Carotid Angioplasty and Stenting under Embolic Protection Device: Experience in Department of Neurology, Ain-Shams University

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ABSTRACT

Background: Carotid endarterectomy is more effective than medical management in the prevention of stroke in patients with severe symptomatic or asymptomatic atherosclerotic carotid artery stenosis. Stenting with the use of embolic protection device is emerging as an effective and less invasive revascularization strategy than endarterectomy in carotid artery disease. Methods: we performed carotid angioplasty and stenting under embolic protection device (EPD) for patients with significant carotid stenosis (>70% symptomatic and >80% asymptomatic carotid stenosis). All patients underwent clinical assessment together with National Institute Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS). This was done before stenting, within 24 h, one week, one month, 3 months, 6 months and one year after stenting. Patients also performed carotid duplex ± MRA neck (before stenting, 6 months and one year after stenting), MRI brain, MRA brain and echocardiography. Results: We included our first 20 patients with significant carotid stenosis for whom carotid angioplasty and stenting under embolic protection device was done (21 stents for 20 patients). Fourteen patients had symptomatic carotid stenosis (66.6%) while 6 patients had asymptomatic carotid stenosis. The mean degree of stenosis based on angiography was 83.9±7.9%. We succeeded to pass the embolic protection device distal to the stenosis prior to predilatation or stenting and to deploy our stent in the adequate position in all our cases and there was no need to use a second adjunctive stent (21/21; 100%). This resulted in successful revascularization in all our patients (<30% residual diameter stenosis). One patient developed a TIA (1/21 procedure; 4.7%) while none of our patients developed minor or major stroke or intracranial hemorrhage. One patient (4.7%) developed a myocardial infarction. During long-term follow-up (mean 16 months, max 34 months), none developed recurrent TIA or stroke while one patient died of myocardial infarction (1 year after carotid stenting). Concerning re-stenosis at 1 year post-stenting, none out of nine patients showed re-stenosis based on carotid duplex in 7 and angiography in 2. Conclusion: carotid angioplasty and stenting under EPD seems to be a less invasive alternative and at least not inferior to carotid endarterectomy. (Egypt J. Neurol. Psychiat. Neurosurg., 2007, 44(1): 193-206).

INTRODUCTION

Stroke is the third most common cause of death in the United States. There are approximately 700,000 strokes/year; 80% are ischemic, and 20-30% of ischemic strokes are secondary to carotid disease. In a study performed on 100 Egyptian patients with acute stroke, 16% had cervical internal carotid artery (ICA) stenosis >50%. Carotid endarterectomy (CEA) has high efficacy in stroke prevention in selected patients with symptomatic carotid stenosis. Randomized clinical trials demonstrated that carotid endarterectomy reduces the stroke risk, compared to medical therapy alone, for patients with 70-99% symptomatic stenosis with 16% absolute risk reduction at 5 years. The benefit for patients with 50-69% symptomatic stenosis is lower i.e. absolute risk reduction 4.6% at 5 years. Endarterectomy is not indicated for symptomatic patients with <50% stenosis. On the other hand, Carotid endarterectomy for asymptomatic stenosis reduces the risk of
ipsilateral stroke, and any stroke, by 30% over 3 years. However, the absolute risk reduction is small (2%/y rate was reduced to 1%/y; number needed to treat =17; p=0.004)\(^4\).

In the recent years, appearance of angioplasty and stenting under distal protection devices and potent antiplatelet regimes, provides competitive alternatives to the classical endarterectomy. The limiting steps for angioplasty and stenting were the risk of compression of the stent and the potential for cerebral embolisation during the procedure. However, the ready availability of crush-resistant self expandable stents and embolic protection device has placed this technology into the hands of many endovascular specialists\(^1\).

Despite the fact that carotid endarterectomy was established as the gold standard for treatment of carotid occlusive disease by several landmark papers, several decades of experience with CEA, however, has revealed high-risk subsets of patients in whom CEA carries increased risk of adverse events. These patients have been excluded from the North American Symptomatic Carotid Endarterectomy Trial and Asymptomatic Carotid Atherosclerosis Study (NASCET and ACAS trials). These patients have subsequently been the focus of several randomized trials and registry databases which evaluated and proved non-inferiority of carotid angioplasty and stenting (CAS) in recent years. CAS is now considered an appropriate and equivalent alternative to CEA in these high-risk patients, defined by the presence of severe cardiac, pulmonary, or renal disease or by the presence of local factors such as prior neck radiation, prior neck operations, contralateral carotid occlusion, or surgically inaccessible lesions or in patients with combined symptomatic carotid and coronary artery disease. Ongoing trials in normal-risk patients may ultimately expand the indications for CAS\(^5\).

