Endovascular therapy for stenosis of the petrous or cavernous portion of the internal carotid artery: percutaneous transluminal angioplasty compared with stent placement

TOMOAKI TERADA, M.D., MITSUHARU TSUURA, M.D., HIROYUKI MATSUMOTO, M.D., OSAMU MASUO, M.D., TOMOYUKI TSUMOTO, M.D., HIROO YAMAGA, M.D., AND TORU ITAKURA, M.D.

Department of Neurological Surgery, Wakayama Medical University, Wakayama City, Japan

Object. The effects of percutaneous transluminal angioplasty (PTA) and stent placement for stenosis of the petrous or cavernous portion of the internal carotid artery (ICA) were compared.

Methods. Twenty-four patients with symptomatic, greater than 60% stenosis of the petrous or cavernous portion of the ICA were treated using PTA or stent placement; 15 were treated with PTA and nine with stent insertion. Initial and follow-up results (>3 months posttreatment) were compared in each group.

Stenotic portions of the ICA were successfully opened in 13 of 15 patients in the PTA group, and in all nine patients in the stent-treated group. In one case in the PTA group stent delivery was attempted; however, the device could not pass through the vessel’s tortuous curve, and PTA alone was performed in this case. Postoperatively, the mean stenotic ratio decreased from 72.1 to 29.6% in the PTA group, and from 75.6 to 2.2% in the stent-treated group. In four patients in the PTA group, stenoses greater than 50% were demonstrated on follow-up angiography performed at 3 to 6 months after PTA. In the stent-treated group, no restenosis was encountered, although in one case acute occlusion of the stent occurred; the device was recanalized with PTA and infusion of tissue plasminogen activator. This case was the only one of the 24 in which any neurological deficits related to the endovascular procedure occurred. Stent placement brought a greater gain in diameter than did PTA at the initial and late follow-up period; this gain was statistically significant.

Conclusions. Stent placement is more effective than PTA for stenosis of the petrous or cavernous portion of the ICA from the viewpoint of initial and late gain in diameter.

KEY WORDS • atherosclerosis • arterial stenosis • internal carotid artery • percutaneous transluminal angioplasty • stent

DIFFERENCES in the interventional neuroradiological procedure in which angioplasty balloon catheters and stents were used have made possible the endovascular treatment of intracranial as well as cervical ICA stenosis.16,31,32,35,36 The recent favorable results of endovascular treatment for cervical ICA stenosis in a large number of cases seem to be approaching those of carotid endarterectomy after introduction of protective devices.8,13,17,35,36 Nevertheless, currently there are only a few published reports regarding PTA and/or stent placement for intracranial ICA stenosis that include long-term follow-up results.5,32 The delays in studying endovascular treatment of intracranial ICA stenosis are due to the late introduction of flexible single- or double-lumen balloon catheters and stents that can negotiate the tortuous curve of the ICA siphon. It is also known that the natural history of the ICA stenosis is similar to that of cervical ICA stenosis.7,10 Surgical treatment for stenosis of the petrous or cavernous portion of the ICA is very difficult, and the effectiveness of external carotid–internal carotid bypass surgery as the alternative treatment was denied by the authors of the cooperative study.30 Endovascular treatment for stenosis of the petrous or cavernous portion of the ICA is performed without difficulty by using a newly developed angioplasty balloon catheter or stent.5,6,10,12,15,29,30 We started to treat ICA stenoses with PTA in 1995 and introduced stent procedures in 1998. Stent placement seems to yield better outcomes than PTA but there are no reports in which the results are compared between PTA and stent insertion for intracranial ICA stenosis. We compared overall results between PTA and stent placement in our series, and the advantages and disadvantages of each treatment are discussed in this paper.

Clinical Material and Methods

Patient Population

Twenty-four patients with symptomatic, greater than
60% stenosis of the petrous or cavernous portion of the ICA were treated with PTA or stent placement between January 1995 and December 2001. The male/female ratio was 16:8 and patients' ages ranged from 53 to 80 years (mean 66.7 years). Eight patients had transient ischemic neurological deficits and 16 had small cerebral infarctions that were believed to be caused by the intracranial ICA stenoses (Table 1). Between 1995 and 1998, the lesions were treated with PTA alone because stents were not available. Between 1998 and 2001, primary stent placement was performed if the device was expected to be successfully delivered to the stenotic portion of the artery and could cover the entire lesion. Fifteen patients were treated with PTA, although a stent procedure was tried in one case but failed due to the vessel's tortuosity. The other nine patients were treated with stent insertion. All patients had a history of hypertension, two had diabetes mellitus, and one suffered from renal failure.

