or hemiparesis to coma and hemiplegia. An early review noted a high morbidity (50%) and mortality (40%),10 but if those patients diagnosed only at autopsy are excluded, the mortality drops to 30%.5 More recently, rates of 20–30% have been reported8,11 and one small series noted no mortality.8

The diagnosis of traumatic ICA dissection is made angiographically, although, as noted above, it is usually the fortuitous finding of angiography in a head-injured patient who is deteriorating. This is one situation in which angiography is still preferable to computerized tomographic (CT) brain scanning in the head-injured patient; dependence solely on the CT scan can lead to a delay in the diagnosis. A high index of suspicion is desirable when one is confronted with a patient who has a marked motor deficit yet normal or nearly normal level of consciousness. Regardless of how the diagnosis is made, angiography is required for definitive delineation of the lesion. It should include demonstration of both extracranial and intracranial carotid vessels to determine the specific hemodynamics, whether there are emboli, and the size of the STA if one is contemplating an ECIC. Arteriography usually shows one of the 2 radiographic pictures: (1) partial or complete occlusion of the ICA beginning 1–3 centimeters distal to the common carotid artery bifurcation and extending as far as the base or carotid siphon; (2) a smaller number of patients have occlusion in the carotid canal, usually associated with evidence of a basilar skull fracture. Occasionally one may see an intimal flap angiographically.

The anatomic findings at surgery or autopsy correlate well with the angiographic picture, in that one of 3 types of lesions is present. Most often there is an intimal tear with hemorrhage into the subjacent media,4 with a variable degree of submedial dissec-

FIGURE 1. The initial left common carotid artery arteriogram showing a long area of stenosis beginning approximately 3 cm. distal to the bifurcation and extending to the base of the skull.

FIGURE 2. The second left common carotid arteriogram demonstrating nearly complete occlusion of the internal carotid artery and the superficial temporal artery to be of sufficient size for an ECIC.
Several theories exist on the pathogenesis of traumatic ICA occlusion, and there is evidence to support each in specific instances. Intimal tears may be caused by a direct blow, stretching of the artery, compression at the atlas, fracture of a pre-existing atheroma or disruption of the artery in the bony canal. An intimal tear may be caused by a direct blow to the artery, usually to the anterolateral portion of the neck, but occasionally this may be due to blunt intracranial trauma. Stretching an artery along its long axis has been shown to cause intimal disruption. Hyperextension and lateral rotation of the neck stretches the artery and probably accounts for most instances where there is no direct neck injury. Stretching is further enhanced by compression of the upper thorax, which fixes the artery at the thoracic outlet. Compression of the artery at the atlas or disruption of a pre-existing atheroma can exacerbate injury caused by either a direct blow or stretch. Approximately 10-15% of patients have had pre-existing atheromatous disease of the ICA, which became disrupted with trauma. Radiologic and pathologic evidence may support basilar skull fracture as a cause of ICA occlusion in the bony canal.

Treatment has varied considerably and is difficult to assess because the natural history of the disease is impossible to predict. Some patients are treated with general supportive measures. Anticoagulation, fibrinolysis, thromboendarterectomy, resection of the damaged portion of the artery with vein patch graft, ligation, and ECIC have all been reported. Ours is the first case of direct intracranial embolectomy reported and brings to 4 the number treated by ECIC.

The small number of patients treated in any manner allows no definitive statements regarding therapy. We believe that patients with ischemic symptoms felt secondary to hypoperfusion resulting from the high grade stenosis or occlusion without completed stroke, should have revascularization in an effort to prevent progression of the ischemic brain lesion to frank...
necrosis. If it is believed that the ischemic symptoms are secondary to emboli from the damaged carotid, then the patient should be anticoagulated. With completed stroke and necrosis of brain tissue, revascularization or anticoagulation is dangerous because of the possibility of converting an ischemic infarct into a hemorrhagic one. The 3 patients treated by ECIC described by Gratzl, et al,21 had no significant change in their course postoperatively as compared with their preoperative status; nor did the present patient. However, as with patients who have TIA's due to atherosclerotic occlusion of the ICA, the purpose of the surgery is to prevent progression of brain dysfunction and not to reverse it.

It is well documented in both traumatic occlusion of the ICA and stroke that an occluded cerebral artery may become revascularized. The efficacy of STA-MCA anastomosis has been questioned in light of this. It is believed that the anastomosis will soon thrombose when it is no longer the primary supply of blood to the region. The continued function of the anastomosis in this patient with a minimal stenosis of the carotid artery is noteworthy.

The finding of an embolus in one of the large middle cerebral cortical arteries, suggests that some of this patient's symptoms were related more to the embolus than to hypoperfusion secondary to stenosis. Documentation of an embolus in the middle artery is noteworthy because it prompts the use of anticoagulation as treatment. Anticoagulation has been successfully employed in other patients; 4,10 and a patient with post-traumatic TIA with angiographically documented embolization should be treated initially with anticoagulation. In this patient, the progression of internal carotid artery stenosis from 80% to 99% over a 48 hour period suggested that the stenosis, rather than embolization, was most likely the cause of symptoms occurring at this time. Because of this, an ECIC was carried out.

References
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