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Predictors of Carotid Artery Stenting-Induced Hemodynamic Instability

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Abstract

Background: To address the predictors of hemodynamic instability (HI) related to carotid artery stenting (CAS) and evaluate the association between HI and periprocedural adverse outcomes. **Methods:** This study comprised all consecutive patients who underwent CAS for atherosclerotic carotid artery stenosis from March 2014 to May 2018. A standardized dose of atropine (0.4 mg) was given prior to stent deployment. Changes in heart rate, blood pressure, and neurological status were monitored and recorded. Potential predictors of HI were tested in multivariate analysis using binary logistic regression model. **Results:** A total of 728 patients were enrolled. Two hundred twenty seven patients (31.2%) developed periprocedural HI. The presence of hypertension (OR, 2.037; 95% CI, 1.292-3.211; $P = 0.0022$), symptomatic carotid lesions (OR, 1.704; 95% CI, 1.057-2.747; $P = 0.0287$), right sided lesions (OR, 3.090; 95% CI, 1.934-4.935; $P \leq 0.0001$), hyperechoic/calcified plaques (OR, 2.195; 95% CI, 1.458-3.304; p $P = 0.0002$), and longer lesions (OR, 1.043; 95% CI, 1.012-1.076; $P = 0.0072$) were significant predictable factors for the occurrence of HI. On the other hand, smoking was significantly associated with a 48.1% decrease in risk of development of HI (OR, 0.519; 95% CI, 0.358-0.754; $P = 0.0006$). There were no statistically significant differences in periprocedural morbidity or mortality between patients with and without HI. **Conclusion:** HI occurs in a considerable percentage of patients undergoing CAS. Hypertension, right sided, symptomatic carotid lesions, calcified plaques, and longer lesions were shown to be independent risk factors for the development of periprocedural HI. Conversely, smoking demonstrated a protective effect. HI did not appear to predispose to periprocedural adverse events.

Keywords

carotid stenting, hemodynamic instability, complications, atropine

Introduction

Ischemic Stroke is considered a major cause for morbidity and mortality and representing a huge economic burden because of disabilities.[1] Carotid artery stenting (CAS) is considered an alternative to carotid endarterectomy (CEA) with proven safety and efficacy in multiple trials due to its less invasive nature and simplicity compared to CEA.[2,3] According to the current guidelines, CAS may be considered as a treatment alternative in patients with high risk for CEA as in cases with age >80 years, clinically significant cardiac disease, severe pulmonary disease, contralateral internal carotid artery occlusion, recurrent stenosis after CEA, contralateral recurrent laryngeal nerve palsy, and previous radical neck surgery or radiotherapy.[4] Hemodynamic instability (HI) is considered a common complication after CAS and can be manifested by hypotension and/or bradycardia.[5] The estimated incidence of HI is varies widely in literature (7.2%-80%).[6,7] It is proposed to occur due to stimulation of carotid sinus baroreceptors (CSBs) by balloon dilation and stent deployment. Consequently, this leads to inhibition of sympathetic and stimulate parasympathetic outflow with the resultant hypotension and/or bradycardia.[8] Numerous studies have investigated the risk factors of HI.[9-11] Others have studied the possible relation between these hemodynamic changes and periprocedural complications as stroke, myocardial infarction, transient ischemic attacks, and even death.[12,13] The aim of the study is to address the predictors of HI related to CAS and evaluate the association between HI and periprocedural adverse outcomes.

Methods

Patients

This is a retrospective study of prospectively collected data comprising all consecutive patients who underwent CAS for atherosclerotic carotid artery stenosis from March 2014 to May

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2018 in the Division of Vascular and Endovascular Surgery, University of Perugia, Perugia, Italy; and Department of Vascular and Endovascular Surgery, Assiut University Hospital, Assiut, Egypt. Both asymptomatic and symptomatic patients with carotid artery stenosis were included. Indications for CAS in our study were symptomatic carotid stenosis of $\geq 50\%$ or asymptomatic carotid stenosis of $\geq 60\%$. Patients with isolated common carotid artery (CCA) lesions, previous ipsilateral CEA, carotid stenosis due to fibromuscular dysplasia, and those with acute or unstable symptoms were excluded. Patients were extracted from the prospectively collected departmental database. The Institutional Review Board of the University of Perugia, Assiut University Hospital waived the need for ethics approval or informed consent for the use of anonymized and retrospectively analyzed data.

