

## Editorial



# Proximal Protected Carotid Artery Stenting and Neurologic Intolerance: Can We Predict before Stenting?

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### Conflict of Interest

The author has no financial conflicts of interest.

The contents of the report are the author's own views and do not necessarily reflect the views of the *Korean Circulation Journal*.

► See the article “Low Common Carotid Artery Systolic Occlusion Pressure and Symptomatic Carotid Artery Stenosis Are Associated with Development of Neurologic Intolerance during Proximal Protected Carotid Artery Stenting” in volume 48 on page 217.

Carotid artery stenting (CAS) is an alternative treatment modality for the management of carotid artery stenosis in stroke prevention. CAS is a preferred strategy in patients with high risk of surgical endarterectomy. The current 2011 guideline for CAS recommended that embolic protection device (EPD) deployment can be beneficial to reduce the risk of stroke when the risk of vascular injury is low (class IIa, level of evidence C).<sup>1)</sup>

Now the EPD usage has become essential and mandatory during CAS. There are 2 major available EPDs in our daily clinical practice. Previous studies showed that the use of proximal protection device (PPD) during carotid stenting is safe and effectively protects the ischemic events during the CAS.<sup>2,3)</sup> However, this type of protection is difficult to use in a case of contralateral stenosis or occlusion, and therefore in such patients, as an alternative protection system, distal protection devices (DPDs) can be used to prevent microembolization, neurologic intolerance (NI) and ischemic stroke.

Despite the active usage of EPD, the neurologic complications can occur. The incidence of transient ischemic attack (TIA) is around 1–2% during the CAS. In the ACCULINK for Revascularization of Carotids in High-Risk Patients (ARCHeR) trial, the overall incidence of stroke was 5.5%, disabling stroke 1.5%, and minor events 2%.<sup>4)</sup> In the Carotid ACCULINK/ACCUNET Post Approval Trial to Uncover Unanticipated or Rare Events (CAPTURE) registry, the incidence of disabling stroke was 2% and the non-disabling stroke was 2.9%.<sup>5)</sup> Intracranial hemorrhage and hypoperfusion syndrome were known to be less than 1%.

It will safe and nice if we can predict the possibility of NI when we perform the proximal protected CAS. In this edition of the journal, Kwon et al.<sup>6)</sup> reported that the incidence of NI during CAS was approximately 40% and 88% of the patients were recovered after the balloon deflation. Among them, the incidence of stroke was higher in the NI group. Low common carotid artery systolic occlusion pressure (CCAOP) and symptomatic carotid artery stenosis are associated with the development of NI during proximal protected CAS.<sup>6)</sup> Authors suggested that the measurement of low systolic CCAOP was easy, simple and safe. They concluded that less than 42 mmHg was associated with NI during CAS with PPD.

These results were concordant with the previous study by Giugliano et al.<sup>7)</sup> using the occlusion pressure. They also suggested that the CCAOP less than 40 mmHg and concomitant occlusion of the contralateral internal carotid artery occlusion were the most powerful clinical predictors of NI.<sup>7)</sup>

Unlike the previous reports of the NI intolerance (6–30%), they experienced a higher incidence of NI in this study, suggesting the relatively higher incidence of NI in a Korean population with all comer carotid artery stenosis.<sup>7)</sup> In this study, because a neurology specialist was directly monitored the incidence of NI during the procedure, there might be very limited chance of neglecting symptoms and signs of NI, and this might be associated with higher incidence of NI as compared with other previous reports.

In daily clinical practice, the development of NI can be a major stress for the operator and can be more burden to the patient because of the higher incidence of real stroke. Thus, if we can predict the NI before the CAS, we may pay attention more strongly to the perfection of the whole procedure as a team approach. If we find these two significant predictors of NI (low CCAOP and symptomatic carotid artery stenosis), we may change the EPD from PPD to DPD for patient's safety even it requires more cost and time. If we find the insufficient or interrupted anterior communicating artery in the circle of Willis or concomitant contralateral internal carotid artery occlusion at pre-procedural magnetic resonance angiography or computed tomography angiography, it would be better to use the DPD instead of PPD.

There are limitations in this study. This is not a randomized study and a retrospective observational study in a single center with the relatively small patients. Instead of the single experienced operator, multi-center experienced operators with larger study population would provide more objective and powerful messages.

In conclusion, it will be safe to avoid PPD when we find the concomitant contralateral internal carotid artery occlusion, an insufficient anterior communicating artery in pre-procedural brain image studies, symptomatic carotid artery stenosis and low CCAOP at the time of CAS to reduce the incidence of NI and subsequent disabling ischemic stroke.

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