Risk factors and frequency of postprocedural hemodynamic instability after carotid artery stent placement

Czynniki ryzyka i częstość występowania niestabilności hemodynamicznej po zabiegach implantacji stentu do tętnicy szyjnej

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Abstract. The frequency, risk factors and long term consequences of reflexive postprocedural hypotension (PH) following carotid artery stenting (CAS) are not well known. Prospective analysis of 30 patients with 6-month follow-up undergoing CAS with an emboli-protection device was performed. A validated 24-hour ABPM was taken 24 hours before and after CAS. PH was defined as systolic blood pressure (SBP) <90 mm Hg, or decrease in mean arterial BP (MAP) of ≥20% or systolic BP (SBP) of ≥30 mm Hg of baseline BP reading. Neurological assessments were performed 24 hours after CAS and at 6 month follow-up visit. Median age was 69 years, 70% were male, 86% of patients had symptomatic carotid stenosis. Twenty patients (67%) experienced PH, 43% had transient bradycardia, 30% had both PH and bradycardia. The cumulated postprocedural mean SBP and DBP decreased from baseline 128/67 mm Hg to 108/54 mm Hg ($p < 0.01$), mean day (69/min) and night HR (58/min) decreased to respectively 58/min and 49/min ($p < 0.01$). We found no association of PH with age, ischemic heart disease, bifurcation involvement, balloon size, inflation pressure, longer lesion length. Patients with PH significantly ($p < 0.05$) less often were treated with Ca-antagonist (25% vs 70%), more often had ipsilateral ulcerated plaque (85% vs 50%) and had hemodynamically significant stenosis of contralateral ICA (60% vs 30%). During 6 month follow-up only 1 case of neurological deterioration was noticed. PH was a common phenomenon after CAS, however it did not result in neurological complications. Patients at risk can be possibly identified through clinical and angiographic variables.
Key words: carotid artery stenting, hemodynamic instability, postprocedural hypotension, stroke

Streszczenie. Częstość występowania, czynniki ryzyka i odległe konsekwencje odruchowego niedociśnienia tętniczego (NDT) po zabiegu stentowania tętnic szyjnych (CAS) nie są dokładnie określone. Przeprowadzono prospektywną analizę z 6-miesięczną obserwacją 30 kolejnych chorych leczonych CAS w prewencji udaru mózgu. U wszystkich wykonano okołozabiegowe 24-godzinne holterowskie monitorowanie ciśnienia tętniczego: NDT zdefiniowano jako okołozabiegowy spadek ciśnienia skurczowego (SBP) <90 mm Hg lub średniego dobowego ciśnienia (MAP) o ≥20%, lub SBP o ≥30 mm Hg względem wartości sprzed zabiegu. Ocena neurologiczna była przeprowadzona 24 godziny po CAS oraz 6 miesięcy po zabiegu. Mediana wieku wyniosła 69 lat, 70% grupy badanej stanowili mężczyźni, u 86% rozpoznano objawowe zwężenie tętnicy szyjnej. U 20 pacjentów (67%) ustalono rozpoznanie NDT, u 43% stwierdzono przejściową bradykardię, a u 30% NDT i bradykardię. Skumulowane średnie pozabiegowe SBP i ciśnienie rozkurczowe (DBP) obniżyły się z wyjściowo 128/67 mm Hg do 108/54 mm Hg ($p < 0,01$), średnia częstotliwość rytmu serca w ciągu dnia (69/min) i nocy (58/min) spadły do odpowiednio 58/min i 49/min ($p < 0,01$). Nie stwierdzono związku pomiędzy NDT a wiekiem, chorobą niedokrwienną serca, zajęciem rozwidlenia tętnicy szyjnej, przebiegiem zabiegu – wielkością balonu, ciśnieniem jego napełniania oraz długością blaszki miażdżycowej. Pacjenci z NDT istotnie rzadziej ($p < 0,05$) byli leczeni blokerami kanału wapniowego (25% *vs* 70%), częściej mieli stwierdzaną tożstronną do stentu owrzodziałą blaszkę miażdżycową (85% *vs* 50%) i przeciwstronnie istotne hemodynamicznie zwężenie tętnicy szyjnej wewnętrznej (60% *vs* 30%). W trakcie 6-miesięcznej obserwacji tylko u jednego pacjenta wystąpiło pogorszenie stanu neurologicznego. Wyniki badania wskazują, że NDT jest częstym zjawiskiem po CAS, ale nie wiąże się z istotnym ryzykiem powikłań neurologicznych. Charakterystyka kliniczna pacjenta i parametry angiograficzne mogą ułatwić ocenę ryzyka wystąpienia NDT.

