ORIGINAL ARTICLE doi: 10.2176/nmc.oa.2016-0155

Neurol Med Chir (Tokyo) 57, 115–121, 2017 Online January 31, 2017

Complications and Predictors of Hypotension Requiring Vasopressor after Carotid Artery Stenting

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Abstract

A significant controversy exists regarding the clinical impact of hemodynamic depression on major adverse events after carotid artery stenting (CAS). The purpose of this study was to evaluate the incidence, predictors, and clinical significance of hypotension after CAS. A total of 118 cases of carotid artery stenosis were treated with CAS. Hypotension was defined as sustained systolic blood pressure <80 mmHg and requiring intravenous administration of vasopressor to maintain adequate systolic blood pressure after the procedure. Baseline characteristics, procedural characteristics, and periprocedural major adverse events were retrospectively compared between postprocedural hypotension group and non-hypotension group. Morphological and procedural characteristics were not significantly different between the two groups. Periprocedural major adverse events, presence of new ischemic lesions, and number of new ischemic lesions were not significantly different between the two groups ($P = 1$, $P = 0.36$, $P = 0.68$). Hypertension was an independent protective factor ($P = 0.037$), and use of proximal protection and the distance from carotid bifurcation to maximum stenotic lesion ≤ 10 mm were independent risk factors for developing hypotension after CAS ($P = 0.034$, $P = 0.027$). There was no significant relationship between hypotension after CAS and major adverse events in this study. Maintenance of periprocedural adequate cerebral perfusion is thought to be important to prevent ischemic complications due to hypotension after CAS, especially in these cases.

Key words: carotid artery stenosis, carotid artery stenting, postprocedural hypotension, periprocedural complication, predictor

Introduction

After a recent prospective and randomized trial[1] that demonstrated no differences in the composite outcomes of stroke, death, and myocardial infarction (MI) between carotid endarterectomy (CEA) and carotid artery stenting (CAS), CAS has become an alternative option to CEA for the treatment of carotid artery stenosis. Hemodynamic depression (HD), which includes hypotension and bradycardia, has been recognized a frequent phenomenon during and after CAS.[2–10] There are some reports concerning HD or hypotension after CAS. Recent review reported that the frequency of HD was 7.2–80%.[11] Persistent hypotension following CAS has been reported in 7–34%,[12] 48–72.7%.[9] The mechanical effects of balloon angioplasty and stenting at the level of the carotid bulb are known to induce HD due to local baroreceptor reflex. Such HD may lead to a temporary reduction in cerebral perfusion,[13,14] and may injure the ability of washing out the microemboli.[7]

Still now, a significant controversy exists regarding the clinical impact of HD on major adverse events. The factors that necessitate the vasopressor administration after CAS have not been defined fully. The purpose of this study was to evaluate the incidence, predictors, and clinical significance of hypotension, after CAS.

Received May 24, 2016; Accepted August 8, 2016

Materials and Methods

We retrospectively analyzed the medical records of patients who underwent CAS for symptomatic and asymptomatic carotid artery stenosis at our institute from April 2010 to August 2015. During this period, 122 cases were treated with CAS. Among them, four cases who suffered progressive stroke and

were treated in the acute phase were excluded from this study. Finally 118 cases were included in this retrospective study.

Morphological characteristics of the carotid artery lesion were evaluated on digital subtraction angiography (DSA), magnetic resonance (MR) plaque imaging, three dimensional computed tomography angiography (3DCTA) and carotid duplex ultrasound. The degree of stenosis was measured according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria.[15] Vulnerable plaque was defined as a high intensity area in the plaque compared with the sternocleidomastoid muscle detected by MR plaque imaging, or echolucent plaque detected by carotid duplex ultrasound. Pseudo-occlusion was defined according to the criteria reported by Hirata et al.[16] In symptomatic patients, CAS procedure was performed more than 3–4 weeks after symptom onset. The indications for CAS were carotid artery stenosis ≥ 50% in symptomatic patients and ≥ 80% in asymptomatic patients. Existence of coronary artery stenosis or occlusion was examined before the CAS procedure in all cases. The coronary artery disease was treated prior to the CAS if the lesion was judged as a significant stenosis with the necessity of interventional treatment by cardiologists. There was no patient who inserted a temporary pacemaker against the postprocedural hypotension.

