

Endovascular Stenting for the Treatment of Traumatic Internal Carotid Injuries: Expanding Experience

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Background: The role of endovascular techniques in the treatment of traumatic vascular injuries, including injury to the internal carotid artery, continues to evolve. Despite growing experience with the usage of these techniques in the setting of atherosclerotic disease, published results in traumatic carotid injuries remain sporadic and confined to case reports and case series.

Methods: We conducted a review of the medical literature from 1990 to the present date using the Pubmed and OVID Medline databases to search for all reports documenting the use of endovascular stenting for the treatment of carotid injuries. Thirty-one published reports were analyzed to abstract data regarding

mechanism, location, and type of injury; use and type of anticoagulation used in conjunction with stenting; type and timing of radiographic and clinical follow-up; and radiographic and clinical outcomes.

Results: The use of endovascular stenting for the treatment of internal carotid injuries was reported for only 113 patients from 1994 to the present date. Stenting was most commonly used after a blunt mechanism of injury (77.0%). The injury types treated by stenting included pseudoaneurysm (60.2%), arteriovenous fistula (16.8%), dissection (14.2%), partial transection (4.4%), occlusion (2.7%), intimal flap (0.9%), and aneurysm (0.9%). Initial endovascular stent placement was successful in 76.1% of patients. Radio-

graphic and clinical follow-up periods ranging from 2 weeks to 2 years revealed a follow-up patency of 79.6%. No stent-related mortalities were reported. New neurologic deficits after stent placement occurred in 3.5%.

Conclusion: Endovascular treatment of traumatic internal carotid artery injury continues to evolve. Early results are encouraging, but experience with this modality and data on late follow-up are still very limited. A large prospective randomized trial is warranted to further define the role of this treatment modality in the setting of trauma.

Key Words: Trauma, Carotid injury, Stenting.

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The management of traumatic injuries of the internal carotid artery has traditionally required operative intervention. The surgical approach for carotid repair may be complex, particularly with base of skull lesions where obtaining proximal and distal control may result in significant morbidity and mortality.¹

The emergence of endovascular techniques offers an alternative to traditional surgical management of select carotid lesions. Initially designed for the treatment of intracranial and high extracranial lesions, endovascular stents have seen expanded use at other extracranial locations. Published experience to date, however, remains limited. Reports of successful endovascular treatment of traumatic internal carotid injuries remain confined to case reports and small series documented in the medical literature. Our study was designed to summarize the experience to date with endovascular stent-

ing for these injuries through a review of the available medical literature.

METHODS

We conducted a systematic review of the English-speaking medical literature using the PubMed (www.pubmed.gov, accessed 25 August 07) service of the National Library of Medicine/National Institutes of Health and OVID Medline databases (Copyright © 2000–2007 Ovid Technologies, Version: rel10.5.1) to identify all case reports carotid artery stenting after traumatic carotid artery injuries. A multidisciplinary group of surgeons, intensivists, and biostatistician used the following criteria to select studies to be included for analysis: adequate information regarding mechanism, location, and type of injury; use and type of anticoagulation used in conjunction with stenting; type and timing of radiographic and clinical follow-up; radiographic and clinical outcomes.

RESULTS

Fifty-two published reports with endovascular stenting for the treatment of traumatic internal carotid injuries were identified. Twenty-one of these publications lacked sufficient information for inclusion and were excluded, leaving 31 published case reports or case series available for review over a time the study period time of 1994 to 2007.^{2–32}

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Table 1 Description of Published Studies Documenting Experience With Endovascular Stenting of Carotid Injuries