Słowa kluczowe: stentowanie tętnic szyjnych, udar mózgu, niestabilność hemodynamiczna, hipotonia pozabiegowa

Nadesłano: 7.05.2021. Przyjęto do druku: 22.09.2021
Nie zgłoszono sprzeczności interesów.
Lek. Wojsk., 2021; 99 (4): 162–168

doi: 10.53301/lw.2100

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Introduction

Carotid endarterectomy (CEA) and carotid artery stenting (CAS) are effective long-term stroke prevention strategies in both symptomatic and asymptomatic patients. The most serious acute complication associated with CAS is stroke, which can occur due to thromboembolism, hyperperfusion syndrome, hemorrhage or hypoperfusion. Acute and/or prolonged hemodynamic instability (HI) involving hypotension or bradycardia during CEA or CAS is a well-recognized phenomenon which occurs in 29–51% of patients [1]. The clinical significance of this phenomenon is not known. A clinical syndrome of intraprocedural and postprocedural hypotension (PH) has been described in association with carotid endarterectomy, and surgical manipulation or compression of the carotid bulb. Activation of highly stretch-sensitive baroreceptors during carotid stent placement and balloon angioplasty may activate an autonomic system resulting in cardiodepression and vasodilatation. That may lead to intraprocedural syncope and/or cerebral hypoperfusion in some patients [2]. Current studies revealed that up to 30% of patients require transient vasopressors due to postoperative hypotension following CAS [3]. Profound hypotension may lead to the neurological complications after CAS especially to watershed infarcts in patients with a severe stenosis of the contralateral carotid artery [4]. It also has been associated with increased incidence of other major vascular (e.g. cardiac) adverse events in the periprocedural period. However, the frequency, risk factors and long-term consequences of fluctuations in blood pressure (BP) have not been clearly defined. Until now, no recommendations have been published concerning the preprocedural medical management including the need of antihypertensive drugs withdrawal prior to angioplasty or maintenance of prolonged hypotension following the procedure. Majority of published studies concentrated on short-term observation of PH complications, used manual BP measurements, and did not assess 24 h BP variability following CAS [5–7]. Therefore, the objective of this study was to evaluate the frequency and factors associated with PH defined and diagnosed by the use of 24 h BP Holter monitoring and to assess risk of neurological deterioration in longer observation.

Methods

Prospective analysis of consecutive 30 patients with 6-month follow-up undergoing elective CAS (self-expanding stent with an emboli-protection device) due to symptomatic or asymptomatic internal carotid artery (ICA) stenosis who were ineligible for CEA [8]. The major inclusion criteria included asymptomatic patients with >60% stenosis (according to ACAS study) and symptomatic patients with stenosis of at least 50% stenosis [9].

24-hour Ambulatory Blood Pressure Monitoring (ABPM)

A validated ABPM was performed 24 hours before and immediately after CAS using a portable non-invasive oscillometric and auscultatory device (Schiller MT-300). Measurements over a 24-hour period were recorded every 15 minutes during daytime hours (07.00 to 23.00) and every 30 minutes during night-time hours (23.00 to 7.00). For recordings to be considered valid, a minimum of 15 daytime and 8 night-time measurements were required. The type of device and the time of application (±3 hours) was the same in all patients. The patients were instructed to attend their usual day-to-day activities. Mean 24-hour arterial blood pressure (MAP), daytime and night-time average systolic BP (SBP), diastolic BP (DBP) were calculated and recorded automatically. Bradycardia and PH were defined as a heart rate and a SBP less than 60 beats/min and 90 mm Hg, respectively, or decrease in MAP of ≥20% of baseline BP reading or decrease in SBP of ≥30 mm Hg of baseline that lasted at least 1 hour during or after procedure as defined according to the CAVATAS Study [10]. The patients were categorized by the presence (group PH) or the absence (group APH) of PH. Antihypertensive medication were maintained before and after CAS, and dosage was adjusted according to BP measurements. In case of hypotonia patients routinely receive intravenous 1000 ml of 0.9% natrium chloratum and had reduction of hypotensive drugs and diuretics.