CAS procedure

All patients received statins at least 14 days before the procedure and dual antiplatelet therapy with aspirin and clopidogrel, aspirin and cilostazol, or clopidogrel and cilostazol at least 7 days before the procedure. All CAS procedures were performed under local anesthesia and intravenous sedation by specialized neuroendovascular physicians. Under adequate systemic heparin administration, predilatation, self-expandable stent placement, and postdilatation, if necessary were performed using embolic protection devices (EPDs). CAS technique including most appropriate EPDs and stent type was at operator's discretion according to the morphological and clinical characteristics. EPDs included distal filter protection, distal balloon protection, proximal protection as a flow reversal technique and a combination of distal and proximal protection. For tight stenosis, vulnerable plaque, or thrombus-containing plaque, proximal protection was preferentially used. Our stent selection strategy was as follows: Closed-cell stent (Carotid Wallstent Monorail, Boston Scientific, Natick, MA, USA) is suitable for cases of vulnerable plaque and straight lesions. Open-cell stent (Precise, Cordis Corporation, Miami, Lakes, FL, USA; and Protégé, ev3 Inc., Plymouth, MN, USA) is suitable for cases of tortuous lesions and fibrous or calcified lesions. Atropine was administered prophylactically just before predilatation in all cases.

Postprocedural management

Blood pressure, heart rate, and neurological symptoms were closely monitored after the CAS procedure. Vasopressor was administrated intravenously when systolic blood pressure (sBP) was decreased under 80 mmHg and sustained. Postprocedural diffusion weighted (DW)-magnetic resonance imaging (MRI) was performed within 72 hours after the CAS procedure, and was evaluated for the presence of new ischemic lesions in the ipsilateral hemisphere compared with DW-MRI before the CAS procedure.

We compared baseline characteristics of the patients, procedural characteristics, and periprocedural major adverse events (MAE) which included any stroke, MI and death within 30 days after the CAS procedure, and new ischemic lesions detected on postprocedural DW-MRI between postprocedural hypotension group (HG) and non-hypotension group (NHG). Stroke including any minor or major stroke was defined as a neurological deficit that persisted for >24 hours. Death was defined as any death related to neurovascular causes. Diagnosis of MI was based on the presenting symptoms and evidence of impaired heart function found by electrocardiography and abnormal troponin levels. Neurological complications were defined as transient ischemic attack (TIA), stroke, and death within 30 days after CAS. Hypotension was defined as sustained sBP <80 mmHg and requiring intravenous administration of vasopressor.

Statistical analysis

Univariate analysis was used to compare the HG and NHG. Fisher's exact test was used to analyze category comparisons. Mann-Whitney *U* test and standard *t*-test were used to compare continuous variables as appropriate. The multivariate logistic regression analysis was performed using a backward stepwise selection to detect clinical and radiological parameters that were independent predictors of HD after CAS. Statistical significance was defined as $P < 0.05$. Statistical analyses were performed using R 3.1.2 (The R Foundation for Statistical Computing, Vienna, Austria).

This study was approved by Institutional Review Board of Kyoto Second Red Cross Hospital (approval number: S27–29).

Results

The patients included 103 men and 15 women, with a mean age of 74.9 years (range 55–87). CAS was