### Equipment Used for PTA

The PTA procedure was performed using single-lumen (Stealth PTA; Boston Scientific, Boston, MA) or double-lumen (Ranger PTA; Boston Scientific) PTA balloon catheters with 3- to 4-mm diameters when inflated and 10- to 20-mm lengths. A 0.014-in guidewire was used for navigation of the catheter and it was exchanged into the valved wire system in single-lumen PTA balloon catheters. In double-lumen devices, the tip of the guidewire was kept in the M₁ or M₂ segment to stabilize the balloon catheter. A PTA balloon was inflated for 1 to 2 minutes at 6 to 8 atm.

### Equipment Used for Stent Treatment

Two GFX (AVE) stents (Medtronic, Minneapolis, MN), one NIR stent (Boston Scientific), five GFX S-670 stents (Medtronic), and one Bx-Velocity stent (Johnson & Johnson, Miami, FL) were used. The diameters of the delivered stents were 3.5 mm in five cases and 4 mm in four cases and (mean 3.72 mm), and the stent length was between 9 and 18 mm (mean 12.4 mm). In cases of high-grade stenoses, the stenotic lesion was slightly dilated with a PTA balloon to allow a stent to pass through. If the stenosis was 60 to 70%, the stent was delivered with the aid of 0.014-in guidewire control without predilation and a balloon was inflated to deposit the stent. In the case of tortuous curves in the lesion, a guidewire and microcatheter were introduced into the distal portion of the M₁ or M₂ segment to stabilize the balloon catheter. A PTA balloon was inflated for 1 to 2 minutes at 6 to 8 atm.

### Table 1

**Characteristics of 24 patients treated with PTA or stents for intracranial ICA stenosis**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Symptom</th>
<th>Location of Lesion</th>
<th>Stenosis (%)</th>
<th>mRS Score (preop/FU)</th>
<th>Restenosis (%)</th>
<th>Complication</th>
<th>FU (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTA-treated group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>70, M</td>
<td>infarct</td>
<td>lt pet</td>
<td>90</td>
<td>30</td>
<td>1/1</td>
<td>80†</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>70, F</td>
<td>infarct</td>
<td>lt pet</td>
<td>80</td>
<td>20</td>
<td>1/1</td>
<td>20</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>64, M</td>
<td>infarct</td>
<td>lt pet</td>
<td>60</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>56, M</td>
<td>infarct</td>
<td>lt cav</td>
<td>60</td>
<td>30</td>
<td>1/1</td>
<td>30</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>65, M</td>
<td>infarct</td>
<td>rt pet</td>
<td>60</td>
<td>40</td>
<td>2/2</td>
<td>50</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>73, F</td>
<td>TIA</td>
<td>lt pet</td>
<td>60</td>
<td>20</td>
<td>1/1</td>
<td>20</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>70, M</td>
<td>infarct</td>
<td>lt pet</td>
<td>80</td>
<td>40</td>
<td>1/1</td>
<td>33</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>56, M</td>
<td>infarct</td>
<td>lt cav</td>
<td>66</td>
<td>33</td>
<td>1/1</td>
<td>33</td>
<td>—</td>
</tr>
<tr>
<td>9‡</td>
<td>53, F</td>
<td>infarct</td>
<td>lt cav</td>
<td>70</td>
<td>30</td>
<td>3/3</td>
<td>30</td>
<td>stent mig</td>
</tr>
<tr>
<td>10</td>
<td>80, M</td>
<td>infarct</td>
<td>lt pet</td>
<td>90</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>11</td>
<td>61, F</td>
<td>TIA</td>
<td>rt cav</td>
<td>80</td>
<td>47</td>
<td>0/0</td>
<td>40</td>
<td>—</td>
</tr>
<tr>
<td>12</td>
<td>68, M</td>
<td>infarct</td>
<td>lt pet</td>
<td>70</td>
<td>10</td>
<td>1/1</td>
<td>20</td>
<td>—</td>
</tr>
<tr>
<td>13</td>
<td>64, M</td>
<td>TIA</td>
<td>rt cav</td>
<td>65</td>
<td>55</td>
<td>0/0</td>
<td>60</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td>78, M</td>
<td>TIA</td>
<td>rt cav</td>
<td>70</td>
<td>40</td>
<td>0/0</td>
<td>40</td>
<td>—</td>
</tr>
<tr>
<td>15</td>
<td>68, F</td>
<td>infarct</td>
<td>rt cav</td>
<td>70</td>
<td>20</td>
<td>1/1</td>
<td>20</td>
<td>—</td>
</tr>
<tr>
<td><strong>mean</strong></td>
<td>66.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>stent-treated group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>69, F</td>
<td>infarct</td>
<td>lt pet</td>
<td>80</td>
<td>0</td>
<td>3/3</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>17</td>
<td>76, F</td>
<td>infarct</td>
<td>lt pet</td>
<td>90</td>
<td>0</td>
<td>3/3</td>
<td>14</td>
<td>—</td>
</tr>
<tr>
<td>18</td>
<td>56, M</td>
<td>TIA</td>
<td>lt cav</td>
<td>70</td>
<td>0</td>
<td>0/0</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>19</td>
<td>70, F</td>
<td>TIA</td>
<td>rt cav</td>
<td>70</td>
<td>10</td>
<td>0/0</td>
<td>16</td>
<td>—</td>
</tr>
<tr>
<td>20</td>
<td>64, M</td>
<td>infarct</td>
<td>lt cav</td>
<td>60</td>
<td>0</td>
<td>1/1</td>
<td>30</td>
<td>occl &amp; recanal</td>
</tr>
<tr>
<td>21</td>
<td>62, M</td>
<td>infarct</td>
<td>rt pet</td>
<td>60</td>
<td>10</td>
<td>3/3</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>22</td>
<td>64, M</td>
<td>infarct</td>
<td>rt pet</td>
<td>70</td>
<td>0</td>
<td>1/1</td>
<td>9</td>
<td>—</td>
</tr>
<tr>
<td>23</td>
<td>74, M</td>
<td>TIA</td>
<td>lt pet</td>
<td>90</td>
<td>0</td>
<td>3/3</td>
<td>20</td>
<td>—</td>
</tr>
<tr>
<td>24</td>
<td>69, M</td>
<td>TIA</td>
<td>rt pet</td>
<td>90</td>
<td>0</td>
<td>0/0</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td><strong>mean</strong></td>
<td>67.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stenosis (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>mRS Score (preop/FU)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Restenosis (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Complication</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>FU (mos)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* cav = cavernous portion; FU = follow up; mig = migration; occl = occlusion; pet = petrous portion; recanal = recanalization.
† Repeated PTA was performed because of severe restenosis.
‡ Stent placement was attempted, but the device migrated and the patient was ultimately treated with PTA.
Endovascular therapy for internal carotid artery stenosis

