

The Prevalence and Factors Contributing to Hemodynamic Depression in Patients Undergoing Carotid Angioplasty and Stenting

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Abstract

Background—Hemodynamic depression, including bradycardia and hypotension, is among the most common complications of carotid angioplasty and stenting.

Methods and Material—A prospective, cross-sectional study was conducted at Shiraz University of Medical Sciences in southern Iran from 2011 to 2013. Consecutive patients undergoing carotid angioplasty and stenting were included. Demographic data, atherosclerotic risk factors, preprocedural blood pressure, the site of stenosis, the degree of stenosis, and data regarding technical factors were recorded. Hemodynamic depression was defined as a systolic blood pressure less than 90 mmHg and/or heart rate less than 50 beat/min.

Results—About 170 patients (67% male, mean age: 71±9.8, 55.9% right side, 82.9% symptomatic) were recruited. Mean degree of stenosis was 79.4% in operated side and 40.7% in nonoperated side. Predilation, postdilation, or both were conducted in 18(10.5%), 141(83%), 11(6.5%) patients respectively. Thirteen (7.6%), 41(24%), and 12(7%) of patients developed postprocedural bradycardia, hypotension or both, respectively. Two patients had a stroke after CAS and periprocedural mortality was 0%. Hemodynamic depression after CAS had a significant association with preprocedure blood pressure and the use of an open cell stent design, but not with atherosclerotic risk factors, site and/or degree of stenosis, predilation, or post-dilation. Hemodynamic depression significantly increased hospital stay too.

Conclusion—Preprocedural hydration and close-cell stents may decrease the risk of poststenting hemodynamic depression.

Keywords

angioplasty; bradycardia; carotid; hemodynamics; hypotension; ischemic; stenosis; stenting; stroke

Introduction

Stroke is one of the most common causes of mortality and morbidity in both developed and developing countries [1]. Stenosis of the carotid bulb is the most common site of large artery atherosclerotic disease in patients with ischemic stroke [2]. Carotid angioplasty and stenting (CAS) is an emerging treatment option for carotid stenosis [3]. The procedural steps, the use of pre- and/or poststent angioplasty of the stenotic lesion in particular, vary from center to center.

Baroreceptors located in carotid sinuses are a crucial part of the regulation of blood pressure and heart rate [4]. The stretching of the carotid sinus caused by angioplasty and stenting can trigger the baroreceptors to transmit signals to the brainstem, resulting in a transient decrease in sympathetic tone and an increase in parasympathetic output. Bradycardia and hypotension may result [5] that in turn may increase CAS complications such as myocardial infarction and stroke [6].

We investigate the frequency and predictors of the hemodynamic depression in patients undergoing CAS.

Methods and Materials

This is a prospective cross-sectional study conducted in Kowsar and Alzahra hospitals affiliated with Shiraz University of Medical Sciences in Iran from 2011 to 2013. These are high-volume referral centers for stroke in southern Iran.

Consecutive patients with ischemic stroke documented by a brain computed tomography (CT) or an magnetic resonance imaging (MRI) were recruited. Patients underwent noninvasive vascular and cardiac testing along with laboratory studies to find the etiology of ischemic stroke.

Patients with intracranial hemorrhage, cerebral infarcts due to cardioaortic embolic causes, lacunar stroke, vasculitis, arterial dissection, and fibromuscular dysplasia were excluded. Patients with Modified Rankin Scale (MRS) more than four after stroke were excluded from undergoing CAS. Patients who had relative contraindications of angiography were also excluded.

Major cerebrovascular risk factors were investigated for all subjects. They included current, or previous cigarette

smoking, hyperlipidemia (positive history fasting total cholesterol level >200 mg/dL, LDL >130 mg/dL, and/or fasting triglycerides level >180 mg/dL), arterial hypertension (positive history, systolic blood pressure >140 mmHg, and/or diastolic pressure >90 mmHg, out of the acute phase, treated or not), and diabetes mellitus (positive history and/or fasting plasma glucose greater than 126 mg/dL out of the acute phase).

Patients with ischemic stroke and more than 70% stenosis of the ipsilateral internal carotid artery by noninvasive imaging underwent digital subtraction angiography (DSA) as part of a preoperative evaluation for CAS. The severity of stenosis was calculated according to the North American symptomatic carotid endarterectomy (NASCET) criteria [7]. Symptomatic patients with more than 50% stenosis as documented by catheter angiography and asymptomatic patients with more than 70% were included.