performed in 81 symptomatic cases (68.6%) and 37 asymptomatic cases (31.4%). EPDs were used in all cases. Out of 118 cases, 26 (22.0%) cases experienced hypotension. The baseline characteristics of the patients are summarized and compared in Table 1. There were no significant differences with respect to age, sex, diabetes mellitus, dyslipidemia and coronary artery disease between the two groups. Hypertension was significantly less frequent in the HG compared with the NHG (69.2% vs 90.1%; P = 0.023). Morphological characteristics of the lesions and procedural characteristics are summarized and compared in Tables 2 and 3. The preprocedural stenosis rate was significantly higher in the HG compared with the NHG (84.5% vs 80.3%; P = 0.047). However, lesion length, internal carotid artery (ICA) diameter, the distance from the carotid bifurcation to the maximum stenotic lesion (MSL), pseudo occlusion, soft plaque or severe calcification were not significantly different between groups. Open-cell stent was significantly more frequently used in the HG compared with the NHG (76.9% vs 50.0%; P = 0.024), on the other hand, closed-cell stent was significantly less frequently used in the HG compared with the NHG (19.2% vs 47.8%; P = 0.012). Proximal protection was significantly more frequently used in the HG compared with the NHG (57.7% vs 33.7%; P = 0.04). Stent diameter, postprocedural residual stenosis rate, ICA/stent diameter ratio, or dilatation ratio (pre/post procedural stenosis ratio) were not significantly different between groups. The outcomes of the CAS procedure are summarized and compared in Table 4. The overall 30-day stroke occurred in six cases (5.1%). Stroke occurred in one case (3.8%) of the HG and in five cases (5.4%) of the NHG (no significant difference, P = 1). No patient experienced postprocedural MI in either group. New ischemic lesions detected on postprocedural DW-MRI were observed in 12 cases of the HG group (46.2%) and 32 cases of the NHG (35.2%). Most of these lesions were asymptomatic and there was no significant difference between groups (P = 0.36). Number of new ischemic lesions detected on postprocedural DW-MRI was not also significantly different between groups (P = 0.68).

Predictors of postprocedural hypotension

Multivariate logistic regression analysis showed that hypertension (odds ratio (OR) 0.26, 95% confidence interval (CI) 0.08 to 0.92, P = 0.037) was an independent protective factor, while proximal protection (OR 3.06, 95% CI 1.09 to 8.62, P = 0.034) and the distance from carotid bifurcation to MSL ≤ 10 mm (OR 3.4, 95% CI 1.15 to 10.1, P = 0.027) were independent risk factors for developing hypotension after CAS (Table 5).

Discussion

HD frequently occurs during and after the CAS procedure, and has been reported to associate with an increase in periprocedural MAEs. The frequencies of hypotension or HD were different between studies because the definitions for hypotension or HD as a composite measure differed between studies. [2,7,17,18] Nonaka et al. reported postprocedural hypotension in 42.4%, of which medical treatment was necessary in 21.2%.[9] Wu et al. reported that the incidence of hypotension was 10.6%.[10] In this study, hypotension occurred in 22.0% of the patients.

When carotid sinus baroreceptors are dilated and expanded by the balloon or stents, stimulation of the carotid sinus inhibits sympathetic neurons in the nucleus tractus solitarius (NTS) and reduces sympathetic tone to peripheral blood vessels, leading to a reduction in systemic blood pressure.[7,19] Impulses from the carotid sinus also stimulate the nucleus ambiguus and the dorsal vagal activity results in bradycardia.[9] Atherosclerosis and hypertension lower the baroreceptors' sensitivity because the carotid sinus becomes stiffer and more resistant to deformation.[20–24] That might be the reason why hypertension was significantly more frequent in the NHG compared with the HG in this study. The preprocedural stenosis rate was significantly

Table 1 Baseline characteristics of the patients

		Hypotension group (n = 26)	Non-hypotension group (n = 92)	test	P value
age	mean (SD)	74.5 (7.8)	74.7 (6.7)	unpaired t test	0.88
sex, man	n (%)	22/26 (84.6%)	81/92 (88.0%)	Fisher	0.74
HT	n (%)	18/26 (69.2%)	82/91 (90.1%)	Fisher	0.023
DM	n (%)	7/26 (26.9%)	40/91 (44.0%)	Fisher	0.17
dyslipidemia	n (%)	15/26 (57.7%)	58/91 (63.7%)	Fisher	0.65
CAD	n (%)	8/26 (30.8%)	30/92 (32.6%)	Fisher	1

CAD: coronary artery disease, DM: diabetes mellitus, HT: hypertension.

