Carotid Stenting with the Use of Wallstent

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Key words: carotid artery, stent, Wallstent, carotid stenting, self-expanding stent

Summary

We performed carotid stenting with Wallstent for 22 cases. The mean stenosis was $74.3 \pm 11.2\%$ before stenting and $14.2 \pm 8.8\%$ after stenting. The technical success ($<40\%$ residual stenosis) was 100%. We experienced 2 embolic complications (one TIA and one minor stroke). Permanent neurological deficits were not encountered in all the patients (0%). Restenosis was observed in 2 cases (12.5%). The both lesions revealed intimal hyperplasia on intravascular ultrasound (IVUS). MRI diffusion weighted study showed that distal embolism happened more often (46.2%) than we expected although most of them were asymptomatic. We must wait for the long term result to conclude that carotid stenting has become one of the treatment modalities.

Introduction

Recently neurovascular interventions make remarkable progress and the biggest topic of them is stenting of carotid artery. Carotid endarterectomy (CEA) is the most popular treatment modality, but patients with high-positioned lesions or ischemic heart disease and high aged patients are often excluded.

We have performed carotid stenting with

Wallstent for them from 1997¹. We report the procedures, the indication and the results in detail.

Methods

Stenting was performed for 22 lesions and on 20 patients (2 patients had bilateral lesions) from June 1997 to October 1999. The mean age was 70.5 ± 5.7 . 20 patients consisted of 18 males and 2 females. For all the lesions, we used Wallstent (includes EasyWallstent and MagicWallstent).

The indication is $>70\%$ stenosis for symptomatic lesions and $>60\%$ stenosis for asymptomatic lesions according to NASCET and ACAS criteria for CEA^{2,3}. If it is assumed that the lesions can be safely treated by CEA, we usually select CEA. But we perform stenting on patients with high-positioned lesions, bilateral lesions, or ischemic heart diseases. We usually select stenting on the high aged patients (>70 years old). 26 lesions were treated by CEA in the same period.

The procedures will now be described in detail. We usually give 100-200 mg of tycolpidine and 80 mg of aspirin at least 1 week before the procedure. Angiography, IVUS, carotid sonography and single photon emission computed tomography (includes the acetazolamide test) are

Figure 1 Case 1. A) Initial angiogram showed severe stenosis in the internal carotid artery. B) The lesion was successfully dilated by stenting. C) Angiogram showed restenosis 2 years and 3 months after stenting. D) The lesion was successfully treated by balloon angioplasty.

performed. We add the WAIS-R test for patients with dementia.

We usually use 8 mm / 20 mm of EasyWallstent or 6 mm of MagicWallstent. The Wallstent delivery catheter is so stiff and bulky that predilatation is necessary for safe introduction into the lesion. Before predilatation we give 1mg of atropine intravenously and intentionally cause tachycardia in the patients. We use 0.014 inches of guidewire with every 10 mm radiopaque marker. We use guidewires for coronary angioplasty because variable devices are available and they have better crossability than any other guidewires for cerebral vessels. We prefer to use Athlete[®] marker wire. We dilate lesions 1 or 2 times using a 3.5 mm balloon such as Savvy[®] (Cordis, Johnson and Johnson). Plaques immediately after predilatation are very unstable and it is necessary to deploy stents quickly. Wallstent is the most popular self-expanding stent. SMART[®] stent (self-expanding stent) and Palmaz[®] stent (balloon expandable stent) are also used for carotid stent-

ing^{4,5}. We prefer to use Wallstent because it is resistant to deforming by external force⁶. However, Wallstent's biggest drawback is that it always shortens when deployed. That is why we get a visual grasp of its length by using a marker wire. The distal end of Wallstent is first fixed when it's deployed into the lesion. We deploy the distal end at the marker and figure out where the proximal end will be deployed if Wallstent expands as expected. After Wallstent is released completely, we remove the delivery catheter very carefully. If the predilatation is not wide enough, the delivery catheter is sometimes caught by the stent strut. We need to be especially careful if we use EasyWallstent with weak radial strength.

Finally, postdilatation will be discussed. The most important aspect is that we never overdilate the lesion. Plaque rupture by overdilatation often causes distal embolism. We should not persist in the angiographically good results. Jupiter[®] (Cordis) and MAXXAM[®] (SCIMED, Boston Scientific) balloons are usually used.