**Drug Regimen**

Ticlopidine (200 mg/day) was administered at least 2 weeks before PTA or stent placement and continued after treatment. Systemic heparinization was performed to keep the activated clotting time between 200 and 300 seconds during PTA or stent placement, and was continued for 1 to 7 days depending on the angiographic findings after treatment. In one case, acute thrombosis occurred 2 days after stent delivery. Endovascular treatment with PTA in this patient resulted in immediate recanalization, and $1.2 \times 10^9$ U tPA was infused for 24 hours with systemic heparinization for 7 days.

**Follow-Up Neuroimaging**

Angiographic follow up was usually performed between 3 and 6 months after PTA or stent placement, unless ischemic symptoms appeared. The mean angiographic follow-up period was 5.2 months in the PTA group, and 5.6 months in the stent-treated group. All patients were clinically followed for periods of time ranging from 3 to 72 months after the initial treatment (mean 32.9 months). Restenosis greater than 70% was treated with PTA or stent placement, even if the lesion was asymptomatic. A CT scan was obtained in all patients within 1 month posttreatment to evaluate the asymptomatic ischemic or hemorrhagic lesions.

The stenotic ratio was determined by dividing the stenotic diameter by the distal diameter of the petrous or cavernous portion of the ICA according to the North American Symptomatic Carotid Endarterectomy Trial and the neurological status of the patient was evaluated according to an mRS before and 3 months posttreatment. The changes in the vessel diameter before and after treatment (% gain = % stenosis before treatment − % stenosis after treatment) and residual stenosis immediately after treatment and at follow-up review were compared between PTA and stent-treated groups. These results were statistically analyzed using the Mann–Whitney U-test (Table 2).