All patients underwent duplex ultrasound (DUS) examination by a validated operator for detection of the degree of stenosis, diameters of both CCA and internal carotid artery (ICA), and specifying the plaque morphology according to Gray Weale scale.[14] All data was confirmed by Computed Tomography Angiography (CTA). The degree of carotid stenosis was determined according to North American Symptomatic Carotid Endarterectomy Trial (NASCET).[15]

Procedure

All patients scheduled for CAS received dual antiplatelet therapy consisting of aspirin (75 mg/day) and clopidogrel 300 mg as a loading dose 12 hours before the procedure. All procedures were performed by experienced vascular surgeons, under local anesthesia and conscious sedation, in a hybrid operating room equipped with either a fixed digital angiographic system (Perugia) (Axiom Artis FA, Siemens Healthineers, Erlangen, Germany), or mobile C-arm (Assiut) (BV Pulsera, Philips Medical Systems, Eindhoven, the Netherlands).

Percutaneous transfemoral access was utilized in all patients. After sheath insertion, 100 IU/kg IV bolus of heparin was administered. Afterward, angiography was performed in two different projections to confirm the diagnosis, and assess the intracerebral circulation.

Cerebral protection devices (CPDs) and self-expandable stent of different models were applied in all procedures, tailored to vessel and lesion characteristics according to operator evaluation and experience. Distal filters, passed across the carotid lesion over a wire and deployed distally in the ICA to capture any debris, were preferred in cases of concomitant contralateral occlusion as it allows antegrade cerebral flow throughout the entire procedure. On the other hand, internal clamping devices, entailing deployment of an occlusion balloon in the CCA and the ECA causing flow stasis during stent deployment with subsequent aspiration of any debris, were usually deployed in cases where placement of filter was deemed difficult or amenable to complications as in high grade stenosis and/or vessel tortuosity.

Pre-stent balloon angioplasty, using 3-4 mm balloon, was performed selectively in cases of tight lesions. Stent diameter and length were selected according to artery diameter and lesion length. The type of stent was chosen according to the symptomatic state of the patient and plaque morphology. Post-stent balloon angioplasty, using 5-6 mm balloon at a pressure of 8-10 atm for 5 seconds, was performed routinely in all patients. Finally, completion angiography was performed in different views to assess both local and central results and detect possible complications.

Following CAS procedures, vital signs and neurologic state were monitored for a minimum of 24 hours. Patients were usually discharged on the first postoperative day if no complications were encountered. Aspirin (75 mg/day) and clopidogrel (75mg/day) were continued postoperatively for at least 1 month followed by lifelong aspirin. Afterward, they were scheduled for routine follow-up visits comprising both physical and duplex ultrasound examination at our outpatient clinic after 1 month then every 6 months thereafter.

HI was defined as the occurrence of hypotension and/or bradycardia regardless the need for adjunctive atropine, fluid support or vasopressor agents. Hypotension was defined as symptomatic or asymptomatic decrease in the systolic BP <90 mm Hg, and bradycardia as decreased HR <60 beats per minute. Hypotension lasting >1 hour requiring vasopressor support was defined as persistent hypotension. Transient ischemic attacks (TIAs) were defined as any new neurological focal event with complete recovery within 24 hours, while stroke as any new neurologic deficit lasting >24 hours with associated radiographic evidence of acute intracranial abnormality. Myocardial infarction (MI) was diagnosed on the basis of cardiac enzymes elevation in combination with electrocardiographic (ECG) changes consistent with new ischemia and/or evidence of new regional wall motion abnormality on echocardiography.

Hemodynamic Protocol

According to the hemodynamic protocol, routine prophylactic atropine at a standardized dose of 0.4 mg was given intravenously to all the patients before stent deployment. If bradycardia is not improved, additional intravenous atropine (0.5-1 mg) was given. Hypotension was managed by intravenous fluids (250-500 ml of 0.9% hydrochloride). The fluid rate was adjusted according to the blood pressure and cardiac state of the patient. Vasopressors were given to patients in case of persistent hypotension after failed response to fluid infusion. Norepinephrine was administered at rate of 2-6 mcg/kg boluses and dopamine sometimes was needed. Patients with persistent HI were referred for cardiology consultation.

