Periprocedural Monitoring with Regional Cerebral Oxygen Saturation in Carotid Artery Stenting

H. KAMII, K. SATO, Y. MATSUMOTO*, E. FURUI**, M. EZURA***, A. TAKAHASHI***

Departments of Neuroanesthesia, *Neuroendovascular Therapy, and **Stroke Neurology, Kohnan Hospital, and

***Department of Neuroendovascular therapy, Tohoku University Graduate School of Medicine, Sendai; Japan

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Summary

Hemodynamic instability during and after carotid artery stenting (CAS) may reduce cerebral blood flow (CBF), leading to cerebral ischemia. To investigate changes in CBF in the periprocedural period, we continuously recorded the regional cerebral oxygen saturation (rSO₂) using near-infrared spectroscopy.

In 46 consecutive patients with carotid artery stenosis, rSO₂ was continuously recorded during and after CAS. In addition, the patients underwent SPECT to evaluate a change in CBF on the next day after CAS.

Intraprocedural bradycardia (heart rate <50 bpm) occurred in 21 patients (46%) including one transient cardiac arrest. Intraprocedural hypotension (systolic blood pressure <80 mmHg) occurred in 18 patients (39%), and 16 of them showed prolonged hypotension. The rSO₂ in patients with bradycardia/hypotension during CAS was significantly less than that in patients without them ($p<0.01$). Moreover, the SPECT on the next day after CAS demonstrated that the ipsilateral CBF in patients with bradycardia/hypotension during CAS significantly more than that in patients without them ($p<0.05$).

Intraprocedural hemodynamic instability resulted in a significant decrease in rSO₂, leading to a possible severe cerebral ischemia. In addition, intraprocedural bradycardia/hypotension might be related with postprocedural hyperperfusion, causing the morbidity and mortality after CAS.

Introduction

Carotid artery stenting (CAS) has been introduced as an alternative to carotid endarterectomy for the treatment of carotid artery stenosis¹⁻⁴. Although the safety of CAS has been supported by the recent SAPHIRE trial⁵, bradycardia and hypotension often occur during and after CAS⁵⁻¹¹, and they might cause severe cerebral ischemia, leading to the morbidity and mortality⁶. However, there have been not enough informations about cerebral blood flow on such an occasion as bradycardia/hypotension during CAS procedures. Therefore, we continuously recorded the regional cerebral oxygen saturation (rSO₂) using near-infrared spectroscopy to investigate changes in cerebral blood flow in the periprocedural period of CAS.

Material and Methods

Between January 2004 and December 2005, CAS procedures were attempted at our hospital in 46 consecutive patients (40 men, six women, ranging in age from 48 to 82 years; mean age 70.7 years). Blood pressure and heart rate were monitored continuously throughout the procedure. The blood pressure was measured at five-minute intervals by an automated-cuff-inflation sphygmomanometer. Bradycardia was defined as heart rate less than 50 beats per minutes, and was treated with intravenous at-

Figure 1 Changes in HR (A) and SBP (B) during CAS. HR significantly decreased from 82.8 ± 2.7 to 59.6 ± 3.8 (bpm) ($n = 46$, $p < 0.001$), and SBP significantly decreased from 137.8 ± 3.3 to 95.2 ± 4.7 (mmHg) ($n = 46$, $p < 0.001$).

ropine sulfate. Hypotension was defined as systolic blood pressure less than 80 mmHg, and was treated with intravenous ephedrine hydrochloride.

The rSO₂ was continuously recorded using INVOS 4100 near-infrared spectrophotometers (Somanetics Corporation, Troy, MI, USA) during and after CAS. The rSO₂ sensor was attached on the forehead of a surgical side to evaluate cerebral oxygen saturation, which can reflect cerebral blood flow (CBF) when the cerebral metabolism is stable^{12,13}. In addition, single photon emission computed tomography (SPECT) was performed to evaluate a change in CBF on the next day after CAS.

In the interventional neuroradiology suite,

CAS was performed using a transfemoral approach under cerebral protection afforded by a PercuSurge GuardWire (Medtronic Inc., Santa Rosa, CA, USA) balloon occlusion device. After the occlusion balloon was inflated in the distal ICA, the lesion was pre-dilated, and a self-expandable stent was deployed. Then, the blood was aspirated through the aspiration catheter, and the protection balloon was deflated. After evaluating a diameter of the lesion with intravascular ultrasound sonography, the protection balloon was inflated again, and post-dilatation was cautiously performed. A final angiogram was obtained to confirm that the dilatation was adequate and no local complications such as dissection occurred.

Results

During CAS, HR significantly decreased from 82.8 ± 2.7 to 59.6 ± 3.8 (bpm, mean $\pm$ SE) ($n = 46$, $p < 0.001$), and SBP significantly decreased from 137.8 ± 3.3 to 95.2 ± 4.7 (mmHg) ($n=46$, $p < 0.001$) (figure 1A, 1B). Bradycardia (<50 bpm) occurred in 21 cases (46%) of the patients, including a case of cardiac arrest, and hypotension (<80 mmHg) occurred in 18 cases (39%) of them. Moreover, in 16 cases of them, the hypotension prolonged after CAS, and intravenous norepinephrine was continuously injected to maintain blood pressure.