This is an observational study to report the results of our first 20 patients with significant carotid stenosis for whom carotid angioplasty and stenting under embolic protection device (EPD) was done.

### SUBJECTS AND METHODS

**Subjects**

Between January 2004 and June 2006, we performed carotid angioplasty and stenting under embolic protection device (EPD) for 20 patients (21 stent for 21 lesions) with significant carotid stenosis fulfilling the following criteria:

**Inclusion criteria:**
- Symptomatic more than 70% carotid stenosis.
- Asymptomatic more than 80% carotid stenosis.
- Bilateral carotid stenosis more than 70%, both symptomatic and asymptomatic.

**Exclusion criteria:**
- Significant neurologic deficit (modified Rankin scale, mRS ≥3).
- Ischemic stroke within 48 h.
- Thrombocytopenia or GIT hemorrhage (3 months).
- Patients with allergy to aspirin or clopidogrel.
- Fresh thrombus at the carotid lesion site.
- Chronic carotid total occlusion.
- Vascular disease precluding use of catheter-based techniques.
- Intracranial aneurysm >9mm in diameter.
- Life expectancy < 1 year.

**Methods**

**All patients underwent:**
- Clinical assessment together with National Institute Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS). This was done before stenting, within 24h, one week, one month, 3 months, 6 months and one year after stenting.
- Laboratory investigations: complete blood count, liver and renal function tests, fasting
and post-prandial blood sugar and lipid profile.
* Carotid duplex ± MRA neck (3D time of flight; 3TOF). This was done before stenting, 6 months and one year after stenting.
* MRI brain, MRA brain (3D time of flight; 3TOF).
* Echocardiography.

Significant cervical carotid stenosis (more than 50%) based on carotid duplex was diagnosed when there was an absolute increase in the peak systolic velocities more than 125 cm/sec or a peak systolic velocity ratio of 2 (peak systolic velocity at the stenosis/ peak systolic velocity at normal CCA) with spectral broadening. A stenosis more than 70% represented an absolute increase in the peak systolic velocities > 230 cm/sec, peak systolic velocity ratio of 4, or an end diastolic ratio of more than 5.5 (end diastolic velocity at the stenosis/ end diastolic velocity at normal CCA) with spectral broadening\(^6\).

Atherosclerotic lesions detected by MRA or digital subtraction angiography (DSA) were interpreted as follows\(^7\):
* Vessel wall irregularity.
* Percent of stenosis was graded according to the following equation:
\[
\text{(I-stenosis diameter/normal segment diameter) x 100}
\]
correlating the stenosed ICA segment to the more distal healthy part of the ICA (NASCET criteria)
* Flow void (signal loss) with a clearly defined proximal and distal portion of the vessel on MRA images was rated as ≥ 70% stenosis.

MRI brain: cerebral infarction was classified as follows:
* Territorial infarction
  Partial anterior circulation (involving either the deep gray matter nuclei ± internal capsule or the cerebral cortex)
  Total anterior circulation (involving both the deep gray matter nuclei, internal capsule and cerebral cortex)
  Posterior circulation
* Lacunar infarction
* Water-shed infarction

**Carotid Angioplasty and Stenting under Embolic Protection Device:**

**Medical treatment**

**Pre procedure:**
All patients were treated with acetyl-salicylic acid (150 mg/d) associated with clopidogrel (75 mg/d) for one week or 300 mg clopidogrel at least 24 h before the procedure

**During the procedure:**
IV heparin was given to maintain a therapeutic activated partial thromboplastin time of at least 80 sec. Just before the post-stenting dilation phase, atropine (0.5-1 mg IV) was given in order to reduce the bradycardia and hypotension potentially associated with carotid dilation.