Continuous monitoring of the heart rate (HR), blood pressure (BP), oxygen saturation, and neurological state was done throughout the procedure every 5 minutes. Following the procedure, monitoring was continued every 15 minutes for the first 2 hours. Afterward, the follow-up was done every hour for 24 hours. The presence and duration of any periprocedural HI was recorded. Cardiac morbidity, based on cardiac enzymes and

Table 1. Demographics of the Study Cohort.

	Overall (n = 728)	HI (n = 227)	Non-HI (n = 501)	*P* value
Age, years				
Mean ± SD	71.00 ± 7.40	70.84 ± 7.56	71.07 ± 7.33	0.695
Range	51-87	51-85	53-87	
Median (IQR)	72 (12)	72 (12)	72 (12)	
Male gender	479 (65.8)	150 (66.1)	329 (65.7)	0.981
Diabetes	216 (29.7)	78 (34.4)	138 (27.5)	0.076
Hypertension	581 (79.8)	190 (83.7)	391 (78.0)	0.097
CAD	191 (26.2)	65 (28.6)	126 (25.1)	0.369
Previous MI	124 (17.0)	39 (17.2)	85 (17.0)	0.972
COPD	135 (18.5)	38 (16.7)	97 (19.4)	0.459
Current smoking	385 (52.9)	110 (48.5)	275 (54.9)	0.126
Dyslipidemia	263 (36.1)	88 (38.8)	175 (34.9)	0.360
CKD	56 (7.7)	17 (7.5)	39 (7.8)	0.991

HI: Hemodynamic Instability, CAD: Coronary Artery Disease, COPD: Chronic Obstructive Lung Disease, CKD: Chronic Kidney Disease.

ECG changes or clinical evidence of congestive heart failure (CHF), was recorded.

Patient preparation, procedure technique, hemodynamic protocol, and monitoring were the same in the 2 centers.

Study Outcome Measures

The primary outcome measure was the occurrence of any periprocedural HI including potential contributing factors. The periprocedural occurrence of TIAs, stroke, MI, and death were secondary outcomes.

Statistical Analysis

Statistical analysis was performed using SPSS 25.0 (SPSS Inc, Chicago, IL, USA), and MedCalc 16.8 (MedCalc Software, Ostend, Belgium). Continuous variables were expressed as mean ± standard deviation (SD) and/or median and interquartile range (IQR), and categorical variables as frequency and percentage. Baseline characteristics were compared between the 2 cohorts (patients with and without HI) using the Student t-test for continuous variables and chi-square test for categorical variables. Multivariate analysis using binary logistic regression model with stepwise approach was generated to assess the influence of various demographic, and lesion characteristics on hemodynamic instability, with results presented as odds ratio (OR) and 95% confidence interval (CI). A P value <0.05 was considered statistically significant.

Results

A total of 728 patients, including 479 men, with a mean age of 71.00 ± 7.40 years, underwent CAS for atherosclerotic carotid artery stenosis between March 2014 and May 2018. The demographic characteristics of the patient population are summarized in Table 1.

Five hundred seventy eight patients (79.4%) were asymptomatic. The mean degree of carotid stenosis was 77.82 ± 4.73%, with a mean plaque length of 1.76 ± 0.55 cm. Other lesion characteristics are illustrated in Table 2.

Embolic protection devices were used in all patients. FilterWire EZ system (Boston Scientific, Marlborough, MA, USA) was used in the majority of cases (n = 691, 95%), followed by Emboshield NAV (Abbott Laboratories, Chicago, Ill, USA) (n = 20, 2.7%). Other types of protection devices used were: SpiderFX (Medtronic Inc., Santa Rosa, CA, USA) (n = 7, 1%), Mo. Ma (Medtronic Inc.) (n = 7, 1%), and Angioguard RX (Cordis, Santa Clara, CA, USA) (n = 3, 0.3%).

Various types and designs of self-expandable stents were used. The most common stent used was XACT Carotid Stent System (Abbott Laboratories) (n = 495, 68%), followed by Cristallo Ideale (Invatec Inc., Brescia, Italy) (n = 76, 10.4%), and Carotid Wallstent (Boston Scientific) (n = 73, 10%). Other types of stents were: Precise Pro RX (Cordis) (n = 57, 7.8%), CGuard (InspireMD, Winsen, Germany) (n = 43, 6%), Protégé RX (Medtronic) (n = 22, 3%), RX Acculink (Abbott) (n = 2, 0.3%), and Adapt Carotid Stent (Boston Scientific) (n = 1, 01%).