**Results**

**Initial Results**

In the PTA group, stenoses in 13 patients were less than 50% after treatment and stenoses in the other two patients were greater than 50%. The stenotic rate was reduced from a mean of 72.1 to 29.6%. The cause of insufficient dilation was assumed to be elastic recoil because a PTA balloon was inflated to the normal artery diameter. In the stent-treated group, all patients demonstrated successful dilation and the stenotic rate decreased from a mean of 75.6 to 2.2%. In one case, however, we failed to deliver the stent to the lesion because of vessel tortuosity, and as a result, we finally treated the patient with PTA alone. This case was assigned to the PTA group. Therefore, the success rate of stent placement was 90%. The stenotic rate before treatment was not significantly different between the PTA and stent-treated groups. Nevertheless, the PTA group demonstrated significantly greater residual stenosis posttreatment than did the stent-treated group. The initial gain (% stenosis before treatment − % stenosis after treatment) in the stent-treated group (73.3%) was significantly greater than that in the PTA group (42.4%) (p = 0.0014, Mann–Whitney U-test).

**Follow-Up Results**

In the PTA group, four patients demonstrated greater than 50% stenosis on follow-up angiography after the initial procedure. One patient with 80% stenosis received repeated PTA, although the individual was asymptomatic. The mean stenotic rate on follow-up angiograms was 34.4% in the PTA group. In the stent-treated group, all patients exhibited successful dilation, even on the follow-up angiogram. The mean stenotic rate on the follow-up angiogram in this group was 9.9%. The PTA group demonstrated significantly greater late stenosis than the stent-treated group after the procedure. The late gain (% stenosis before treatment − % stenosis after treatment) in the stent-treated group (65.6%) was significantly greater than that in the PTA group (38.3%) (p = 0.0046, Mann–Whitney U-test).

Four patients (Cases 2–5) suffered cerebral infarction during the follow-up period. One patient suffered a left posterior cerebral artery thrombotic occlusion that resulted in a massive infarction of the posterior cerebral artery territory 1 year after PTA. One patient had a right MCA branch occlusion in the right posterior temporal lobe 3 years after PTA. Two patients suffered from lacunar infarct nearly 1 cm in size in the corona radiata at 3.5 and 5 years posttreatment. These lesions were not believed to be related to the intracranial ICA stenosis. Two patients (Cases 1 and 2) died of old age and myocardial infarction, respectively. The patient in Case 2 demonstrated a permanent neurological deficit related to the infarction, but the other three patients had only transient symptoms.

No patient exhibited ischemic symptoms after stent placement during the follow-up period (mean 15.2 months). As for the mRS score, there were no changes before and 3 months after the treatment in their group. No new ischemic or hemorrhagic lesions were found on CT scans performed within 1 month after treatment, except in Case 20, in which an acute stent occlusion occurred.

**Postoperative Complications**

Small dissections were found angiographically in six of 15 cases treated with PTA; all cases were asymptomatic. In Case 9, stent placement was tried for the stenotic portion but the device could not be maneuvered into the lesion. Furthermore, the stent slipped off the catheter and migrated into the cavernous ICA; the device was snared and retrieved from the sheath placed in the femoral artery. This case was
treated by PTA alone, even though a small dissection remained. In Case 20, the cavernous ICA stenosis was treated with stent placement. Stent-related thrombosis appeared 2 days after placement of the device, despite continuous systemic heparinization. The occluded portion was recanalized with PTA and continuous infusion of tPA. Nevertheless, a small hemorrhage around the old infarct was demonstrated on MR images. Sensory dominant aphasia was slightly aggravated in this case. The morbidity rate related to the procedure was one (4.2%) of 24 in our series.

Illustrative Cases

Case 7

This 70-year-old man with a history of Wallenberg syndrome had experienced a transient disturbance of calculation 1 year previously. Two infarcts were demonstrated in the left parietal area and the left lateral medulla on MR images. Cerebral angiography was also performed to examine the CA lesions because of bilateral CA bruits; nearly 80% stenosis was demonstrated at the left petrous ICA (Fig. 1 left). This lesion was treated with PTA by using a single-lumen PTA balloon catheter via the transfemoral route. The balloon was 3.5 mm in diameter and 10 mm long and was inflated twice for 1 minute at 6 atm. The patient suffered transient Gerstmann syndrome during PTA but completely recovered at the end of the procedure. The stenotic lesion was dilated with small wall dissection (Fig. 1 center). The patient was maintained on systemic heparinization for several days, and he had no new neurological deficits during a 60-month follow-up period. Follow-up angiography studies performed at 3 and 9 months demonstrated a widely patent petrous ICA without dissection (Fig. 1 right).