Endovascular procedure

The written consent was obtained from each patient before endovascular procedure. All patients received

dual antiplatelet therapy for at least 5–7 days, intravenous heparin bolus 5000 U at the beginning of the procedure and premedication with Atropine 0.5 mg shortly before balloon angioplasty and carotid stent placement. The procedure was done under local anaesthesia using standard technique described previously [11,12]. Beside ABPM measures, patient's BP, electrocardiogram and neurological status were continuously monitored during the procedure. Direct bilateral common carotid artery angiography was performed in order to confirm internal carotid artery stenosis. Placement of self-expanding Precise stent (Cordis, USA) was proceeded after balloon angioplasty (Viatrac, Abbot, USA). Angioguard (Cordis, USA) – a distal embolic protection device was used in each patient. Post dilatation was performed if needed, after stent placement using 5 mm or 6 mm balloon. Cerebral and common carotid arteriography was performed at the end of procedure to confirm proper stent patency and lack of vascular complications (i.e. periprocedural cerebral artery embolism). Technical success of the procedure was described as carotid stent placement with no residual stenosis.

Atherothrombotic risk evaluation

Based on medical records, physical examination and comprehensive history available at baseline, we evaluated atherothrombotic risk factors, including tobacco use, diabetes, hyperlipidemia, hypertension, coronary artery disease (CAD), peripheral artery disease (PAD). Hypertension was defined as systolic BP (SBP) ≥140 mm Hg and/or diastolic BP ≥90 mm Hg, or use of antihypertensive medication. Diabetes mellitus was defined as fasting serum glucose level ≥126 mg/dL, hemoglobin A1c levels ≥6.5%, or use of antidiabetic medication. Patients who were smokers at the time of analysis were classified as current smokers. All patients received optimal medical treatment, according to guidelines.

Study endpoints

The primary study endpoint was PH, secondary endpoints included in-hospital mortality, any postoperative neurologic events (stroke/TIA), MACEs (myocardial infarction, congestive heart failure, and dysrhythmias) and progression of neurological deficit during 6 months of follow-up. Neurological assessment in NIHS score (NIHSS) and modified Rankin Score (mRS) was performed at baseline and 24 hours after CAS. Functional status in mRS and occurrence of secondary endpoints were assessed during telephone assessment performed 6 month after CAS. As a routine management in our center, all patients have control carotid ultrasound performed before discharge and in case of neurological deterioration the control brain CT scan to rule out acute infarction or hemorrhage. The study has been conducted in the accordance of Declaration of Helsinki.

Statistical analysis

Statistical analysis was performed with the use of Student's t-test for continuous data, the Chi-square test and Fisher exact test for categorical data. The difference between baseline risk factors and characteristics of carotid artery stenosis, procedures, and pre- and intraoperative hemodynamics were compared between patients without and with PH. A probability value of $p < 0.05$ was considered significant. All data are presented as mean ±SD values. All analyses were performed using Statistica 10.0 software (StatSoft Inc., USA).

Results

30 consecutive patients undergoing CAS between 2013––2017 were recruited and all had complete follow-up. Median age was 69 years (range 62–78), 70% were males, 86% of patients had symptomatic internal carotid artery stenosis, all subjects were functionally independent at baseline, suffered from hypertension, almost half were smokers (Table 1). No patients had concomitant atrial fibrillation, PAD, nor previous CAS or CEA. In all 30 patients technical success was achieved at the end of carotid stenting procedure. There was no periprocedural serious adverse events. Sixty seven percent of patients (n = 20) experienced PH, 43% had transient bradycardia (n = 13), 33% had both PH and bradycardia (n = 10). There were no differences in demographic data and vascular risk factors between those subjects who experienced hypotension versus those without PH (Table 1). However, patients with PH significantly less often were treated with Ca-antagonist, more often had ipsilateral to CAS ulcerated plaque or had hemodynamically significant stenosis of contralateral ICA. The frequency of bifurcation involvement was similar among groups as well as maximal balloon pressure, stent diameter and length. Mean degree of stenosis of ipsilateral carotid artery was not different between PH and groups (70% vs 65%, p = 0.9). Intraprocedural hypotension was more common in patients with PH (90% vs 40%, p = 0.02). The cumulated postprocedural mean SBP and DBP decreased from baseline 128/67 mm Hg to 108/54 mm Hg ($p < 0.01$), mean day (69/min) and night HR (58/min) at baseline decreased to respectively 58/min and 49/min ($p < 0.01$) (Figure 1).