The procedure was technically successful in all 728 cases. Two hundred twenty-seven patients (31.2%) developed periprocedural HI. One hundred ninety patients (26.1%) developed only hypotension, 18 patients (2.5%) developed only bradycardia, and 19 patients (2.6%) developed both. In 61 of the 227 patients (26.9%) who developed HI, adjunctive atropine (13 patients, 5.7%) or vasopressor (48 patients, 21.1%) treatment was required for management of bradycardia or persistent hypotension, respectively. There were no significant differences in patient and lesion characteristics between the two cohorts (Tables 1, 2).

A multivariate logistic regression model was constructed to determine possible predictors of HI during and after CAS. The presence of hypertension (OR, 2.037; 95% CI, 1.292-3.211; $P = 0.0022$), symptomatic carotid lesions (OR, 1.704; 95% CI, 1.057-2.747; $P = 0.0287$), right sided lesions (OR, 3.090; 95% CI, 1.934-4.935; $P \leq 0.0001$), hyperechoic/calcified plaques (OR, 2.195; 95% CI, 1.458-3.304; $P = 0.0002$), and longer lesions (OR, 1.043; 95% CI, 1.012-1.076; p $P = 0.0072$) were significant predictable factors for the occurrence of HI. On the other hand, smoking was significantly associated with a 48.1% decrease in risk of development of HI (OR, 0.519; 95% CI, 0.358-0.754; $P = 0.0006$) (Table 3).

Periprocedural complications occurred in 27 patients (3.7%). Nineteen patients (2.6%) developed periprocedural neurologic deficits. Nine patients had full resolution of neurologic deficits at the time of hospital discharge, 9 patients were discharged with residual neurologic deficits, whereas 1 patient died of massive cerebral hemorrhage. Eight patients (1.1%) experienced myocardial infarction in the periprocedural period. During the study period, 3 patients died due to cerebral hemorrhage (1 patient) and myocardial infarction (2 patients). There were no statistically significant differences in periprocedural morbidity or mortality between the 2 cohorts (Table 4).

Table 2. Lesion Characteristics.

	Overall (n = 728)	HI (n = 227)	Non-HI (n = 501)	*P* value
Side				
Rt	359 (49.3)	123 (54.2)	236 (47.1)	0.091
Lt	369 (50.7)	104 (45.8)	265 (52.9)	
Symptomatic				
TIA	124 (17.0)	46 (20.3)	78 (15.6)	0.461
Stroke	26 (3.6)	5 (2.2)	21 (4.2)	
Asymptomatic	578 (79.4)	176 (77.5)	402 (80.2)	0.461
Degree of stenosis, %				
Mean ± SD	77.82 ± 4.73	78.30 ± 6.43	77.60 ± 3.70	0.065
Range	60-90	60-90	65-85	
Median (IQR)	80 (5)	80 (5)	80 (5)	
Plaque length, cm				
Mean ± SD	1.76 ± 0.55	1.82 ± 0.38	1.74 ± 0.61	0.059
Range	0.7-2.5	1.0-2.5	0.7-2.5	
Median (IQR)	1.9 (0.8)	1.9 (0.5)	1.9 (1.0)	
Plaque echogenicity				
Type I/II	289 (39.7)	78 (34.4)	211 (42.1)	0.058
Type III/IV	439 (60.3)	149 (65.6)	290 (57.9)	
Contralateral occ.	73 (10.0)	23 (10.1)	50 (10.0)	0.944
Hostile neck	25 (3.4)	11 (4.8)	14 (2.8)	0.235

HI: Hemodynamic Instability, TIA: Transient Ischemic Attack.

Table 3. Binary Logistic Regression Model for Predictors of Hemodynamic Instability.

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Age	0.996 (0.975-1.017)	0.694		
Male gender	1.018 (0.732-1.418)	0.914		
Diabetes	1.377 (0.983-1.929)	0.063		
Hypertension	1.445 (0.958-2.179)	0.079	2.037 (1.292-3.211)	**0.0022**
CAD	1.194 (0.840-1.697)	0.323		
Previous MI	1.015 (0.670-1.540)	0.943		
COPD	0.837 (0.554-1.266)	0.399		
Current smoking	0.773 (0.564-1.058)	0.108	0.519 (0.358-0.754)	**0.0006**
Dyslipidemia	1.179 (0.853-1.631)	0.318		
CKD	0.959 (0.530-1.734)	0.890		
Rt side	1.328 (0.970-1.819)	0.077	3.090 (1.934-4.935)	**<0.0001**
Symptomatic	1.177 (0.804-1.723)	0.403	1.704 (1.057-2.747)	**0.0287**
Stenosis degree	1.032 (0.998-1.067)	0.065		
Plaque length	1.028 (0.999-1.059)	0.059	1.043 (1.012-1.076)	**0.0072**
Calcified plaque	1.390 (1.003-1.926)	**0.048**	2.195 (1.458-3.304)	**0.0002**
Contralateral occ.	1.017 (0.604-1.712)	0.950		
Hostile neck	1.772 (0.791-3.966)	0.164		