Patients at high risk and standard risk of carotid endarterectomy were included.

All patients undergoing CAS received 80 mg of aspirin and 75 mg of clopidogrel for at least 14 days before the procedure or 600 mg loading dose of clopidogrel. After the procedure, all patients were treated with 75 mg of clopidogrel daily for 12 months and 80 mg of aspirin for life. During the procedure, patients received heparin 80 U/kg after successful femoral artery puncture to maintain an activated clotting time (ACT) time more than 250.

Distal embolic protection devices (EPD) were crossed the lesion and opened before predilation if possible. If not usually with the stenotic segment was <1 mm, predilation was performed before EPD insertion. Predilation balloon diameters were 2.5 to 3 mm. If the diameter of the stenotic segment was >3 mm, we deployed the EPD without predilation.

In some patients, we did not use an EPD due to distal tortuosity. A self-expanding stent was then placed across the stenotic lesion. Stent diameters were 6 and 7 mm. An open cell stent, the Protege® RX Carotid Stent (ev3 Endovascular, Inc., Plymouth, USA); a closed cell stent, the Wallstent (Boston Scientific, Natick, USA); and a

hybrid open and closed design stent, the Cristallo Ideale (Invatec Technology, Frauenfeld, Switzerland) were used in this study.

After stenting, an residual stenosis was measured by angiography. The treatment was considered successful when the residual stenosis was <50%. If the residual stenosis was >50%, a balloon catheter was advanced for postdilation. Postdilation balloon diameters were 5 to 6 mm. We tried not to perform multiple balloon dilatations neither for pre- nor for postdilations .

Hemodynamic depression was defined as hypotension and/or bradycardia. Hypotension was defined as systolic blood pressure less than 90 mmHg or more than 30% decrease in systolic blood pressure in comparison with baseline recordings. Bradycardia was defined as heart rate less than 50 beat/min.

Blood pressure and heart rate were measured from the left arm by manometer and an automated electrocardiogram monitor, 10 minutes before, during and after the procedure. Before pre- or postdilation atropine 1 mg were intravenously administered to all patients regardless to their baseline heart rate.

When the bradycardia did not recover spontaneously, additional atropine was administered. After the procedure, the patients' hemodynamics were closely monitored for 24 hours or more. For persistent hypotension, we first started a saline infusion. An intravenous infusion of dopamine (3–10 µg/kg per minute) was used to maintain the blood pressure if the saline infusion failed.

The baseline clinical data collected included age, gender, and the presence of hypertension, hyperlipidemia, diabetic mellitus, and smoking. We also recorded preoperation systolic and diastolic blood pressure, the site and degree of stenosis on the operated and nonoperated sides. Technical factors included in this study were predilation balloon length, diameter and maximal pressure, stent type and size, postdilation balloon length, diameter and maximal pressure if performed, maximal balloon diameter, and maximal balloon length.

All patients provided written informed consent. The study protocol was approved by the institutional review board (IRB) of Shiraz University of Medical Sciences. (89-01-01-2614)

Statistical analyses were performed using the Statistical Package for the Social Sciences version 16.0 (SPSS Inc., Chicago, IL, USA). Continuous data are presented as the mean±standard deviation (SD), and categorical data are

presented as the count (percentage). The normality of distribution of continuous variables was assessed by the Shapiro–Wilk test, and then, the independent samples Student's *t*-test and Mann–Whitney U-test were used to compare the mean scores of variables with normal or abnormal distribution respectively. The chi-squared test and Fisher's exact test were performed to compare the proportions of categorical data when appropriate. Subsequently, multivariate analysis using binary logistic regression was performed on variables that were significant ($p < 0.05$ or very close to it) in univariate analysis.

Results

One hundred and seventy patients were recruited. Of these, 114 patients (67%) were male. The mean±SD of age was 71±9.8 [Min = 43, Max = 93, 95%CI: (69.5–72.5)]. 74.1%, 71.2%, 32.9%, and 11.8% of patients had hypertension, hyperlipidemia, diabetic mellitus, and smoking, respectively. All patients were treated electively. CAS was performed under conscious sedation in all patients.