| Year | Study | No. Patients | Type of Injury (n) | Gender (n) | Age | Injury Location (n) | Mechanism (n) |
|------|--|--------------|---|--------------|-------------|--------------------------------|----------------------------|
| 1997 | Duke et al. <i>J Neurosurg.</i> | 6 | Partial transection (3), pseudoaneurysm (3) | M (3) F (3) | Avg 26 | Unilateral (5), bilateral (1) | Blunt (6) |
| 1997 | Matsuura et al. <i>J Endovasc Surg.</i> | 1 | Pseudoaneurysm (1) | F | 20 | Unilateral (1) | Blunt (1) |
| 1997 | Klein et al. <i>AJNR Am J Neuroradiol.</i> | 1 | Aneurysm (1) | F | 30 | Unilateral (1) | Penetrating (1) |
| 1997 | Perez-Cruet et al. <i>Neurosurgery.</i> | 1 | Pseudoaneurysm (1) | M | 20 | Unilateral (1) | Blunt (1) |
| 1997 | Bernstein et al. <i>J Vasc Interv Radiol.</i> | 1 | Pseudoaneurysm (1) | F | 20 | Unilateral (1) | Blunt (1) |
| 1998 | Reiter et al. <i>J Vasc Interv Radiol.</i> | 1 | Pseudoaneurysm (1) | M | 72 | Unilateral (1) | Penetrating (1) |
| 1999 | Shames et al. <i>J Trauma.</i> | 1 | Pseudoaneurysm (1) | M | 29 | Unilateral (1) | Blunt (1) |
| 1999 | Liu et al. <i>Neurosurgery.</i> | 2 | Dissection (1), pseudoaneurysm (1) | M (1) F (1) | Avg 15 | Unilateral (2) | Blunt |
| 1999 | Parodi et al. <i>Ann Vasc Surg.</i> | 3 | Pseudoaneurysm (2), fistula (1) | M (3) | Avg 40 | Unilateral (3) | Penetrating (2), blunt (1) |
| 2000 | Coldwell et al. <i>J Trauma.</i> | 14 | Pseudoaneurysm (14) | M (7) F (7) | Avg 27 | Unilateral (12), bilateral (2) | Blunt (14) |
| 2000 | Kerby et al. <i>J Trauma.</i> | 1 | Intimal flap (1) | F | 37 | Bilateral (1) | Blunt (1) |
| 2000 | Malek et al. <i>J Neurosurg.</i> | 3 | Occlusion (3) | F (3) | Avg 34 | Unilateral (1), bilateral (2) | Blunt (3) |
| 2000 | Malek et al. <i>AJNR Am J Neuroradiol.</i> | 2 | Dissection (2) | F (2) | Avg 41 | Unilateral (2) | Blunt (2) |
| 2001 | Brandt et al. <i>J Trauma.</i> | 2 | Pseudoaneurysm (1), partial transection (1) | F (2) | 44 | Bilateral (1), unilateral (1) | Blunt (2) |
| 2001 | Redekop et al. <i>J Neurosurg.</i> | 6 | Fistula (2), pseudoaneurysm (4) | M | Avg 19 | Unilateral (6) | Blunt (2), penetrating (4) |
| 2001 | Scavee et al. <i>Cardiovasc Intervent Radiol.</i> | 1 | Pseudoaneurysm (1) | M (1) | 53 | Unilateral (1) | Blunt (1) |
| 2002 | Patel et al. <i>Clin Radiol.</i> | 1 | Pseudoaneurysm (1) | M | 29 | Unilateral (1) | Penetrating (1) |
| 2002 | Duane et al. <i>J Trauma.</i> | 2 | Pseudoaneurysm (1), unknown (1) | F (2) | Avg 58 | Unilateral (2) | Blunt (1), penetrating (1) |
| 2002 | McNeil et al. <i>J Vasc Surg.</i> | 1 | Pseudoaneurysm (1) | M | 18 | Unilateral (1) | Penetrating (1) |
| 2003 | Duncan et al. <i>J Endovasc Ther.</i> | 1 | Fistula (1) | M | 22 | Unilateral (1) | Penetrating (1) |
| 2003 | Kubaska et al. <i>J Endovasc Ther.</i> | 2 | Pseudoaneurysm (2) | M (2) | Avg 40 | Unilateral (2) | Penetrating (2) |
| 2004 | Lee et al. <i>J Neurosurg.</i> | 1 | Fistula (1) | M | 19 | Unilateral (1) | Blunt (1) |
| 2004 | Self et al. <i>J Trauma.</i> | 1 | Pseudoaneurysm (1) | M | 32 | Unilateral (1) | Penetrating (1) |
| 2004 | Layton et al. <i>AJNR Am J Neuroradiol.</i> | 1 | Pseudoaneurysm (1) | M | 23 | Unilateral (1) | Penetrating (1) |
| 2004 | Fusonie et al. <i>Ann Vasc Surg.</i> | 1 | Pseudoaneurysm (1) | M | 37 | Unilateral (1) | Blunt (1) |
| 2005 | Cothren et al. <i>Arch Surg.</i> | 23 | Pseudoaneurysm (23) | M (15) F (8) | Avg 32 | Bilateral (1), unilateral (22) | Blunt (23) |
| 2005 | Fateri et al. <i>Ann Vasc Surg.</i> | 1 | Partial transection (1) | M | 52 | Unilateral | Penetrating (1) |
| 2005 | Joo et al. <i>J Trauma.</i> | 10 | Fistula (5), pseudoaneurysm (5) | M (9) F (1) | Avg 36 | Unilateral (10) | Blunt (9), penetrating (2) |
| 2005 | Cohen et al. <i>Neurol Res.</i> | 12 | Dissection (12) | Unk (12) | Avg 41 | Unilateral (12) | Blunt (12) |
| 2005 | Szopinski et al. <i>Euro J Vasc Endovasc Surg.</i> | 2 | Pseudoaneurysm (1), dissection (1) | M (1) F (1) | Avg 46 | Unilateral (2) | Blunt (2) |
| 2007 | Archondakis et al. <i>AJNR Am J Neuroradiol.</i> | 8 | Fistula (8) | M (5) F (3) | Range 14–70 | Unilateral (8) | Unknown (8) |