**Post procedure:**
Clopidogrel (75 mg/d) was continued for at least 3 months after the interventional procedure and acetyl-salicylic acid is continued indefinitely.

**Description of the procedure**
All procedures were carried out via puncture of the right or left femoral artery. The common carotid artery was selectively engaged directly by using a proper 8 F guiding catheter. When the use of a primary guide catheter was not possible due to the particular anatomy of supra-aortic vessels, we placed a stiff wire into the external carotid artery, for positioning of a long sheath or a guiding catheter (co-axial exchange technique) into the common carotid artery.

All patients underwent an angiographic examination of the culprit (accused) carotid lesion in at least two different projections and an angiographic examination of intra-cranial circulation in antero-posterior and lateral projection. The same angiographic check-up was performed at the end of the procedure in order to determine if there was any variation in the intra-cranial blood flow.

**Cerebral protection devices**
In the present series we used only filter-wire devices (AngioGuard - Cordis or FilterWire EZ - Boston Scientific or Spider - ev3). Reversal flow and proximal occlusive devices were not used. The device consists of a guidewire with a 0.014 inch diameter and an expandable filter bag. The guidewire was used to cross the carotid-artery stenosis first, and then the filter was expanded before the stent was deployed. At the end of the procedure, the filter containing the captured emboli was collapsed and removed.

**Carotid stenting**

Carotid stenting was carried out by using self-expandable crush-resistant stents (Smart Precise – Cordis or Carotid Wallstent -Boston Scientific). In tight, sub-occlusive carotid stenosis pre-dilation was performed with low profile balloons 0.014 inch wire compatible. The pre-dilation balloons were routinely undersized (artery/balloon ratio: 2-1.5) in order to reduce vessel dissection and/or distal embolization. Stent placement was optimized through single or multiple post-dilatations by using suitably sized balloons based on angiographic quantitative analysis of the vessel.

**Procedural success was defined as:**

* To succeed to cover the whole lesion by the use of a single stent.
* To achieve a <30% residual diameter stenosis of the treated lesion in at least two matched views on angiography.
* Absence of distal embolisation.

**RESULTS**

Our study included our first 20 patients with significant carotid stenosis for whom carotid angioplasty and stenting under embolic protection device was done (21 stents for 20 patients; one patient performed bilateral carotid stenting). They were 15 male (75%) and 5 females (25%). Their age ranged from 50 y to 74 y with a mean of 64.2 +/-8.3y and 40% of our patients were ≥70y. Atherogenic risk factors included, hypertension in 16 (80%), smoking or ex-smoking in 12 (60%), dyslipidemia in 11 (55%), diabetes mellitus in 9 (45%). Eight patients had ischemic heart disease (40%) while two had lower limb ischemia (10%) (Table 1).

The majority of our patients presented with either TIA or minor stroke (complete recovery within 4 weeks) (18 patients; 90%) and so none of our patients had neurologic deficit in the territory of the stented artery. Only two patients had significant disability (mRS 3-4) due to neurologic deficit related to the UN-STENTED ICA. Concerning these later two patients, one had total occlusion of the Rt ICA resulting in a large total anterior circulation infarction on this side (mRS 4) while we stented his asymptomatic Lt ICA that was more than 80% stenosed. The second patient had bilateral strokes with only residual Lt sided weakness (mRS 3) and the Lt ICA was stented (Table 2).

Fourteen patients had symptomatic more than 70% carotid stenosis (66.6%) while 6 patients had asymptomatic more than 80% carotid stenosis. Of those with asymptomatic carotid stenosis more than 80%, two patients had contralateral total occlusion of the ICA and one patient had bilateral asymptomatic carotid stenosis more than 80%. One patient had bilateral carotid stenosis more than 70% where one side was symptomatic while the other side was asymptomatic (Table 3).

Concerning the MRI brain findings in territory stented artery, 9 patients (45%) had partial anterior circulation infarction ± lacunar, 9 patients (45%) had only lacunar infarcts while two had normal MRI. None had total anterior circulation infarction. As regard the vascular condition, apart from the stented artery, 11 patients had significant intracranial arterial stenosis (55%) while 7 patients (35%) had contralateral extracranial arterial stenosis (50-69%), 2 patients (10%) had contralateral extracranial carotid stenosis (70-99%) and 2 patients (10%) had contralateral extracranial total carotid occlusion (Table 4).
Our study included 3 high risk patients (15%), two had contralateral carotid occlusion and one had severe cardiac disease.