The site of carotid stenosis was 95(55.9%) in right side. Mean degree of stenosis were 79.4±14.5 % on the operated side and 40.7±35.1 on the nonoperated side. Mean preoperation systolic and diastolic blood pressure were 126±20 [95%CI: (121.6–130.3)] and 76.7±10.9 [95%CI: (74.3–79)], respectively. A total of 141 patients (82.9%) were symptomatic.

Predilation, postdilation and both were conducted in 18(10.5%), 141(83%), and 11(6.5%) patients. Mean length and diameter of balloons for predilation were 14.7±1.9 mm and 2.7±1 mm, respectively.

Mean length and diameter of stents were 39.3±2.5 mm and 6.9±0.1 mm, respectively. For tapered stents, the diameter of proximal end was considered.

Mean length, diameter of balloons for postdilation were 20.1±1.9 mm and 5.1±0.3 mm, respectively. Deployed Stents were the Crystallo in 61(35.9%) patients, Wall-stent in 97(57.1%) patients and Protégé in 12(7%) patients.

Of 170 patients in our study, 13(7.6%), 41(24%), 12(7%) patients developed postprocedural bradycardia, hypotension or both. Postprocedural hypotension was transient and treated with hydration in eight patients (19.5%) and dopamine administration in 33 patients (80.5%). Four patients developed cardiac asystole after balloon dilation which reversed with an additional dose of atropine. Electrical shock was not indicated for any of

Table 1. Risk factors and outcomes of patients who underwent carotid angioplasty and stenting with and without hemodynamic depression.

Variables	All patients (n = 170)	Patients who developed hemodynamic depression after stenting (n = 41)	Patients who did not develop hemodynamic depression after stenting (n = 129)	P-value
Age (mean ± SD)	71±9.8	71.1±10.7	71±9.5	0.7
Male gender (%)	114(67%)	29(71%)	85(66%)	0.5
Diabetes mellitus (%)	56(33%)	15(37%)	41(32%)	0.5
Hypertension (%)	126(74%)	26(63%)	100(78%)	0.07
Hyperlipidemia (%)	121(71%)	31(76%)	90(70%)	0.4
Smoking (%)	20(12%)	6(15%)	14(11%)	0.5
Site of stenting:	95(56%)	27(66%)	68(53%)	0.3
Right side (%)	75(44%)	14(34%)	61(47%)	
Left Side (%)				
Stenosis in operated side (mean ± SD)	79.4±14.5	82.6±12.7	78.4±14.8	0.1
Stenosis in nonoperated side (mean ± SD)	40.7±35.1	38.8±36.3	41.3±35	0.5
Residual stenosis (mean ± SD)	11.3±13	12.6±12.5	10.8±13.3	0.1
Preprocedure SBP(mean ± SD)/DBP (mean ± SD)	126±20/76.7±10.9	112.2±19.4/67±10.4	131.1±17.7/80.3±8.6	0.001/0.001
Stent Type	97(57%)	21(51%)	76(59%)	0.002
Closed cell (%)	12(7%)	8(19.5%)	4(3%)	
Open cell (%)	61(36%)	12(29%)	49(38%)	
Mixed type (%)				
Predilatation (%)	29(17%)	8(19.5%)	21(16%)	0.6
Postdilatation (%)	152(89%)	39(95%)	113(88%)	0.1
Stent length(mean + SD)	39.3±2.5mm	38.9±3.1mm	39.5±2.3mm	0.2
Stent diameter (mean ± SD)	6.9±0.1 mm	7±0.1mm	7±0.1mm	0.3
Myocardial infarction (%)	0(0%)	0(0%)	0(0%)	--
Stroke (%)	2(1%)	0(0%)	2(1.5%)	1.00
Death (%)	0(0%)	0(0%)	0(0%)	--
Duration of admission in days (mean ± SD)	2±0.3	2.1±0.5	2±0.2	0.04

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure.

* Significant *P-values* are bolded and demonstrate statistical difference between patients who develop hypotension after stenting and patients who did not.

these patients. Two patients had a stroke after CAS. There was no periprocedural mortality. Table 1 demonstrates risk factors and outcomes in patients who developed hemodynamic instability in comparison with the patients who did not.