Case 13

This 64-year-old man with transient left hemiparesis was admitted to our hospital. A 65% stenosis of the right cavernous ICA was found on the angiogram (Fig. 2 left). The PTA procedure was performed using a 3-mm double-lumen balloon catheter. The balloon was successfully inflated but the lesion was not dilated because of elastic recoil (Fig. 2 right). Restenosis was found on the follow-up angiogram but the lesion was left untreated because the patient was asymptomatic after the initial PTA.

Case 17

This 76-year-old woman had a history of progressing right hemiparesis. A small low-density area was detected in the right basal ganglia. A diffuse area of low perfusion area was demonstrated in the MCA territory on single-photon emission CT scans performed using $^{99m}$Tc-hexamethylpropyleneamine oxime contrast material. The left ICA angiogram revealed severe stenosis in the left petrous ICA (Fig. 3 left). The PTA was performed using a double-lumen balloon catheter 3 mm in diameter, and a $3.5 \times 12$-mm stent (S-670) was successfully placed across the lesion (Fig. 3 right). Follow-up angiography performed 6 months after the initial stent placement also revealed successful dilation with no new neurological deficit.

Case 20

This 64-year-old man had a history of mild hemiparesis
and mild sensory dominant aphasia. A small low-density area was detected in the left parietal lobe on CT scans. Cerebral angiography demonstrated a 60% stenosis in the left cavernous ICA. Antiplatelet therapy (ticlopidine, 200 mg/day) was administered and further ischemic events did not occur. Six months after the initial ischemic stroke, transient aggravation of aphasia and hemiparesis appeared despite administration of the antiplatelet drug. The stenotic rate was still 60% but the lesion was irregular with a small dissection on cerebral angiography (Fig. 4A). The patient was treated with stent insertion for the cavernous ICA stenosis. A No. 7 French guiding catheter was introduced into the cervical ICA and a 3.5 × 12-mm coronary stent (S-670) was introduced into the lesion by using a 0.014-in guidewire. The stent was successfully placed across the lesion, and the stenotic portion was dilated, resulting in mild wall irregularity (Fig. 4B). The patient’s course was uneventful during the entire procedure. In addition to antiplatelet therapy, systemic heparinization was continued from the beginning of the procedure to keep the activated clotting time level between 200 and 300 seconds. Two days after stent placement the patient suddenly became totally aphasic with dense hemiparesis; at this time the activated clotting time was reduced to 150 seconds. Emergency CT scanning demonstrated no early ischemic sign or hemorrhagic lesion. Subsequent angiographic studies revealed total occlusion of the ICA at the cavernous portion.

A 3 × 10-mm single-lumen PTA balloon catheter was introduced and PTA was performed through the stent. The blood flow was partially restored and the patient's hemiparesis improved immediately after the treatment, although his aphasia continued (Fig. 4C). Vision in his left eye was preserved. Residual stenosis, irregularity of the vessel wall, and obliteration of the OphA remained in spite of PTA, and 1.2 × 107 U tPA was continuously infused for 24 hours with systemic heparinization and antiplatelet therapy (Fig. 4D). The MR images obtained 1 week after stent placement demonstrated a high-intensity area on T₁-weighted images in the previous infarct; however, no new infarcts were found (Fig. 4E). The wall irregularity disappeared on the follow-up angiogram obtained 1 week after recanalization (Fig. 4F). The stenotic lesion was widely open and mild stenosis was noticed at the proximal edge of the stent on follow-up angiographic studies obtained 6 months after the initial treatment (Fig. 4G). The patient was free of hemiparesis but had mild sensory dominant aphasia, which worsened slightly after the treatment.

**Discussion**

Marzewski, et al.,11 reported that the risk of causing ischemic events in patients with greater than 50% intracranial arterial stenosis was 27.3% (12.1% for a TIA; 15.2% for stroke) in 3.9 years. Craig, et al.,7 reported that the annual stroke rate in patients with intracranial ICA stenosis was 27.3% (12.1% for a TIA; 15.2% for asymptomatic events in patients with greater than 50% intracranial arterial stenosis was 6.7% per year. Considering these data, the risk of causing an ischemic event in patients with symptomatic intracranial stenosis is 7 to 9% per year.