We performed stenting for 11 Rt ICA and 10 Lt ICA. The mean degree of stenosis based on angiography was 83.9±7.9%. 3 of the lesions were heavily calcified while 6 plaques were ulcerated (Table 5).

We succeeded to pass the embolic protection device distal to the stenosis prior to predilatation or stenting so all our stenting procedure was done under EPD (21/21; 100%). We succeeded to deploy our stent in the adequate position in all our cases and there was no need to use a second adjunctive stent (21/21; 100%). This resulted in successful revascularization with a <30% residual stenosis in all our patients (Table 6).

Only one patient developed a TIA (1/21 procedure; 4.7%) in the form of word finding difficulty few hours after the procedure. His MRI with diffusion weighted image did not reveal a new lesion. None of our patients developed minor or major stroke or intracranial hemorrhage. One patient (4.7%) developed a myocardial infarction 4 days after stenting. The later was one of the three high risk patients we included since he had total occlusion of the contralateral ICA. No complications were faced in the other two high risk patients (Table 7).

Two of our patients were particularly difficult. The first was a 73y old female with recurrent TIAs who had tight (90%) long segment (1.5 cm) stenosis of her Lt ICA in addition to a 50% stenosis of her Rt ICA. She had a bovine aortic arch with the Lt CCA arising from the brachiocephalic trunk with an acute angle which represented a technical difficulty to ascend the guiding catheter into the Lt CCA. However, we succeeded to stent her tight Lt ICA resulting in its complete recanalization (Fig. 1). The second was a 71y old male with bilateral tight ICA stenosis. The Lt 95% ICA stenosis was due to a large hypoechoic plaque of 1.8 cm length. This large plaque represented a high risk for distal embolisation. Stenting was successfully done without complications and a big atherosclerotic tissue debris was retrieved from the EPD. Two months later his Rt ICA was stented (asymptomatic 80% stenosis) (Fig. 2).

During long-term follow-up (mean 16 months, max 34 months), none developed recurrent TIA or stroke, one patient died of myocardial infarction (1y after carotid stenting). Untill the publishing of this work, 12 of our patients completed 1 year follow-up. Concerning re-stenosis at 1year post-stenting, none out of nine patients showed re-stenosis based on carotid duplex in 7 and angiography in 2 (2 refused to do carotid duplex and 1 patient died due to myocardial infarction) (Table 8).

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**Table 1.** Baseline characteristics and medical history.

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>15</td>
<td>75</td>
</tr>
<tr>
<td>Female</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>Age mean ±8.3y)</td>
<td></td>
<td>40% ≥70y</td>
</tr>
<tr>
<td>Hypertension</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td>Smoking</td>
<td>12</td>
<td>60</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>Ischemic Heart Disease</td>
<td>8</td>
<td>40</td>
</tr>
<tr>
<td>Lower Limb ischemia</td>
<td>2</td>
<td>10</td>
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**Table 2.** Presentation and neurologic state of the patients at time of stenting.

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>TIA/minor stroke</td>
<td></td>
<td>18</td>
</tr>
<tr>
<td>Neurologic state</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Neurologic Deficit (territory stented artery)</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>mRS 0-1</td>
<td></td>
<td>18</td>
</tr>
<tr>
<td>mRS 3-4</td>
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**Table 3.** Symptomatic versus asymptomatic carotid stenosis.

<table>
<thead>
<tr>
<th></th>
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<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic (territory stented artery)</td>
<td>14/21</td>
<td>66.6</td>
</tr>
<tr>
<td>Asymptomatic (territory stented artery)</td>
<td>7/21</td>
<td>33.4</td>
</tr>
</tbody>
</table>

**Table 4.** MRI brain and MRA +/- DSA findings.

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI brain (territory stented art)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial anterior circulation ± lacunar</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>Total anterior circulation ± lacunar</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Only Lacunar</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>Normal MRI</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>MRA ± DSA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Significant intracranial stenosis (&gt;50%)</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>Contralat carotid stenosis (50-69%)</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>Contralat carotid stenosis (&gt;70%)</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Contralat carotid occlusion</td>
<td>2</td>
<td>10</td>
</tr>
</tbody>
</table>