The binary logistic regression was performed in the next step. The model contained five independent variables (hypertension, preoperative systolic and diastolic blood pressures, stent type and duration of admission). The full model containing all predictors was significant, $\chi^2(6) = 48.03$, $p < 0.001$, indicating that the model was able to distinguish between the patients who developed hemodynamic depression after stenting and who did not. The model as a whole explained between 37.6% (Cox and Snell *R* square) and 53.9% (Nagelkerke *R* square). The model revealed that, of five mentioned variables, preoperative diastolic blood pressure ($p < 0.001$, OR = 0.86), duration of admission ($p = 0.001$, OR = 14.5), and hypertension ($p = 0.04$, OR = 0.26) made a significant contribution to the model.

Discussion

The current study shows that hemodynamic depression after CAS was significantly associated with preprocedure low blood and open cell stent design. Hemodynamic depression significantly increased hospital length

of stay. There was numerically more hemodynamic depression associated with postdilatation and greater length and diameters of postdilatation balloons; however, this association did not reach statistical significance.

The frequencies of post-CAS bradycardia and hypotension in the current series were 7.6% and 24%, respectively. These frequencies ranged between 5%–76% and 14%–28%, respectively in previous series [8–13].

In a meta-analysis of 4,204 patients who underwent CAS by Mylonas et al [5], hypotension, bradycardia or both were seen in 39.4%, 12.1%, and 12.2% of patients, respectively. The pooled estimate for persistent hemodynamic depression was 19.2% [5]. Our rate of HD is comparatively low. Preemptive administration of atropine before ballooning may contribute in this.

In the current study, HD was not associated with increased risk of MI and stroke but was associated with increased hospital stay. HD may have negative effects on CAS outcome. HD is associated with a greater risk of postprocedural MI and mortality rate [14] in some previous studies, but these results were not reproduced in the meta-analysis by Mylonas [5]. Meanwhile, it has been shown that patients with HD after CAS have significantly more DWI lesions than those without[15].

However, our study confirms that HD increases hospital stay as has been seen in previous studies.

In the current study, 79.4% of patients underwent balloon dilatation after stenting. Postdilatation was not significantly associated with increased risk of hemodynamic instability.

Some endovascular surgeons prefer to perform carotid stenting without postdilatation [16,17]. Qazi et al found postdilatation was associated with hemodynamic depression [18]. Others advocate for postdilatation, arguing that it increases flow to brain, decreasing hemodynamic strokes and in addition provides a smoother surface and reduces the risk of artery to artery embolization. In a series by Baldi et al [16], 5.4% of the patients needed another procedure due to lack of stent expansion [16].

In previous studies, age [5,19] degree of stenosis [8,20], plaque calcification [8], plaque diameter [20], and increased balloon pressure [8], history of coronary artery disease [19], and hypertension before CAS were associated with HD [21].

We did not see protective effects of DM and smoking for HD which has been seen in some previous studies. This protective effect is thought to result from the impairment of baroreceptors in diabetic autonomic neuropathy and increased sympathetic tone by long-term smoking [9].

As dopamine and fluid therapy was immediately started for nearly all patients who develop HD, we did not encounter any MI or stroke in hemodynamically unstable patients. The efficacy of dopamine has been previously shown in similar patients [22].

The current study focused on technical and procedural variables and their association with HD. We did not investigate the significance of plaque characteristics [23] and pretreatment drugs [14] that have been studied before. This represents one study limitation. As all patients were treated under conscious sedation, a comparison of conscious sedation versus general anesthesia was impossible in current study. The low rate of death and disability limits binary logistic regression and increases the chance of type-II error.

In conclusion, we studied the frequency of HD in patients undergoing carotid angioplasty and stenting in a high-volume single-center recruiting both high risk and standard risk. Although we routinely perform postdilatation, our rate of HD was not higher than previous studies. As HD was associated with procedural hypotension and high radial force open-cell stents, Preemptive hydration and closed-cell stents are advocated.

Conflict of Interest

The authors have no conflict of interest.

Authors Contribution

A. Borhani Haghighi provided his contribution to the concept, design, and drafting of the article, revision of the article, and also approval of the article. Data collection and drafting of the article are done by S. Kokabi and S. Yousefi. M. Emami performed data analysis and interpretation. Data collection was also performed by A. Shariat, A. Nikseresht, N. Ashjazadeh, S. Izadi, P. Petramfar, M. Poursadegh, A. Rahimi Jaber, S. Emami, H. Agheli, R. Nemati, E. Yaghoubi, M.H Abdi, M. Panahandeh, S. M. Heidari- Khormizi. Cruz-Flores and R. Edgell carried out critical revision and approval of the article.

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