Of 13 subjects who experienced bradycardia, 11 (85%) had bradycardia during the day compared to none before CAS (p = 0.002) and all experienced bradycardia at night compared to 7 (54%) before the procedure (p = 0.01). Only 3 subjects (23%) had symptomatic bradycardia (2 patients had vertigo, 1 experienced syncope) and

Tabel 1. Clinical characteristics of studied groups
Tabela 1. Charakterystyka kliniczna badanej grupy

clinical characteristics	all n	%	group PH	%	group APH	%	*p* value
no. of patients	30		20	67.0	10	33	
age (mean ±SD) yr	69 ±11		67 ±10		70 ±8		NS
IHD	15	50.0	9	45.0	6	60	NS
male sex	21	70.0	16	80.0	5	50	NS
history of stroke	12	40.0	7	35.0	5	50	NS
history of TIA	9	30.0	5	25.0	4	40	NS
diabetes	7	23.3	4	20.0	3	30	NS
hyperlipidemia	21	70.0	13	65.0	8	80	NS
smoking	13	43.3	8	40.0	5	50	NS
hypertension	30	100.0	20	100.0	10	100	NS
statins	15	50.0	10	50.0	5	50	NS
antihypertensive treatment	27	90.0	20	100.0	7	70	NS
β-blockers	12	40.0	8	40.0	4	40	NS
ACE-inhibitors	21	70.0	14	70.0	7	70	NS
diuretics	10	33.3	6	30.0	4	40	NS
Ca antagonists	12	40.0	5	25.0	7	70	0.02
baseline mRS*	2 ±1		3 ±1		2 ±1		NS
baseline NIHSS*	4 ±2		4 ±1		4 ±2		NS
symptomatic ICA stenosis	26	86.7	18	90.0	8	80	NS
ulcerated plaque	22	73.3	17	85.0	5	50	0.04
bifurcation involvement	13	43.3	9	45.0	4	40	NS
contralateral stenosis >50%	15	50.0	12	60.0	3	30	0.04
maximal balloon pressure (atm)	13 ±1		13 ±1		13 ±1		NS
stent diameter (mm)	7 ±1		7 ±1		7 ±1		NS
stent lenght (mm)	30 ±10		30 ±10		30 ±10		NS
intraprocedural bradycardia	14	46.7	10	50.0	4	40	NS
intraprocedural hypotension	22	73.3	18	90.0	4	40	0.02

* in symptomatic patients

APH – absent PH, IHD – ischemic heart disease, PH – periprocedural hypotension

required transient atropine infusion, any patient had required pacemaker implementation. Majority of subjects with HI had asymptomatic PH (n = 13, 65%) or experienced mild symptoms (weakness, lightheadedness or pale skin, n = 4, 20%), 3 subjects (15%) had orthostatic hypotension which resulted in syncope with a spontaneous rapid and complete recovery. Any patient experienced other neurological deterioration during PH or later on during follow-up. The neurological condition in PH and APH groups at discharge from the hospital and after 6-months was not different from the baseline status in both NIHSS score and mRS. Any patient experienced vascular events or death during hospitalisation. Only 1 studied patient from PH group (5%) had secondary endpoint (TIA at 4 month following CAS), which was probably related to antiplatelet and statin therapy noncompliance.

Discussion

The studies performed so far, showed that HI following CAS occurrence is unpredictable, and its association with adverse events has not been well defined. Our study revealed, that postprocedural HI is a common phenomenon after CAS, however, it was not linked with