**Table 5.** Angiographic details of the stented carotid arteries.

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rt</td>
<td>11</td>
<td>52.3</td>
</tr>
<tr>
<td>Lt</td>
<td>10</td>
<td>47.7</td>
</tr>
<tr>
<td>Degree stenosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean 83.9±7.9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean 1.0 cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heavy calcif</td>
<td>3</td>
<td>14.2</td>
</tr>
<tr>
<td>ulcer</td>
<td>6</td>
<td>28.5</td>
</tr>
</tbody>
</table>
Table 6. Procedural success.

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPD positioning success (before predilatation or stenting)</td>
<td>21</td>
<td>100</td>
</tr>
<tr>
<td>Stent deployment success</td>
<td>21</td>
<td>100</td>
</tr>
<tr>
<td>Successful dilatation (&lt;30% residual stenosis)</td>
<td>21</td>
<td>100</td>
</tr>
<tr>
<td>Distal embolization</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Procedural success</td>
<td>21</td>
<td>100</td>
</tr>
</tbody>
</table>

**Fig. (1):** (a and b) Lt ICA origin showing a large plaque inducing a long segment (1.5 cm) of 90% stenosis before stenting. (c) Bovine arch with the Lt CCA arising from the brachiocephalic trunk. (d) Lt CCA after stenting with complete recanalization of the ICA.
Fig. (2): (a) Lt ICA origin showing a large plaque inducing a long segment (1.5 cm) of 90% stenosis before stenting. (b) Lt CCA after stenting with complete recanalization of the ICA.

Table 7. Complication rate.

<table>
<thead>
<tr>
<th>Events</th>
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<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 0-Day 30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIA</td>
<td>1/21 (Day 0)</td>
<td>4.7</td>
</tr>
<tr>
<td>Minor-major stroke</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Intracranial Hemorrhage</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Myocardial Infarction</td>
<td>1 (day 4)</td>
<td>4.7</td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 8. Long-term follow-up.

<table>
<thead>
<tr>
<th>Events</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recurrent TIA/stroke</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Re-stenosis</td>
<td>0</td>
<td>0</td>
</tr>
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</table>

**DISCUSSION**

Extracranial internal carotid artery stenosis accounts for 15-30% of ischemic stroke. Carotid endarterectomy has high efficacy in stroke prevention in selected patients with symptomatic carotid stenosis. A pooled analysis of final data from the 3 randomized controlled trials, the North American Symptomatic Carotid Endarterectomy Trial, the European Carotid Surgery Trial and the Veterans Affairs Cooperative Studies (NASCET, ECST, and VACS) demonstrated that surgery had marginal benefit in those with 50% to 69% stenosis (absolute risk reduction =4.6%; p=0.04), and was of greatest benefit in patients with more than 70% stenosis (absolute risk reduction =16%; p<0.001). For those with more than 70% stenosis, maximum benefit was reached by 3 years, with the event curves becoming largely parallel thereafter.
On the other hand, carotid endarterectomy for asymptomatic stenosis reduces the risk of ipsilateral stroke but the absolute risk reduction is small (1%/y). Therefore, carotid revascularization in asymptomatic patients or in symptomatic patients with 50-69% stenosis requires special consideration.

Patients with asymptomatic severe carotid narrowing (> or =80%) might benefit from CAS. The Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) is a study aimed at identifying a subgroup or subgroups of patients with asymptomatic ICA stenosis more than 50% at a risk for stroke higher than 4% (high risk) and a group at a risk for stroke less than 1% per year (low risk) using systemic and local risk factors (plaque characterization) in addition to the degree of stenosis. A linear relationship was found between degree of ICA stenosis and ipsilateral ischemic events. The degree of stenosis, history of contralateral TIAs and creatinine more than 85 micromol/L were independent risk predictors. The combination of these three risk factors can identify the high-risk group (7.3% annual event rate and 4.3% annual stroke rate) and a low risk group (2.3% annual event rate and 0.7% annual stroke rate).