The rSO₂ showed significant changes during the periprocedural period (figure 2). In all 46 cases, a baseline of rSO₂ before the procedure was $61.6 \pm 0.8\%$. During only test occlusion with a PercuSurge GuardWire, the rSO₂ significantly decreased to $58.2 \pm 0.9\%$ ($p < 0.001$). Then, just after post-dilatation, the rSO₂ significantly dropped to $52.1 \pm 1.4\%$ ($p < 0.001$). The decrease in rSO₂ after post-dilatation in eight cases with bradycardia and hypotension (13.4 ± 1.5 points) was significantly more than that in nine cases without both of them (4.9 ± 1.1 points) ($p < 0.01$) (figure 3).

The ipsilateral CBF (%) on the next day after CAS was calculated by a ratio of CBF on the ipsilateral to contralateral side of the CAS. In cases that showed a vascular reserve less than 30% in SPECT before CAS, the ipsilateral CBF in seven cases with bradycardia and hypotension ($109.8 \pm 4.9\%$) was significantly more than that in nine cases without both of them ($96.5 \pm 8.3\%$) ($p < 0.05$) (figure 4).

Discussion

Distension of the carotid bulb leads to stimulation of the baroreceptor, which increases parasympathetic stimulation to the heart causing bradycardia, and inhibits sympathetic stimulation to the blood vessels causing hypotension¹⁰. In previous reports, hemodynamic instability including bradycardia and hypotension often occurred during and after CAS⁵⁻¹¹. Also in our studies, bradycardia (<50 bpm) and hypotension (<80 mmHg) occurred during CAS in 46% and 39%, respectively. Since patients with the hemodynamic instability are at a significantly increased risk of a periprocedural major adverse clinical events or stroke⁶, it is important to perform the rapid recovery from

Figure 2 Changes in rSO₂ during CAS procedures. In all 46 cases, a baseline of rSO₂ before the procedure was $61.6 \pm 0.8\%$. During only test occlusion with a PercuSurge GuardWire, the rSO₂ significantly decreased to $58.2 \pm 0.9\%$ ($p < 0.001$). Then, just after post-dilatation, the rSO₂ significantly dropped to $52.1 \pm 1.4\%$ ($p < 0.001$).

bradycardia/hypotension and the prophylactic management avoiding them.

The regional cerebral oxygen saturation (rSO₂) indicates a change in the balance between the oxygen supply and demand using near-infrared spectroscopy. In a stable state of cerebral metabolism, therefore, rSO₂ demonstrates the oxygen supply, which can reflect

Figure 3 Influences of bradycardia/hypotension on rSO₂ after post-dilatation. The decrease in rSO₂ after post-dilatation in eight cases with bradycardia and hypotension (13.4 ± 1.5 points) was significantly more than that in nine cases without both of them (4.9 ± 1.1 points) ($p < 0.01$) ($p < 0.01$).

Figure 4 Relationship between intraoperative bradycardia/hypotension and postoperative hyperperfusion. In cases that showed a vascular reserve less than 30% in SPECT before CAS, the ipsilateral CBF in seven cases with bradycardia and hypotension ($109.8 \pm 4.9\%$) significantly more than that in nine cases without both of them ($96.5 \pm 8.3\%$) ($p < 0.05$).

CBF, under the sensor^{12,13}. In our previous studies on rSO₂ changes in balloon test occlusion for giant aneurysm surgery¹⁴, we showed that a decrease in rSO₂ during the balloon occlusion was significantly related with a decrease in CBF measured by SPECT. Also in CAS, it is believed that a change in rSO₂ can indicate a change in CBF. However, no reports have demonstrated intraoperative monitoring of rSO₂ in CAS, although there have been a lot of studies on rSO₂ changes in carotid endarterectomy (CEA)¹⁵. In the present study, we showed that the rSO₂ during CAS in patients with bradycardia and hypotension significantly less than that in patients without both of them ($p < 0.01$). That is, bradycardia/hypotension can cause a decrease in CBF, which might induce

severe cerebral ischemia, resulting in the morbidity and mortality in CAS.

There have been few reports demonstrating a change in CBF after CAS. Henry et al¹⁶ reported that the incidence of cerebral hyperperfusion syndrome was 3.5% in their series of 57 patients. Meyers et al¹⁷ demonstrated the incidence of the syndrome was 5% in 140 patients. In the present study, hyperperfusion syndrome occurred in one (2.8%) of all 46 patients. When ipsilateral hyperperfusion is defined as a state with CBF more than 110% over the contralateral hemisphere in SPECT on the next day after CAS, it occurred in seven (24.1%) out of 29 patients in our series. Interestingly, in patients that showed a vascular reserve less than 30% in SPECT before CAS, the ipsilateral CBF after CAS in patients with bradycardia and hypotension during CAS significantly more than that in patients without both of them ($p < 0.05$). Therefore, intraoperative bradycardia/hypotension, even if the recovery from them is rapid, may be related with hyperperfusion after CAS. We believe that prevention of bradycardia/hypotension as well as rapid recovery from them during CAS is important to reduce the morbidity and mortality after CAS.

Conclusions

In 46 consecutive patients treated with CAS, we demonstrated that patients with bradycardia/hypotension during CAS showed a significant rSO₂ decrease compared with patients without them.

In addition, intraoperative bradycardia/hypotension might be related with postoperative hyperperfusion measured by SPECT. It is conceivable that prevention of bradycardia/hypotension as well as rapid recovery from them during CAS is important to reduce the morbidity and mortality after CAS.

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Hideyuki Kamii, M.D.
 Department of Neuroanesthesia
 Kohnan Hospital
 4-20-1 Nagamachi-minami, Taihaku-ku
 Sendai 982-8523, Japan
 E-mail: kamii@kohnan-sendai.or.jp