The use of endovascular stenting after carotid artery injury was described in 113 patients (Table 1), 57.5% of which were male. Age ranged from 12 years to 72 years. Although 92.9% underwent attempt at endovascular treatment of unilateral lesions, 7.1% bilateral lesions were also attempted. The majority of injuries stented were completely extracranial lesions of the internal carotid (85.8%), with

14.2% demonstrating an intracranial component. Stenting was most commonly used after a blunt mechanism of injury (77.0%). Injuries treated included pseudoaneurysm (60.2%), arteriovenous fistula (16.8%), dissection (14.2%), partial transection (4.4%), occlusion (2.7%), intimal flap (0.9%), and aneurysm (0.9%) (Table 2). Successful endovascular stent placement, defined as complete occlusion of the lesion with

Table 2 Types of Carotid Injuries Treated With Stenting

| Injury Type | n | Percent |
|---------------------|-----|---------|
| Pseudoaneurysm | 68 | 60.2 |
| A-V fistula | 19 | 16.8 |
| Dissection | 16 | 14.2 |
| Partial transection | 5 | 4.4 |
| Occlusion | 3 | 2.7 |
| Aneurysm | 1 | 0.9 |
| Intimal flap | 1 | 0.9 |
| Total | 113 | 100.0 |

restoration of normal blood flow at the conclusion of the initial procedure, occurred in 76.1% of patients treated. Initial endovascular attempt at restoration of flow was unsuccessful in 17.8%, and was not adequately documented in 6.2%.

Adjunctive anticoagulation after stent placement was used in 89.4% of patients. Most commonly (55.8%) antiplatelet agents were used, although heparin (15.9%), warfarin (17.7%), and a combination of agents (1.8%) were also commonly used (Table 3). The type of anticoagulation could not be adequately discerned from review of the literature in 8.8% of published case reports or series.

After stent placement, 88.5% of patients underwent documented radiographic follow-up. In most instances, this follow-up was achieved through the use of angiography (61.9%) to document stent patency. Alternative imaging modalities used for this purpose included color-flow Doppler (21.2%), CT angiography (4.4%), and magnetic resonance angiography (0.9%) (Table 4). Radiographic follow-up periods ranged from 2 week to 2 years, revealing stent patency in

Table 3 Adjunctive Anticoagulation After Carotid Artery Stenting

| Anticoagulation | n | Percent |
|-----------------|-----|---------|
| Antiplatelet | 63 | 55.8 |
| Coumadin | 20 | 17.7 |
| Heparin | 18 | 15.9 |
| Unknown | 10 | 8.8 |
| Combination | 2 | 1.8 |
| Total | 113 | 100.0 |

Table 4 Follow-up Modalities of Carotid Artery Stenting

| Follow-up Modalities | n | Percent |
|------------------------|-----|---------|
| Angiography | 70 | 61.9 |
| Color Doppler | 24 | 21.2 |
| Unknown | 6 | 5.3 |
| Clinical | 6 | 5.3 |
| CT Angio | 5 | 4.4 |
| MRA | 1 | 0.9 |
| Angiography and duplex | 1 | 0.9 |
| Total | 113 | 100.0 |

Table 5 Outcomes After Carotid Artery Stenting

| | n | Percent |
|------------------------------------|-----|---------|
| Stent-related outcome | | |
| Patent | 90 | 79.6 |
| Occlusion | 11 | 9.7 |
| Leak | 6 | 5.3 |
| Unknown | 3 | 2.7 |
| Stenosis | 1 | 0.9 |
| Unsuccessful attempts | 1 | 0.9 |
| AV Fistula | 1 | 0.9 |
| Clinical outcome | | |
| Alive, without neurologic sequelae | 106 | 93.8 |
| Alive, with neurologic sequelae | 4 | 3.5 |
| Unknown | 2 | 1.8 |
| Died | 1 | 0.9 |

79.6%. Imaging also revealed occlusion in 9.7% and leak in 5.3% of initially successful stent placements (Table 5).

Neurologic outcomes were adequately documented in 111 patients. After successful placement of an endovascular stent for treatment of carotid artery trauma, 93.8% patients remained alive without new neurologic sequelae because of stent placement at a range of follow-up from 2 weeks to 2 years. New neurologic deficits after stent placement occurred in 3.5% (Table 5). Mortality occurred in only one patient, as a result of multisystem organ failure because of severe multiple injuries for a survival of 99.1%.