Similarly, Sherif et al. reported a propensity score-adjusted analysis of the long-term risks for stroke after CAS compared to medical therapy in patients with asymptomatic high-grade carotid artery stenosis. A total of 946 consecutive patients with asymptomatic high-grade carotid artery stenosis (> or =70%) were treated either medically (n=525) or with CAS (n=421). The risk of stroke increased in parallel with the degree of stenosis in conservatively treated patients, but remained unchanged in patients undergoing CAS. Compared to conservatively treated patients with 70% to 79% stenosis, the adjusted hazard ratios for stroke were 2.36 (p=0.044) for conservatively treated patients with 80% to 89% and 3.17 (p=0.026) for those with 90% to 99% stenosis. For CAS patients with 70% to 79%, 80% to 89%, and 90% to 99% stenosis, the adjusted hazard ratios for stroke were 1.32 (p=0.63), 0.91 (p=0.84), and 0.98 (p=0.98).

Therefore, patients with asymptomatic severe carotid narrowing (> or =80%) might benefit from CAS with respect to stroke-free survival. Randomized controlled trials are needed to confirm these findings.

In line with this, management of asymptomatic patients with more than 60% or symptomatic patients with 50-69% stenosis may requires, in addition to determining the degree of stenosis, to study plaque characterization and vulnerability, occurrence of embolization, presence of other stenotic or occluded segments such as a contralateral carotid occlusion, presence of collateral circulation and cerebrovascular reactivity. In addition, regular surveillance of these patients is necessary to detect progressive stenosis.

Several studies showed that echolucent plaques generate a higher number of embolic particles following carotid stenting. Echolucency can be measured using the gray scale median (GSM), which is an objective and quantitative computer-assisted grading of the echogenicity of carotid plaques. Similarly, intraplaque hemorrhage, enlarging lipid and necrotic cores, and their proximity to the flowing lumen and a thin or ulcerated fibrous cap are important determinants of carotid plaque rupture and neurological complications. Carotid duplex may be used to identify high-risk carotid atherosclerotic plaques and thereby improve the selection of patients requiring carotid endarterectomy or carotid artery stenting. Recent studies have also demonstrated that high-resolution MRI (black blood MRI) can identify these plaque components.

Embolus detection using transcranial Doppler can also help in decision making. In a group of patients with asymptomatic ICA stenosis more than 60%, patients with microemboli were more likely to have a stroke during the first year of follow-up (15.6% vs 1%; p< 0.0001). In line with this, patients with 60-<70% asymptomatic carotid stenosis and contralateral carotid occlusion carry a higher incidence of ipsilateral strokes and all strokes than those without contralateral occlusion (60% over 5 years
compared to the 12% reported by the ACAS and ACST trials. Therefore, prophylactic CEA may be justified in these patients.\textsuperscript{15}

In contrast, blood supply through collateral circulation improves regional cerebral blood flow and may protect against ischemic events. In the NASCET, the two-year risk of hemispheric stroke in medically treated patient with severe ICA stenosis was reduced in the presence of collaterals (27.8% to 11.3%; \(P=0.005\)). Nevertheless, the reduction in disabling or fatal strokes was not statistically significant (13% vs 6.3%; \(P=0.11\)).\textsuperscript{16} Hence, a study of the collateral circulation, the cerebral perfusion and the cerebral perfusion reserve may also help in decision making.

On the other hand, progression of ICA/CCA stenosis was noted in a significant number of patients particularly with a baseline degree of stenosis. Upon following-up of 420 patients, AbuRahma et al.\textsuperscript{17} reported that in overall, carotid artery stenosis progressed in 26% of patients at mean follow-up of 41 months. Progression of stenosis was noted in 3% of patients with baseline normal carotid arteries. On the other hand, progression occurred in 36% of patients with less than 50% stenosis versus 47% of patients with 50% to 79% stenosis (\(P=0.003\)). Median time to progression was 24 months for \(<50\%\) carotid stenosis, and 12 months for \(50\%\) to \(79\%\) carotid stenosis (\(P=0.035\)). Therefore, serial clinical and duplex ultrasound scanning every 6 to 12 months in patients with \(50\%\) to \(79\%\) carotid stenosis, and every 12 to 24 months in patients with \(50\%\) or less carotid stenosis is adequate. Raman et al.\textsuperscript{18} reported that clinical and demographic factors were not helpful in predicting which patients would have disease progression.