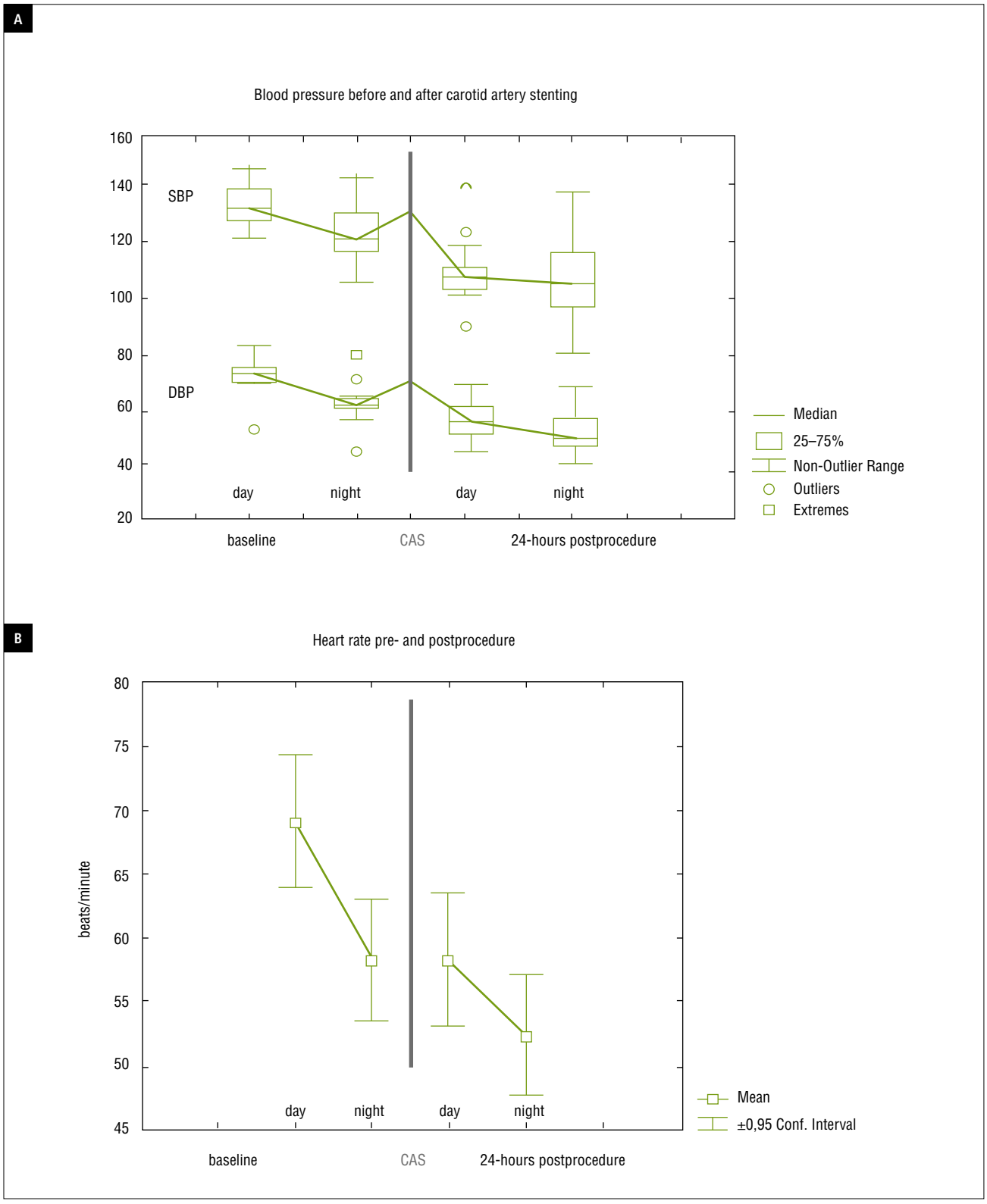

Figure 1. Comparison of baseline and postprocedural systolic (SBP) and diastolic (DBP) blood pressure (**A**) and heart rate (**B**)
Rycina 1. Porównanie podstawowego i pozabiegowego ciśnienia skurczowego (SBP) i rozkurczowego (DBP) (**A**) oraz tętna (**B**)

neurological or other vascular complications in the studied cohort in short- and long-term observation. We demonstrated that patients at risk of PH can be possibly identified through clinical and angiographic variables (no antihypertensive Ca-antagonists treatment prior to CAS, hemodynamic instability during CAS, ulcerated ipsilateral plaque, significant stenosis of contralateral carotid artery).