DISCUSSION

The treatment and outcome of traumatic carotid injuries are influenced by many factors; including the mechanism, type of injury, and associated neurologic function. Blunt carotid injuries, although rare, have been associated with mortality rates of 20% to 40% and permanent neurologic impairment in 40% to 80%.³³⁻³⁵ Penetrating injuries, occurring in approximately 6% of penetrating neck trauma, accounting for 22% of all penetrating cervical vascular injuries,³⁶ and are associated with a mortality rate ranging from 6.6% to 33% with an average of 17%.³⁷ The prognosis for penetrating mechanisms has also been clearly linked with neurologic status at admission.³⁷⁻⁴⁰

Several types of injuries may result from carotid trauma, regardless of mechanism. Those that do not commonly result in the operative indications of hemorrhage or expanding hematoma include intimal flaps, dissections, and pseudoaneurysms. The natural history and appropriate management of these injuries remains ill-defined. Anticoagulation after blunt carotid injury is now known to be associated with improved outcome after blunt trauma,⁴¹ but some types of injuries are more likely to fail conservative therapy. Although some small intimal injuries due to blunt mechanism will respond well to conservative approaches including anticoagulation, Panetta et al.⁴² have demonstrated that only up to a third of these types of injuries resolve without subsequent complication. Similar

observations regarding the natural the course of dissections treated in this fashion have also been observed, with Fabian et al.⁴¹ noting that 29% of these types of injuries progress to pseudoaneurysm on repeat imaging. Pseudoaneurysms themselves classically fail to resolve with anticoagulation alone and constitute continued risk for embolic stroke if not addressed more aggressively.^{41,43,44}

The approach to carotid injuries requiring more aggressive intervention has evolved significantly during the past 60 years. Ligation remained the most widely practiced treatment of penetrating carotid artery injuries through both World War I and II, with an associated mortality rate of 40% to 47% and a cerebral complication rate of 30%. During the Vietnam and Korean conflicts, however, arterial repair became more commonplace and resulted in a decrease in morbidity to 15%.³⁶ In the modern era, surgical repair of carotid artery injuries is associated with mortality rates of 0% to 22% and postoperative progression of neurologic deficit of 0% to 21%.^{38,39,45–55}

Endovascular approaches to carotid injuries have seen increasing utilization. Borrowing on the expanding experience with the use of endovascular stents for cerebrovascular disease,⁵⁶ stenting has most commonly been used for high extracranial internal carotid lesions.⁵⁷ These types of interventions are ideally suited for this region, where surgical approaches are most difficult, and are associated with a high rate of local and cerebrovascular complications.^{1,43,54} An endovascular approach may also prove particularly useful in the treatment of select types internal carotid injuries, as surgical resection or repair of internal carotid pseudoaneurysms in particular, are associated with a high mortality rate (30%) and high incidence of cerebral complications.^{43,54}

Compared with surgical treatment of carotid injuries, with an associated mortality rate of up to 22%, carotid stenting appears to be much lower at 0.9%. In addition, stroke rates associated with carotid stenting of trauma, at 3.5%, appear comparable to those after operative intervention for carotid injuries (0–21%).^{38,39,45–55} As well as stroke rates after elective stenting for cerebrovascular disease (4.7%).⁵⁸

There are several unique complications to be considered when using endovascular approaches. Local access site complications after these types of percutaneous interventions have been shown to occur in 3% of elective cases in the treatment of cerebrovascular disease.⁵⁹ Several other factors may also adversely affect the placement and patency rates of these devices. Technical inexperience and anatomic difficulties may preclude effective placement. Redekop et al.¹² have shown that small vessel size, proximal or distal dissection, and under dilation of the stent have all been associated with a higher probability of carotid stent thrombosis after placement. Additionally, as no device is currently FDA approved for this indication, the limitations of available stents types that may be used for these approaches remain largely unknown. Even if these devices are effectively placed for initial treatment, no consensus agreement as of yet exists to provide guidance for the need and type of adjunctive anticoagulation

that should be used or ideal type or interval for subsequent follow-up.

Despite these uncertainties, the role of endovascular stenting after carotid trauma warrants further investigation. Unresolved issues facing this emerging technology include the adequate definition of the types of injuries ideally indicated for endovascular approach. The need for emergent operation in many penetrating carotid injuries, for example, confounds the ability to appropriately compare outcomes after these mechanisms of injury. Better definition of the optimal blunt injuries likely to benefit is, likewise, lacking. The ideal adjunctive anticoagulation regimens and appropriate follow-up protocols must also be defined. Documentation of long-term outcomes also remains among the most important concerns, particularly given the relative young age of the patients for whom these devices are used. From initially published reports, endovascular treatment appears to have comparable stroke rates and lower associated mortality compared with traditional operative approaches. Comparisons between patients requiring operative intervention and those undergoing stenting are problematic, however, because these populations may represent groups that are not similar. For all of these reasons, further prospective analysis of the role for endovascular treatment of carotid injuries is warranted.

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