The endovascular treatment of carotid artery stenosis has undergone substantial refinement since its introduction including the development of crush-resistant self expandable stents, protection devices, in addition to potent antiplatelet regimes. The development of embolic protection device provided the answer to the problem of atheroembolism from the intervention site. The availability of multiple embolic protection systems has been shown in many single and multicenter registries to confer a remarkably low risk of embolic complications after carotid stenting.\textsuperscript{19,23} In a prospective registry, carotid stenting procedures with \(n=538\) versus without \(n=775\) embolic protection were associated with lower 30-day rates of any stroke (1.9% versus 5.8%, respectively; \(P=0.0003\)) and stroke or death (2.4% versus 6.5% respectively; \(P=0.001\)). Utilization of embolic protection was the strongest multivariate predictor of freedom from periprocedural stroke. Thus, embolic protection should be considered the standard of care in carotid stenting.\textsuperscript{24}

Carotid artery angioplasty and stenting is now widely performed on symptomatic and asymptomatic patients.\textsuperscript{25} Goodney et al.\textsuperscript{26} revised large case series, registries, and randomized trials of carotid artery stenting. In >20 case series that was studied more than 24,000 patients undergone CAS, 51% of patients were symptomatic, most procedures (97%) resulted in successful stent deployment, and 30-day stroke rates varied from 1% to 8%, with a trend toward lower rates as experience and embolic protection device (EPD) use increased. In 12 registries, 30-day stroke rates varied from 2% to 7%, and 30-day combined adverse events, including stroke, death, and myocardial infarction, were 3% to 9%. The ELOCAS Registry\textsuperscript{27} included 2172 patients with 5 years follow-up. The Procedural success rate was 99.7%, the 30 days major stroke/death: 1.2% while the restenosis rate at 1y was 1%, at 3y was 2% and at 5y it was 3.4%.

More than 12 randomized trials comparing CAS and CEA have been initiated since 1998. Results have varied over time, depending on the population studied and the technology used. The largest and most recent trial is the Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trial\textsuperscript{28} which demonstrated that in high-risk patients CAS with the use of EPDs is at least not inferior to CEA with a strong trend toward superiority. Stenting resulted in rates of complications for all adverse events (death, stroke, or myocardial
infarction) that were statistically equivalent to or lower than those among patients who underwent endarterectomy both in the overall study population and in the subgroups with asymptomatic or symptomatic stenosis. The rate of the peri-procedural primary end point (a composite of death, stroke, or myocardial infarction within 30 days) was 39% lower among patients who were randomized to protected carotid stenting than those who were assigned to undergo CEA (4.4% among CAS and 9.9% among CEA; p=0.06). The cumulative incidence of the primary end point at one year (ipsilateral stroke and death) showed a nearly significant difference between CAS and CEA (12% among CAS and 21.1% among CEA; p=0.048). The rates of bleeding complications were similar in the two groups, and the rates of cranial-nerve palsy (4.9% among CEA vs 0% among CAS; p=0.004) and need for repeated revascularization (4.3% among CEA vs 0.6% among CAS; p=0.04) and the duration of the hospital stay were greater among those in the CEA group than among those in the stenting group.

Large scale, multicenter, randomized trials are in progress to address not only whether CAS could be superior to CEA in high-risk patients but also, more importantly, whether CAS is beneficial in other subgroups, such as low-risk and asymptomatic patients. Of these is the Carotid Revascularization Using Endarterectomy or Stenting Systems (CaRESS) study. There was no significant differences in combined death/stroke rates at 30 days (3.6% CEA vs 2.1% CAS) or at 1 year (13.6% CEA vs 10.0% CAS). Similarly, there was no significant difference in the combined end point of death, stroke, or MI at 30 days (4.4% CEA vs 2.1% CAS) or at 1 year (14.3% CEA vs 10.9% CAS). There were no significant differences between CEA and CAS in the secondary end points of residual stenosis (0% CEA vs 0.9% CAS), restenosis (3.6% CEA vs 6.3% CAS), repeat carotid revascularization (1.0% CEA vs 1.8% CAS). The CaRESS phase I study suggests that the 30-day and 1-year risk of death, stroke, or MI with CAS is equivalent to that with CEA in symptomatic and asymptomatic patients with carotid stenosis. Concerning asymptomatic patients with more than 70% stenosis, Marine et al. and Brooks et al. reported that CAS demonstrated equivalent outcomes compared to CEA. With the completion of the Carotid Revascularization Endarterectomy versus Stent Trial (CREST), the Stent-Protected Percutaneous Angioplasty of the Carotid artery versus Endarterectomy trial (SPACE) and the Asymptomatic Carotid Stenosis Stenting versus Endarterectomy trial (ACT 1) the role of CAS in the prevention of stroke in all individuals with significant carotid stenosis should be better demarcated.