Figure 2 Case 2. A) Angiogram before stenting showed severe stenosis in the internal carotid artery. B) The lesion was successfully treated. C) Angiogram showed restenosis 8 months after stenting. D) The lesion was successfully treated by balloon angioplasty.

After the lesion is adequately dilated, we treat both ends of the Wallstent. Since Wallstent does not necessarily conform smoothly to winding arteries, it sometimes fails to adhere to the intima if it is deployed in a winding lesion. To attach Wallstent tightly to the lesion we dilate both ends at low pressure. But there is not too much cause for worry because self-expanding stents such as balloon expandable stents barely migrate⁷. That is one of the important reasons why Wallstent is used worldwide for carotid stenting.

Results

The mean stenosis was $74.3 \pm 11.2\%$ before stenting and $14.2 \pm 8.8\%$ after stenting. The technical success ($<40\%$ residual stenosis) of our series was 100%. We experienced 2 embolic complications (9.1%). The complications consisted of TIA in one patient (4.5%) and a minor stroke in one patient (4.5%). The clinical success was 90.9%. The patient with TIA had

hemiparesis immediately after predilatation but it disappeared completely within 24 hours and the patient was up and walking and discharged 2 weeks later. The patient with the minor stroke had agnosia after the final angiography of the procedure. Heparin should have been given intravenously but was actually given subcutaneously. We thought that the inadequate heparinization caused thrombosis and distal embolism. Actually, the activated clotting time (ACT) was < 200 seconds even though we gave 5000 u of heparin. After this procedure, we changed the intravenous heparin to intrare-arterial. There were no minor complications, not even re-bleeding at the puncture sites. In 13 lesions we got MRI diffusion images within one week of the procedures.

We experienced asymptomatic cerebral infarctions in 5 cases (38.5%) and a symptomatic cerebral infarction in one case (7.7%). In total, there were 6 cerebral infarctions (46.2%) in 13 procedures. The 5 asymptomatic infarcts were lesions so small that they could not be detected

Figure 3 A) IVUS image of case 1 showed severe intimal hyperplasia 2 years and 3 months after stenting. B) IVUS image of case 2 showed intimal hyperplasia and stent recoil 8 months after stenting.

by CT scan. Although most of them were asymptomatic, distal embolism happened more frequently than we expected and is one of the serious problems in carotid stenting. Hypotension and bradycardia continued for >24 hours in one patient and he needed dopamin. Within 48 hours he recovered from the hypotension.

We performed follow-up angiography in 16 cases (72.7%) and the mean follow up period was 8.2 ± 6.2 months. Restenosis was observed in 2 cases (12.5%) and intimal hyperplasia was observed on IVUS in both cases (figure 3).

We performed balloon angioplasty for the both lesions and they were successfully dilated (figures 1, 2).

Discussion

Stenting of the carotid artery has recently been performed on more and more patients. More than three thousand people have been treated by stenting in Europe and the USA and good initial results have been reported^{4,5}. They reported the high technical success of 98.8%, the low major complication rate of 4.83% and the low restenosis rate of 2.3%⁴. But some report that distal embolism occur more frequently in carotid PTA (not stenting) than in CEA, during which the distal side of the lesion is clumped and protected completely⁶. Others report that they got better results than CEA using distal protective devices such as a protec-

tive balloon and a filter device⁸⁻¹¹. We have to wait for the result of randomized studies. The focus will be on how we can decrease embolic complications using distal protection devices. At present, the protective balloon that can be used as a complete coaxial system is not available in Japan and we hope that such new devices will be developed very soon.

Now let us discuss which stents should be selected for carotid stenting. Today, Wallstent is remarkably popular in Europe and the USA for the reason mentioned above. But there have been some reports that restenosis has been observed in 41% of the series in coronary angioplasty with the use of long Wallstents¹². It is our supposition that angioplasty with Wallstent more often causes restenosis because the stress to the intima from the self-expanding stents causes intimal hyperplasia. We guess that a certain amount of intimal hyperplasia does not matter because the lumen diameter of the carotid artery is larger than of coronary arteries. However, we will have to wait for the long term results.

Conclusions

The initial result of carotid stenting was satisfactory. We will have to wait for the long term results to be able to conclude that it is suitable as one of the treatment modalities for carotid artery stenosis.

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