Table 2 Morphological characteristics of the patients

		Hypotension group (n = 26)	Non-hypotension group (n = 92)	Test	*P* value
left	n (%)	13/26 (50.0%)	45/92 (48.9%)	Fisher	1
symptomatic	n (%)	14/26 (53.8%)	67/92 (72.8%)	Fisher	0.093
length					
lesion length	median (IQR)	18.3 mm (13.3–24.6)	20.4 mm (15.0–26.0)	Mann-Whitney *U*	0.3
ICA diameter	median (IQR)	5.2 mm (4.8–6.2)	5.1 mm (4.7–5.7)	Mann-Whitney *U*	0.19
bifurcation to MSL	median (IQR)	7.2 mm (4.5–11.8)	10.1 mm (6.2–15.5)	Mann-Whitney *U*	0.053
bifurcation to MSL ≤ 10 mm	n (%)	17/26 (65.4%)	44/91 (48.4%)	Fisher	0.18
lesion					
preprocedural stenosis rate	median (IQR)	84.5 (76.7–91.9)	80.3 (69.5–87.0)	Mann-Whitney *U*	0.047
soft plaque	n (%)	17/25 (68.0%)	79/92 (85.9%)	Fisher	0.073
severe calcification	n (%)	1/26 (3.8%)	2/92 (2.2%)	Fisher	0.53

ICA: internal carotid artery, MSL: maximum stenotic lesion.

Table 3 Procedural characteristics of the patients

		Hypotension group (n = 26)	Non-hypotension group (n = 92)	Test	*P* value
type of stent					
open-stent	n (%)	20/26 (76.9%)	46/92 (50.0%)	Fisher	0.024
closed-stent	n (%)	5/26 (19.2%)	44/92 (47.8%)	Fisher	0.012
combination	n (%)	1/26 (3.8%)	2/92 (2.2%)	Fisher	0.53
stent diameter	median (IQR)	10 (9–10)	10 (9–10)	Mann-Whitney *U*	0.46
proximal protection	n (%)	15/26 (57.7%)	31/92 (33.7%)	Fisher	0.04
postprocedural residual stenosis rate	median (IQR)	25.2% (13.6–34.1)	17.5% (9.7–26.0)	Mann-Whitney *U*	0.051
ICA/stent diameter ratio	median (IQR)	60.2% (51.8–66.8)	54.2% (48.5–62.1)	Mann-Whitney *U*	0.062
dilatation percentage (pre/post stenosis ratio)	median (IQR)	61.2% (49.6–67.6)	60.3% (47.7–70.3)	Mann-Whitney *U*	0.9

ICA: internal carotid artery.

higher in the HG. Lavoie et al. reported that a larger difference in stenosis severity before and after CAS was associated with a higher risk of developing hypotension or bradycardia that required drug intervention.[6] Carotid sinus baroreceptors might be more severely stretched and baroreceptor reflex could be more frequently induced by the CAS procedure in cases with severe carotid artery stenosis. The type of stents used in a CAS procedure was significantly different between the two groups in this study. Open-cell stents were significantly more frequently used in the HG. Closed-cell stents were significantly more frequently used in NHG. The difference of the intrinsic property of the stent cell design and radial force of the stent could affect the hypotension or HD. Open-cell stents put more pressure on the carotid sinus compared with closed-cell stents. Nii et al. reported that the type of self-expandable stent placed may affect the risk of procedural HD in patients undergoing CAS.[8] Proximal protection was also more frequently used in the HG compared with the NHG in this study. To the best of our knowledge, there have been no reports concerning the relationship between postprocedural hypotension and proximal protection. We could not make clear the reason why proximal protection induced hypotension, however, balloon pressure on the long segment of the arterial wall from common carotid artery (CCA) to ICA may induce the baroreceptor reflex more frequently.

Table 4 Clinical and radiological outcomes of the patients

		Non-hypotension group	Hypotension group	test	*P* value
MAE (≤ 30 day)	n (%)	5/92 (5.4%)	1/26 (3.8%)	Fisher	1
stroke (≤ 30 day)	n (%)	5/92 (5.4%)	1/26 (3.8%)	Fisher	1
MI (≤ 30 day)	n (%)	0/92 (0%)	0/26 (0%)	Fisher	NA
death (≤ 30 day)	n (%)	0/92 (1%)	0/26 (1%)	Fisher	NA
New DWI high lesion	n (%)	32/91 (35.2%)	12/26 (46.2%)	Fisher	0.36
DWI high lesion number	median (IQR)	0 (0–1)	0 (0–1)	Mann-Whitney *U*	0.46
0	n (%)	61/91 (67.0%)	15/26 (57.7%)	Fisher	0.68
1	n (%)	13/91 (14.3%)	6/26 (23.1%)		
2 to 5	n (%)	9/91 (9.9%)	2/26 (7.7%)		
>5	n (%)	8/91 (8.8%)	3/26 (11.5%)		

DWI: diffusion weighted image, MAE: major adverse event, MI: myocardial infarction.

