Ultrasonographic Character of Carotid Plaque and Postprocedural Brain Embolisms in Carotid Artery Stenting and Carotid Endarterectomy

Hiroshi Mitsuoka, MD, PhD; Tsunehiro Shintani, MD, PhD; Hidekazu Furuya, MD, PhD; Yoshinaga Nakao, MD, PhD; and Shigeki Higashi MD, PhD

Objective: To investigate ultrasonographic character of carotid plaques, and incidences of brain embolism in carotid angioplasty and stenting (CAS) and carotid endarterectomy (CEA).

Materials and Methods: CEA (22/25 symptomatic lesions) and CAS (17/20 symptomatic lesions) between 2007 and 2010. Embolic protection devices (15 occlusion and 5 filtering devices) were used during CAS. Carotid plaques were classified into three categories (I: calcificated, II: intermediately echogenic, III: echolucent). Magnetic resonance imaging (MRI) was used to investigate brain embolisms.

Results: Ultrasonographic character of the plaques in CEA cases (I: 4%, II: 88%, III: 8%) was different from the one in CAS cases (I: 10%, II: 90%, III: 0%). The incidence of brain embolism in the CAS cases was 52.6% while 0% in the CEA cases (p = 0.00037). CAS had high incidences of brain embolism in any plaques (I: 100%, II: 43.8%). In the most recent 9 procedures of CAS using occlusion devices, averaged number of embolic lesion was 1.0 (0 post operative day; 0 POD). The number increased as 1.4 (1 POD) and 2.0 (7 POD).

Conclusion: CEA should be currently the first choice for most patients with a high-grade and symptomatic carotid artery stenosis.

Key words: carotid endarterectomy, carotid angioplasty, brain embolism, diffusion-weighed magnetic resonance imaging, carotid plaque

INTRODUCTION

Carotid angioplasty (CAS) emerged as an alternative treatment for carotid artery stenosis for carotid endarterectomy (CEA) to prevent neurological ischemic events. Ironically, one of the frequent complications in these invasive procedures is brain infarction, most of which are caused by embolic particles from carotid plaques. Especially, ultrasonographic frequency of embolic events in CAS is significantly higher than in CEA.\(^1\) The release of embolic materials may be related to the character of culprit plaques.\(^2\) In the present study, we investigated ultrasonographic character of carotid plaques (i.e. echolucent, intermediately echogenic, and calcificated), and embolic events in CEA and CAS using diffusion-weighted magnetic resonance imaging (DW-MRI). The incidence of brain emboli was calculated in each category of ultrasonographic character, and safety and efficacy of both invasive procedures were evaluated.

MATERIALS AND METHODS

Between July 2007 and May 2010, 25 and 20 procedures of CEA and CAS were performed in our hospital.
Before the treatments, all culprit lesions were examined by ultrasonography, and the plaques were categorized into three categories. Category I indicates the lesion with a highly calcified plaque, II intermediately echogenic, and III echolucent. CAS and CEA were indicated for symptomatic carotid lesions (at least 3 weeks after the most recent infarction, transient ischemic attack) and asymptomatic high-grade stenotic lesions (>80% by NASCET method\(^3\)). CEA was performed on 22 symptomatic lesions, or and 3 asymptomatic lesions, with a high-grade stenosis (70%–97%, MEAN 91.7%), while CAS on 17 symptomatic and 3 asymptomatic lesions (80%–97%, MEAN 89.5%).

Basically, CAS was indicated for CEA high-risk cases (19 out of 20 lesion), as defined by SAPPHIRE inclusion criteria.\(^4\) In one case, although the patient had an ipsilateral radiation therapy for laryngeal cancer, the lesion with an echo-lucent plaque was treated by CEA. Embolic protection was performed using an occlusion balloon (Percu Serge, Medtronic, Santa Rosa, CA) in 15 CAS cases, or a filtering device (AngioGuard\(^\text{TM}\) XP, Cordis Corporation, Johnson and Johnson, Miami, FL) in 5 of CAS cases. A closed cell type stent (Wallstent, Boston Scientific Corporation, Boston, MA) was used in combination with the occlusion balloon, while an open cell type stent (Precise, Cordis Corporation, Johnson and Johnson, Miami, FL) was used with the filtering device.

All patients were examined by neurologists pre- and post-procedurally. Pre- and post-procedural DW-MRIs were performed to investigate the postprocedural brain emboli within a post-procedural week, using Signa Horizon (GE health care, Japan), at the settings of TR/TE = 1000/135 milliseconds, b factor = 1500, 6 mm thick slice, 2 mm gap, and matrix size 128 × 128. An acute ischemic lesion was diagnosed when it was seen on DW-MRI images. To be considered indicative of acute injury, the lesion could not appear on pre-procedural imaging, and no corresponding FLAIR abnormality could be present for the lesion to be considered as acute.