HI: Hemodynamic Instability, CAD: Coronary Artery Disease, COPD: Chronic Obstructive Lung Disease, CKD: Chronic Kidney Disease.
Note. Bold values highlight clinically significant predictors of hemodynamic instability.

There were no statistically significant differences between patient population of the 2 centers regarding demographics, lesion characteristics, and outcomes.

Discussion

Carotid artery stenosis is thought to be responsible for about 20%-30% of all ischemic strokes.[16] In recent years, CAS has attracted much attention in the treatment of extracranial carotid artery stenosis for its minimally invasive, safe, and effective advantages specially in high surgical risk group. Many clinical randomized controlled trials have confirmed that there is no significant difference between CAS and CEA regarding periprocedural major adverse cardiovascular events.[17-19] This confirms that both have the same efficacy in long-term carotid stroke prevention and that the effect of CAS is not inferior to CEA.

Table 4. Periprocedural Outcomes Following CAS.

	Overall (n = 728)	HI (n = 227)	Non-HI (n = 501)	*P* value
TIAs	9 (1.2)	4 (1.8)	5 (1.0)	0.616
Stroke	10 (1.4)	6 (2.6)	4 (0.8)	0.102
MI	8 (1.1)	3 (1.3)	5 (1.0)	0.997
Death	3 (0.4)	2 (0.9)	1 (0.2)	0.481

HI: Hemodynamic Instability, TIAs: Transient Ischemic Attacks, MI: Myocardial Infarction, MACE: Major Adverse Cardiovascular Events.

HI is a frequent periprocedural complication of CAS.[20] Stretching of CSBs, during balloon angioplasty and stent deployment, is followed by activation of nerve fibers to the nucleus tracts solitaries through the glossopharyngeal nerve. Subsequently, this allows inhibition of sympathetic outflow and enhancing the parasympathetic system with the resultant effect of decrease in the blood pressure and heart rate.[8]

HI is usually transient and self-resolving, not requiring pharmacological adjunct to the prophylactic intravenous dose of 0.4 mg atropine. Less frequently, it has a prolonged and more severe course requiring vasopressors or additional atropine.[21]

In the present study, periprocedural HI developed in 31.2% of patients undergoing CAS. The true incidence of HI associated with CAS is widely variable in literature. Mlekusch et al.[6] reported HI in 7.2% of their cohort. On the other hand, Tsurumi et al.[7] reported an incidence of 80%.

Many demographic variables have been explored as potential risk factors for the development of HI. Various studies have reported that patients with increased age are more vulnerable to CAS-induced HI.[6,10,22] Increased age is associated with decreased sensitivity of cardiovagal baroreflex, and consequently impaired response to acute changes in blood pressure.[23]

Some authors have investigated patient gender as a potential predisposing factor for HI. Gupta et al.[24] concluded that female sex was an independent predictor of HI in a study of 584 consecutive CAS patients. In contrast, Taha et al.[25] reported male sex as a risk factor of HI. This sex difference may be related to variability in quantitative carotid plaque characteristics and carotid bifurcation anatomy.[26]

Other studies have suggested that patients with a history of cardiac risk factors,[10,21,27] and SBP >180 mm Hg[28] are more susceptible for the development of HI due to increased sensitivity of carotid sinus stimulation.

In contrast, it has been reported in literature that diabetes, COPD, and smoking provide a protective effect against the development of HI during CAS. Patients with COPD have been shown to have impaired baroreflex sensitivity leading to an increase in sympathetic activity.[29] Smoking impairs the carotid baroreceptor response and augments sympathetic tone.[12,30] Diabetic patients have an attenuated cardiovagal response and overactivity of the sympathetic component, thus elevating blood pressure.[31,32]

In the current study, the presence of hypertension was significantly associated with a 2-fold (OR, 2.037; 95% CI, 1.292-3.211; $P = 0.0022$) increase in periprocedural HI. Moreover, smoking was significantly associated with a 48.1% decrease in risk of development of HI (OR, 0.519; 95% CI, 0.358-0.754; $P = 0.0006$). On the other hand, multivariate analysis failed to find any significant association between other patient characteristics, including age, sex, diabetes, COPD, smoking, cardiac risk factors, and the development of HI.