In 1985, the External Carotid to Internal Carotid Bypass Study Group concluded that patients who underwent bypass surgery for intracranial arterial stenosis or occlusion had no better results than those receiving medical therapy. Although PTA or stent placement was supposed to be a promising technique for intracranial atherosclerotic stenosis, reports of PTA or stent placement for these lesions were still few.1,6,10,12,15,25,29,30,33 The reasons for delayed use of these modalities were the fear of distal embolization, vessel rupture, the risk of occlusion in perforating arteries,5,22,29,30,33 and the unavailability of a balloon catheter system or stent that could negotiate the tortuous course of the intracranial arteries. We speculated that the risk of intracranial extradural atherosclerotic stenosis associated with PTA is not so high, considering the low mortality and morbidity rate of PTA or stent placement for cervical ICA stenosis. Also, the risk of occlusion of a perforating artery or vessel rupture seems very low because the extradural ICA is covered by the bone structure or dura mater and does not have important perforating arteries, in contrast to MCA or basilar artery stenosis.1,5,12,29,30,33 Furthermore, recent advancements in stent technology have enabled the delivery of coronary stents into the intracranial ICA, if the lesion is located in the petrous or cavernous portion. In recent articles on endovascular therapy for intracranial ICA stenosis, in which complications were well documented, good results were reported, with a morbidity rate of one (6.7%) in 15,5,10,12,29,30 The mortality rate was 0% and the morbidity rate was 4.2% in our series.

Judging from our results, PTA or stent placement seems to be promising as a novel method of treating intracranial ICA stenosis. There are no reports, however, in which the initial and follow-up results of PTA and stent insertion are compared for petrous or cavernous ICA stenoses. Restenosis was found in four (26.7%) of 15 patients in the PTA group and in none (0%) of the nine in the stent-treated group in our series. The rate of restenosis in the PTA group was similar to that found in coronary artery PTA (between 30 and 50%).20 Nevertheless, the results in the stent-treated group are extremely good. The rate of restenosis after stent placement in coronary arteries was reported as 10 to 45%,19,24,27 and it was significantly lower than that reported after PTA. The risk factors for restenosis after stent placement have been analyzed in coronary arteries for a
long time. Diabetes mellitus, insufficient dilation, vessels smaller than 3 mm, diffuse lesions (long lesions), and ostium lesions are known as risk factors for restenosis. In our stent-treated group, no patient had diabetes and all lesions were short and large, judging from the mean stent diameter of 3.72 mm and stent length of 12.4 mm in our series. Also, all lesions were successfully dilated after stent insertion, as is apparent from the 2.2% residual stenosis measured immediately after stent placement. From these facts, the lesions in our stent-treated group were considered to be the best candidates to keep long-term patency after the procedures. Including the PTA group, all lesions were less than 15 mm in length and more than 3 mm in diameter. From the viewpoint of lesion characteristics, as long as all lesions are short (< 15 mm) and large (> 3 mm) as they were in our series, the cavernous or petrous ICA might be a good candidate for stent placement.

The mechanism of restenosis is thought to be the following: 1) intimal hyperplasia; 2) elastic recoil; and/or 3) wall dissection. It still seems to be difficult to regulate intimal hyperplasia after PTA or stent placement, although some drugs used in animal experiments are thought to be effective in humans as well. Elastic recoil and wall dissection can be prevented, however, by the stent delivery. The significant initial gain in the stent-treated group was considered to be due to prevention of these two factors. Also, the greater initial gain would result in the reduction of restenosis. In cervical ICA stenosis, stent insertion was reported to be effective in preventing restenosis In petrous ICA stenosis, a few authors have reported successful results of stent delivery for intracranial ICA stenosis although the long-term follow-up results were still unknown.

The initial and follow-up results of stent placement for intracranial ICA stenosis were satisfactory in our series from the viewpoints of stroke prevention and angiographic findings. Nevertheless, there are some problems with stent placement procedures; not all stents can be delivered successfully to the lesion, because of the atherosclerotic changes in these patients, and there is a chance of thrombosis in stents once they are in place. Of the two unsuccessful placements in our series, one stent was not successfully delivered, which resulted in its migration, and in the other case the stent caused thrombosis. Considering the size of the stent compared with the cervical ICA, anticoagulation therapy should be diligently performed after stent placement in intracranial arteries.

Conclusions

In the near future more flexible stents will be developed for intracranial use and restenosis due to intimal hyperplasia will be well controlled using drug-eluting stents such as those already used in coronary artery diseases. The refinement of these devices will accelerate the use of endovascular therapy for intracranial ICA stenosis.
Endovascular therapy for internal carotid artery stenosis

References


Manuscript received May 13, 2002. Accepted in final form November 18, 2002.

Address reprint requests to: Tomoaki Terada, M.D., Department of Neurological Surgery, Wakayama Medical University, 811-1 Kimiidera, Wakayama city, Wakayama 641-0012, Japan. email: teradato@wakayama-med.ac.jp.