Table 5 Multivariate analysis for predictor of hypotension after CAS procedure

	Odds ratio	95%CI	*P* value
HT	0.26	0.08 – 0.92	0.037
Closed stent	0.37	0.11 – 1.19	0.095
Proximal protection	3.06	1.09 – 8.62	0.034
Bifurcation to MSL ≤ 10 mm	3.4	1.15 – 10.1	0.027

CAS: carotid artery stenting, CI: confidence interval, HT: hypertension, MSL: maximum stenotic lesion.

The association with periprocedural HD and increased rate of MAE is controversial. The main source for periprocedural ischemia after CAS is a thromboembolism. DW-MRI can detect ischemia of the brain within minutes of onset, and is currently the best method for evaluating procedure related cerebral ischemic lesions after CAS.[25] Caplan et al. also reported that hypotension associated with CAS may increase the risk of cerebrovascular events by impairing the distal washout of residual emboli not intercepted by the protection device.[26] Aysun et al. reported that prevention of severe hypotension and bradycardia might reduce the number of new DW-MRI lesions after CAS.[2] In this study, there was no significant association between hypotension and thromboembolism detected on DW-MRI. MAE also was not significantly different between the two groups. In our patients, when hypotension occurred after the CAS procedure, the patients were treated with immediate intravenous vasopressor administration and discontinuance of any oral antihypertensive medications to maintain the adequate systolic blood pressure and cerebral perfusion. That might be the reason of no significant difference with the respect to the new lesions detected on DW-MRI and MAE between the two groups in this study.

Prediction of hypotension after CAS is important for preventing periprocedural ischemic complications. Diabetes mellitus, severe calcified plaque, balloon dilatation pressure, octogenarians, contralateral occlusion, female sex, distance from carotid bifurcation to MSL, eccentric plaque formation, open-cell stent, and asymptomatic lesion are the reported risk factors that are independently associated with HD after CAS.[3,5–10] Our study revealed that hypertension was an independent protective factor, and proximal protection was independent risk factors for developing hypotension after CAS. The distance from the carotid bifurcation to MSL ≤ 10 mm was not significantly different between the two groups, but multivariate analysis revealed an independent risk factor for developing hypotension after CAS. In some reports, the distance from the carotid bifurcation to MSL ≤ 10 mm was significantly associated with hypotension.[6,9] The body of the carotid bulb is a dilated segment of the ICA at its origin from the CCA, which is a remnant of the third aortic arch and the third pharyngeal arch, and is innervated by the nerves of the third arch, the glossopharyngeal nerve. In contrast, the apex of the carotid bulb is partially confined to the carotid bulb and is less affected by stimulation of the glossopharyngeal nerve.[27] When a self-expandable stent is deployed at the stenotic lesion, a maximum force of dilatation may affect the arterial wall at the lesion of high-grade stenosis on DSA. That may be why hypotension was more frequently occurred in lesion near the carotid bifurcation.

It is important that patients with severe hypotension after CAS should be treated with intravenous fluids and vasopressor administration,

and discontinuance of any oral antihypertensive medication to maintain adequate cerebral perfusion to prevent ischemic complications.

There are some limitations of the current study. First, this study was a retrospective non-randomized study. Second, we did not evaluate a balloon dilatation pressure. Prospective randomized clinical trial is needed to confirm our results.

Conclusion

Hypotension occurs frequently after CAS. However, there was no significant relationship between hypotension after CAS and MAE. Hypertension is a protective factor and the distance from the carotid bifurcation to MSL ≤ 10 mm and proximal protection are predictive factor for hypotension after CAS. Prediction of hypotension and maintenance of adequate cerebral perfusion after CAS are thought to be important to prevent ischemic complications.

Conflicts of Interest Disclosure

All authors have no conflicts of interest with regard to the manuscript submitted. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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