Various lesion characteristics and procedural details have also been cited as related to HI. Right sided carotid lesion was reported, by Taha et al.[25] as a predictor of HI. This may be due to asymmetrical innervations to the heart as the sinoatrial node is innervated mainly by the right vagus nerve, and the atrioventricular node by the left vagus nerve.[33]

Symptomatic carotid stenosis was concluded by many authors as a predictor of HI,[34-36] due to the combination of autonomic dysfunction and irregularities in baseline hemodynamics and endocrine function.[13]

Calcified lesions are thought to be more prone to HI because CSBs, previously exposed to dampened pressure waves due to impaired expansion of the vessel wall, receive a sudden and extraordinary stimulation by CAS. Moreover, calcified plaques usually require a more aggressive balloon inflation, triggering an amplified CSB response.[21,37]Also, plaque length was positively correlated with HI because the longer the plaque, the more the number of CSBs being triggered by balloon angioplasty and stent deployment.[37]

Other parameters that have been investigated as predisposing factors for CAS-induced HI are eccentric plaque,[25] contralateral carotid stenosis,[38] bilateral carotid stenting,[39] previous ipsilateral CEA,[12,22] stent cell design,[40] and post-stent ballooning,[41] however with contradictory results.

Our data suggest that lesion characteristics namely right sided, symptomatic carotid lesions, calcified plaques, and longer lesions were significantly associated with 3.1-fold (OR, 3.090; 95% CI, 1.934-4.935; $P \leq 0.0001$), 1.7-fold (OR, 1.704; 95% CI, 1.057-2.747; $P = 0.0287$), 2.2-fold (OR, 2.195; 95% CI, 1.458-3.304; $p = 0.0002$), and 1-fold (OR, 1.043; 95% CI, 1.012-1.076; $P = 0.0072$) increase in periprocedural HI, respectively.

Debate still exists regarding the association between HI and periprocedural major adverse events, such as stoke, MI and even death. In a series of 500 patients, Gupta et al.[12] reported a 3-fold increase in the probability not only of periprocedural stroke but also of myocardial infarction and death in patients with persistent HI. More recently, Arhuidese et al.[42] reported a significant increase in periprocedural stroke rate by four-folds among patients who experienced periprocedural HI relative to those who do not in a study included 13,698 CAS procedures. On the other hand, Mylonas et al.[43] did not confirm a statistically significant increase in TIA, stroke, or death rates among patients with HI in a meta-analysis included 27 studies with a total of 4204 patients. Our results, in accordance with these latter findings, did not report statistically significant differences in periprocedural morbidity or mortality between those with and without HI. The pathophysiologic relationship between HI and periprocedural stroke is presumed to be multifactorial.[13] Hypotension and bradycardia cause new

malperfusion or accentuate concurrent or prior thromboembolic insults.[42]

This conflict in results of various studies, concerning incidence, predisposing factors, and association between HI and periprocedural adverse events, is attributed to several factors as patient selection, and heterogeneity in defining thresholds for hypotension, bradycardia, and persistant/prolonged HI. Therefore, standardization of these variables is mandated to facilitate proper comparison of outcomes.

The proper management of CAS-induced HI consists of anticipation and awareness of the condition, strict monitoring of blood pressure and heart rate throughout the procedure and for enough time in the postoperative period, especially in high risk patients, together with prophylactic administration of atropine.[22]

Limitations of this study include its retrospective nature and potential for patient selection and treatment bias. The small number of adverse outcomes may increase the chance of type II error.

Conclusions

HI occurs in a considerable percentage of patients undergoing CAS. Hypertension, right sided, symptomatic carotid lesions, calcified plaques, and longer lesions were shown to be independent risk factors for the development of periprocedural HI. Conversely, smoking demonstrated a protective effect. HI did not appear to predispose to periprocedural adverse events. Identifying patients at high risk for HI together with strict patient monitoring throughout the procedure is crucial for early recognition and correction of these hemodynamic changes.

Declaration of Conflicting Interests

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