The temporal distribution pattern of the post-procedural embolism was examined in the most recent 9 procedures of CAS using an occlusion balloon for embolic protection and a closed cell type stent, using the results of repeated MRIs performed at either two of the following time-points: 0 post operative day (0 POD; 4 procedures), 24–48 hours (1 POD; 7 procedures), and a week after the procedure (7 POD; 5 procedures). The time points of consecutive MRIs were decided by the availability of MRI facility in our hospital. Thus, MRIs were available at 0 POD and 1POD (3 cases), 0 POD and 7 POD (1 case), and 1 POD and 7 POD (5 cases). For statistical analysis, Kai-square method and t-test were used if applicable.

**RESULTS**

**Neurological findings in the 30 postprocedural days**

One patient (5%) in the CAS group had ipsilateral reversible ischemic nerve damage, while one patient (4%) in the CEA group had a contra-lateral brain hemorrhage from brain metastasis of bladder cancer on the third postoperative day. Myocardial infarction, death, or cranial nerve injury was not experienced in these treatments.

**Postprocedural brain emboli detected by DW-MRI**

Incidence of brain embolism in the CAS case was 50.0%. On the other hand, DW-MRI detected no brain emboli in the CEA cases (p = 0.00037).

**Temporal distribution pattern of the postprocedural embolism**

The number of embolic lesion detected by MRI at <3 h was 1.0 on average. The number increased as 1.4 and 2.0, at 1 POD and 7 POD.

**Sonographic categories and brain emboli**

Constitution of ultrasonographic character of the plaques in CEA cases (I: 4%, II: 88%, III: 8%) was different from the one in CAS cases (I: 10%, II: 90%, III: 0%). CEA procedures resulted in 0 percent incidences of brain emboli, even in the cases of echo-lucent and risky plaques. On the other hand, CAS had high incidences of brain emboli in any ultrasonographic characters of plaques (I: 100%, II: 43.8%) (Fig. 1).

**DISCUSSION**

DW-MRI revealed that CAS caused micro-brain emboli (MBE) in half of the cases, although similar neurological results were provided to those of CEA. Because of the limited number of the cases, there may be an effect of a learning curve, which may decrease the embolic events in CAS cases. However, considering a high percentage of symptomatic cases involved (85%), the frequency of MBE did not seem significantly higher than the one in a larger scale study.\(^5\)

Symptomatic and asymptomatic carotid plaques seem to have considerably different pathophysiological charac-
sters. More or less, symptomatic carotid lesions can be considered to have such unfavorable features, that releases embolic particles when activated by unknown mechanisms. In fact, embolic events are ultrasonographically detected more frequently in patients with recent neurological symptoms, compared with patients having similarly high grades but asymptomatic carotid plaques. During the CAS procedures, direct contact of the devices with such vulnerable lesions inevitably influences the release of plaque fragments. Moreover, as detected in this series, CAS leaves the atheromatous plaque, which may cause post-procedural emboli. Thus, not only the usage of per-procedural embolic protection devices, but also understanding the pathophysiological character of the plaques is essential to improve the results of carotid intervention. We did not treat the cases with echolucent plaques with CAS, because it suggests the instability of the culprit lesion and a “CAS high-risk” character, such as lipid rich core in the plaque or newly formed thrombus.

Hypothesizing the outcomes of CAS is strongly influenced by the characters of plaques, our results suggested that applying CAS for all of these CEA cases would have made a similarly high incidence of brain emboli in the group II. Furthermore, a higher incidence of symptomatic emboli might have been detected in the cases with echolucent plaques (group III). Regarding asymptomatic emboli, there is a report indicating that the cognitive faculties in a great number of these “asymptomatic” patients are decreased. Recently published large trials have shown that the mid-term results of CAS were not extremely inferior, but apparently not superior to CEA, especially in terms of preventing new strokes. However, if we indicate CEA for all these CAS cases, there should have been high incidences of complications, including cranial nerve injuries, MI, and even death.

There are limitations in interpreting the results of this study. As afore mentioned, the small number of cases in the current study may have some effects on the results. Furthermore, the results of two different procedural protocols were involved in the CAS cases, mainly due to the availability of devices. However, we detected 2 cases of brain emboli in 5 procedures using an open cell type stent and a filtering device, while 8 in 15 using an occlusion balloon and a closed cell type stent. Therefore, the differences in device seemed to have a small effect on the results.

In conclusion, CEA should be currently the first choice for most patients with a high-grade and symptomatic carotid artery stenosis. CAS should be reserved for the case with such high CEA risks that the expected incidence of brain embolism is reasonably accepted.

References

Brain Microembolisms after CAS