The most serious acute complication associated with CAS is stroke. Although the stroke rate is 3 to 4 percent and has steadily decreased with improvements in device technology and operator experience, risk factors for periprocedural stroke are not known [13]. Frequency, risk factors for other complications that are not specific to the approach to stenting, such as myocardial infarction, renal failure related to intraarterial contrast injection, carotid thrombosis and restenosis, and stent dislocation are also not known. Poorly controlled hypertension preoperatively is predictive of prolonged length of stay due to postoperative hypertension. It is therefore important to assess if aggressive preoperative antihypertensive drugs are related to risk of postprocedural HI [14]. Our data are in a line with Kojuri at al. who reported 63% of PH [15] and are similar to the published meta-analysis which showed overall HI rate of over 40%, however, pooled estimate for hypotension (12.1%) was lower than in our study (12.2% for bradycardia, 12.5% for both hypotension and bradycardia, persistent HI 19.2%) [16]. The Authors reported no statistically significant differences between patients with and without HI after CAS with respect to death, stroke, TIA, or major adverse events but there were statistically significant associations of mean age with HI, of ≤10 mm distance between the carotid bifurcation and the site of minimum lumen diameter with bradycardia, and of prior ipsilateral CEA with persistent HI. On the other hand, one recent retrospective study on nearly 25,000 patients demonstrated, that postoperative hypotension occurred in only 15% of CAS, patients with hypotension (compared to no hypotension) had higher rates of stroke, myocardial infarction, prolonged length of stay, and in-hospital mortality, risk factors associated with HI were atherosclerotic lesion, female sex, age >70, history of myocardial infarction or angina and an urgent (vs elective) procedure [17].

The higher rate of PH in our study is probably related to the use of Holter monitoring which enabled us to recognize all hypotension events occurring within 24 hours post CAS. Difference between risk of intraprocedural hypotension may result from some technical differences between operating methods and also contrast volume, type of stent or embolic protection device [18]. Furthermore, dilation degree may also increase risk of PH [19]. Low rate of vascular events in our study is surprising but it is probably related to low sample size and lower degree of baseline carotid stenosis [18]. The latter could suggest less severe cerebrovascular disease in our cohort than in other studies. Additionally higher incidence of PH could have possibly resulted in longer hospitalization and thus better adjustment of medical treatment during postoperative period. Extension of hospitalization duration due to hypotension has been frequently reported [5].

Our data demonstrated that hemodynamic instability occurred more often in the presence of ulcerated ipsilateral plaque or significant stenosis of contralateral carotid artery. Similar findings have been previously reported by Gökçal at al. [5]. The reason is unknown, but probably it is related to the abnormal baroreflex response. Baroreflex response is probably involved in the genesis of hemodynamic reactions. The baroreflex is maintained through an afferent and efferent limb of a reflex arc. It provides feedback control to blood pressure regulation centres. The highest concentration of baroreceptors which are stretch receptors is in the carotid artery bulb. When they are stretched, a signal is sent via the glossopharyngeal nerve to the nucleus tractus solitarius and this inhibits the activity of sympathetic fibers within the nucleus and concomitantly second order neurons excite synapses of the dorsal vagal nucleus. This results in increased vagal tone, increased parasympathetic activity and reduction in heart rate and blood pressure. The presence of calcified atheroma in the carotid artery chronically sensitizes the carotid baroreceptors to very small changes in the carotid artery tension. External manipulation in this area can therefore produce marked hypotensive response. The chronic administration of Ca antagonists reduced the incidence of hypotension in our cohort. This finding is surprising and to the best of our knowledge it has not been previously reported. That positive effect could be possibly explained by enhanced reflex response to sympathetic tone mediated by Ca antagonists. The limitation of the study is that since the sample is small, we might have missed the true characteristic of CAS population and our results cannot be therefore generalized. Due to low number of enrolled subjects we did not analyze risk factors for bradycardia and characteristics of carotid plaques. On the other hand our study has some strengths. We used BP Holter monitoring to assess HI, therefore we could precisely diagnose PH or bradycardia events and also our subjects were followed for 6-months post CAS.

Conclusions

Postprocedural hypotension is a common phenomenon after CAS without increasing the perioperative risk. Patients at risk can be possibly identified through clinical and angiographic variables. PH was not linked with significant neurological complications within 6 months of

observation. Protective role of Ca antagonists in the development of hemodynamic instability demonstrated in our study should be further evaluated.

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