Another potential advantage of CAS over CEA is the absence of surgical incision in the neck and its complications including cranial nerve palsies, wound hematoma and scar. These local complications occurred in 13.1% of patients included in the NASCET trial. Concerning the long term follow-up, numerous studies have shown that late neurological events and restenosis after carotid stenting are rare. Roubin et al. prospectively followed 528 patients after carotid stenting over a 5-year period. With 30-day events included, long-term freedom from any ipsilateral strokes was 95%. Freedom from any ipsilateral stroke after the first postprocedural month was 99 % and the rate of restenosis that required reintervention was only 3%. Bosiers et al. recently reported the long-term outcomes of 2167 patients undergoing successful carotid angioplasty (stenting rate 95%). At 5-year follow-up, 85% of patients were alive and free from ipsilateral stroke, with restenosis rates less than 4%. The available data thus demonstrate that in a broad spectrum of patients, the excellent early results of carotid stenting are durable in the long term.

In our study, we could achieve peri-procedural results (within 30 days) similar to that published from large case series and randomized trials (TIA, 4.7% and myocardial infarction, 4.7%). We faced no stroke (major or minor) or death. We obtained these results despite the fact
that 40% of our patients were ≥70y old, 66.6% were symptomatic, 15% were high risk patients and 10% had contralateral tight carotid stenosis (more than 90%). In addition, 48% of the patients were treated from a Lt ICA stenosis which is sometimes technically more difficult than the Rt side. Furthermore, the mean degree of stenosis based on angiography was 83.9±7.9%, 14% of the lesions were heavily calcified while 28% of the plaques were ulcerated.

Among the three high risk patients we included, we faced no neurologic complications during the peri-procedural period and during follow-up. One patient developed a myocardial infarction 4 days after the procedure.

Concerning our patients with asymptomatic carotid stenosis (6 patients), all had more than 80% stenosis, two patients had in addition contralateral total occlusion of the ICA and one patient had bilateral asymptomatic carotid stenosis more than 80%. One patient had bilateral carotid stenosis more than 70% where one side was symptomatic while the other side was asymptomatic. One patient had progressive stenosis from 50-69% to more than 80% within 8 months while the last patient had two minor strokes related to the opposite carotid territory. We faced no neurologic complications during the peri-procedural period and during follow-up. One patient developed a myocardial infarction 4 days after the procedure (high risk patient).

We succeeded to pass the embolic protection device distal to the stenosis prior to predilatation or stenting so all our stenting procedure was done under EPD. We succeeded to deploy our stent in the adequate position in all our cases and there was no need to use a second adjunctive stent.

Over a 2.5 years follow-up, none of our patients had recurrent TIA or stroke. One patient died of myocardial infarction (1y after carotid stenting). Concerning re-stenosis at 1year post-stenting, none out of nine patients showed re-stenosis based on carotid duplex in 7 and angiography in 2. Although we performed only 21 CAS procedures, carotid angioplasty and stenting under EPD seems to be a less invasive alternative and at least not inferior to CEA.

Complete vascular assessment of patients with carotid stenosis is mandatory before treatment as they have a high incidence of stenosis in other segments. In our short series, 55% of patients had significant intracranial arterial stenosis while 35% had contralateral extracranial carotid stenosis (50-69%), 10% had contralateral extracranial carotid stenosis (70-99%) and another 10% had contralateral extracranial total carotid occlusion. In addition, adequate cardiac assessment is essential to avoid the occurrence of myocardial infarction during the peri-procedural period and during follow-up.

REFERENCES


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