Endovascular Stent-Assisted Angioplasty in the Management of Traumatic Internal Carotid Artery Dissections

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Background and Purpose—The prognosis of traumatic dissection of the internal carotid artery is worse than for spontaneous dissections. Rapid stenting followed by antiplatelet therapy may prevent complications when anticoagulation therapy is not applicable.

Methods—Patients with angiographically proven traumatic carotid artery dissection and hemodynamic significant hemispheric hypoperfusion, or in whom anticoagulant therapy was either contraindicated or failed clinically, were regarded as being at high risk for stroke and were selected for stenting.

Results—Ten patients with traumatic dissection underwent stenting. Endovascular treatment reduced mean dissection stenosis from 69% to 8%. During a mean clinical follow-up time of 16 months, none had additional transient ischemic attacks or stroke. Doppler ultrasound studies did not detect any signs of de novo in-stent stenosis.

Conclusion—In selected cases of traumatic carotid artery dissections, endovascular stent-assisted angioplasty immediately restored the integrity of the vessel lumen and prevented efficiently the occurrence of new ischemic events, without additional anticoagulation. (Stroke. 2005;36:e45-e47.)

Key Words: carotid arteries ■ stents ■ stroke, ischemic ■ trauma

Traumatic dissection of the internal carotid artery is an uncommon but well-recognized entity.1,2 In the clinical set-up of neurotrauma it is easily missed because of multiple factors contributing to the neurological deficit. The delayed appearance of focal neurological signs, unexplainled by blood collection, or the evolution of ischemic changes on brain imaging should raise the suspicion of dissection. In cases of spontaneous dissection, anticoagulant treatment is commonly used to prevent thromboembolic strokes. However, anticoagulation may be contraindicated in patients with multiple trauma, intracranial hematomas, and penetrating injuries. Also, 40% of traumatic dissections and the majority of pseudoaneurysms do not heal with anticoagulant therapy and constitute a long-term risk of embolization or flow-related complications.3–4

Stenting of carotid arteries by an endovascular approach may provide immediate revascularization and avoids the need of anticoagulation.5–6 A major issue remains on the selection of patients who will benefit most from this procedure. We report our protocol for patient selection for endovascular stenting in the treatment of 10 patients presenting with traumatic carotid artery dissections.

Materials and Methods

All head trauma patients underwent routine noncontrast brain computed tomography. Patients with neurological signs or radiological evidence of injury or with penetrating injury were hospitalized. Cervical and cerebral angiography was performed after appearance of: (1) focal neurological signs that were not explained by the immediate impact or by an intracranial hemorrhage; (2) Horner syndrome or lower cranial neuropathy; and (3) in all patients with penetrating neck injuries. The criteria for placement of stent were: (1) clinical failure of anticoagulation (repetitive transient ischemic attacks, fluctuating neurological signs, or neurological deterioration despite anticoagulation); (2) contraindication for anticoagulation, because of traumatic intracranial or systemic hemorrhagic lesions; and (3) impending stroke, caused by hemodynamically significant dissection with parenchymal hypoperfusion, indicated by lack of parenchymal “blushing” during the capillary phase of angiography, or in the presence of perfusion/diffusion mismatch on magnetic resonance imaging.

Patients with a large infarction on computed tomography (>50% middle cerebral artery territory), large defect on diffusion-weighted imaging (>50% middle cerebral artery territory), or patients with longstanding (>2 weeks) established severe neurologic deficit were excluded from this study. Patients with concomitant vertebral artery dissection, iatrogenic or intracranial dissection, or with contraindication for anti-aggregation therapy were excluded as well.

All patients were kept on aspirin (325 mg/d) and clopidogrel (75 mg/d) for 3 months. Neurologic and neuroradiologic examinations were performed at discharge, 1 month, and 6 months. Evaluation of stent patency was assessed by Doppler ultrasound.

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Results

During 2.5 years, 10 out of 205 patients (4.9%) admitted after head and neck injuries were prospectively selected for endo-
vascular stent-assisted arterial reconstruction. There were 8 men and 2 women (mean age 42.7±13.8 years; range, 17 to 62). Six patients had multiple traumas, and 4 had cranio-cervical trauma. Five patients had major brain trauma. The dissection presented clinically with ischemic stroke in 4 patients, repetitive transient ischemic attacks in 2, local symptoms in 2 (carotidynia, Horner syndrome), and 2 patients were screened because of cervico-cranial penetrating injuries. The clinical manifestations of dissection appeared 4 hours to 19 days (mean, 4.52 days) after the acute injury. Stenting was performed because of parenchymal hypoperfusion and impending stroke (5 patients), neurologic deterioration under anticoagulation (2 patients), and because of contraindication for anticoagulation (6 patients). The interval between the onset of focal ischemic signs and stenting ranged from 2 to 10 hours (mean, 6.1±4.7 hours). Twenty-two stents were implanted (7 patients with multiple stents). Dissection-related stenoses improved from 69±31% (range, 10% to 100%) to 8±9% (range, 0% to 20%). There were no procedure-related complications. Six patients improved and 4 remained stable after 16 months of follow-up (range, 8 to 28 months). There was no evidence of in-stent de novo stenoses or stent thrombosis by sonographic follow-up at 7 to 28 months after trauma. No patient had to discontinue antiplatelet therapy because of side effects.

Three illustrative cases represent the typical circumstances for stenting in traumatic dissections: 1 patient with symptomatic subocclusive carotid dissection complicated by intracranial emboli (Figure A and B); 1 patient with recurrent transient ischemic attacks under anticoagulation (Figure C and D); and 1 patient with multiple emboli secondary to carotid dissection and contraindication for anticoagulation (Figure E to G).

Discussion

Multiple mechanisms of neural injury act in concert in the traumatized brain, including axonal shearing, hemorrhages, and increased intracranial pressure. Consequently, the traumatized brain is especially vulnerable to ischemia after emboli or hemodynamic compromise. Moreover, the lack of collateral blood supply after acute occlusions, as compared with patients with longstanding atherosclerotic stenosis and the loss of vascular autoregulation in the injured brain, reduces its ability to compensate for hypoperfusion. Thus, although spontaneous carotid dissection is considered relatively benign, the mortality rate and severity of neurologic deficit are high in traumatic dissections, as in abrupt internal carotid artery (ICA) occlusion. Anticoagulation is usually practiced in dissections to prevent thromboembolism. Because patients with multiple trauma and penetrating injuries frequently have associated hematomas or are at high risk for bleeding, anticoagulation may be contraindicated. Therefore, we obtained revascularization by stenting when there was clinical and/or radiographic indication for impending stroke, or in stroke in-evolution. The identification of thromboembolic occlusion of cerebral vessels and of hemorrhage is significant reduced perfusion on the angiographic capillary phase (or by perfusion/diffusion mismatch on magnetic resonance imaging) provided the rationale for selecting patients that would benefit from stenting. There were no complications related to the procedures and the clinical outcome was favorable.
In conclusion, endovascular stenting seems to be a rationale and effective way to restore the artery lumen in selected cases of acute traumatic carotid dissections.

References
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