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Hemodynamic Instability after Elective Carotid Stenting: Frequency and Risk Factors

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ABSTRACT

The objective of this study is to investigate the frequency and risk factors for hemodynamic instability (hypotension and/or bradycardia) in response to elective carotid stenting and their association with neurological complications. Carotid artery stenting implies instrumentation of the carotid bulb where the baroreceptors are placed and therefore baroceptor's dysfunction may provoke hemodynamic instability. The study started in the Neurology Clinic of the Emergency University Hospital, Bucharest as a retrospective analysis of the charts of 130 patients (110 men with mean age of 55 years) who underwent elective carotid artery stenting with cerebral protection for high-grade (>70%) symptomatic internal carotid artery stenosis. By hemodynamic instability one can understand the drop in systolic blood pressure of more than 30mmHG and bradycardia. 20% of patients had a drop in systolic blood pressure beyond 30 mmHg and/or bradycardia despite an adequate fluid balance. We did't use atropine as premedication before carotid stenting. There was no need for aggressive resuscitation (dopamine) and none of the patients with bradycardia needed pacemaker support. Neurological complications (transient ischemic attack, minor stroke, major stroke) occurred in 9% of patients and were not significantly associated with hemodynamic instability. Extensive carotid artery manipulation, a long stenosis situated at the bifurcation and the prior use of beta-blockers were associated with an increased adjusted risk for hypotension or bradycardia. Hemodynamic instability due to hypotension and bradycardia in response to carotid artery stenting occurs in a relatively low proportion of patients. Patients who had a long (over 6 mm) stenosis situated near the common carotid artery's bifurcation and therefore underwent extensive carotid manipulation or those who were on beta-blockers were at high risk for hypotension or bradycardia. The preadministrations of intravenous fluids didn't prevent the periprocedural hypotensive response.

Keywords: hypotension, carotid stenosis, bradycardia, stroke

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INTRODUCTION

Carotid artery baroreceptors modulate blood pressure by reciprocal changes in vagus nerve and sympathetic neural activity (1). When the arterial pressure is increased, the sensors are stretched and signals are transmitted into the central nervous system. Feedback signals are sent back to reduce arterial pressure downward toward the normal level. This is a process of negative feedback. This nervous control of the arterial pressure is by far the most rapid of all our mechanisms for pressure control. Afferent nerve fibers lead nervous impulses from the carotid sinus by way of the glossopharyngeal nerve to the brain, innervating the baroreceptors in the wall of the carotid sinus and the chemoreceptors in the carotid body. The impulses arrive at the brain via the nerve of Hering through the Nucleus of Tractus Solitarus witch modulates the neural activity of sympathetic and parasympathetic impulses (1).

Carotid artery stenosis represents a major risk factor for stroke (2). Percutaneous transluminal angioplasty and endovascular stent placement are potential treatments for high-grade and symptomatic stenosis. Complications of these procedures include stroke from distal cerebral embolism or conductance vessel occlusion, possibly in conjunction with systemic hemodynamic compromise (3,4). Hemodynamic complications that occur after carotid artery stenting probably are mediated through dysfunction of adventitial baroreceptors.

During the percutaneous artery stenting of carotid artery stenosis the radiologist places a small guide catheter through the stenosis to allow the placement of the stent. Then it inserts an autoexpandable stent which covers the stenosis and re-establishes the blood flow. Sometimes, during or after angioplasty, because of the anatomical and morphological characteristics of the stenosis, the radiologist uses a balloon to expand the blood vessel. This extensive manipulation of the carotid sinus and carotid walls leads to activation of the baroreceptors and hypotension and/or bradycardia. Cerebral protection devices were used; microporous filters were used in all the cases and included EPI FilterWire. Before the procedure, each patient received Clopidogrel prior to the initiation of the CAS procedure and was maintained on Clopidogrel for at least nine months.

The objective of this study was to investigate the frequency and risk factors for hemodynamic instability in response to elective carotid stenting and their association with neurological complications. ❑

MATERIALS AND METHODS

We reviewed the charts of patients who underwent elective carotid artery stenting with cerebral protection for symptomatic high grade (>70%) internal carotid stenosis between 01.01.2008 till 01.06.2010. From the medical charts we collected the following information for each patient: age, sex, coronary artery disease, hypertension, hyperlipemia, diabetus mellitius, cardiac arrhythmia, cardiac failure, chronic kidney disease and medication used before admission. The following procedural data were collected: the length of the maximal stenosis and the grade of stenosis. We didn't have records of the largest balloon used or the dimensions of the stent. We defined hemodynamic instability as a drop in systolic blood pressure of more than 30 mmHg, hypotension as a systolic blood pressure under 90 mmHg, and bradycardia as a heart rate under 60 beats per minute. Blood pressure and heart rate measurement noted before and after the procedure were obtained; we didn't have any data regarding these variables during procedures. The charts included the protocol for angioplasty in which there were noted the dimensions of the stenosis, length, situation on the medial or lateral wall, whether the stent covered the carotid bulb, bifurcation and internal carotid artery. All of the stenoses were evaluated according to the NASCET method for evaluation of the degree of stenosis (6). We also noted if there was any neurologic incident: minor stroke, major stroke or transient ischemic attack and if there was any premedication before the procedure: subcutaneous atropine, prehydration with isotonic saline. The effects of 10 variables from the patients' charts were eva-

Variable	Odds Ratio	95% CI
BetaBlockers	6.1136	1.0771 to 34.7008
Lenght of stenosis	10.4421	1.7900 to 60.9140
Stenosis over 90%	1.3552	0.1852 to 9.9180
Stenosis between 70-90%	0.0175	0.0024 to 0.1282
Stroke/TIA	0.0945	0.0038 to 2.3438
Prehydration	6.3512	0.6711 to 60.1097

TABLE 1. Odds Ratio and 95% Confidence Intervals - Logistical Reggresion

luated for the following outcomes: post-procedural hypotension and/or bradycardia. These variables were analyzed using the MedCalc software. Stepwise logistic regression was used to analyze these relationships with an entry criterion of p 0.1. A p value of 0.05 was considered significant. ❑

RESULTS

130 patients underwent percutaneous carotid angioplasty with Filterwire protection for symptomatic stenosis over 70%. The mean age of the patients was 55 years, 110 patients were men. No sex or age preponderance was observed for developing periprocedural hemodynamic instability. Periprocedural hypotension and or bradycardia appeared in 26 patients - 20%, none of these 26 patients required aggressive intervention (administration of atropine or inotropic support). 15 patients developed hypotension and bradycardia, 8 patients developed hypotension and two patients developed bradycardia alone. This phenomenon was transitory and resolved itself after maximum 24 hours. None oh the patients received atropine subcutaneously before the procedure. Before the procedure 100 patients received prehydration using isotonic saline, from the total of 26 patients who had hypotension 20 patients received fluids and from the rest of 104 patients who didn't developed hypotension 80 patients received IV fluids. The preadministration of IV fluids to fill the vascular bed didn't prevent the apparition of hypotension (OR=0.28, 95% CI=0.52-1.91, p=1). From the total of 130 patients eleven suffered a transient ischemic attack - 6 patients, minor stroke - 2 patients or major stroke - 2 patients. Only one patient from the group who had hypotension and/or bradycardia suffered a transient ischemic attack witch was monocular blindness on the site ipsilateral with the stenosis. The apparition of neurologic incidents didn't associate with hemodynamic instability (Table 2) (OR=0.4, 95% CI=0.04 -3.2 p=0.3). The patients underwent angioplasty for symptomatic stenosis over 70%, 37% had a stenosis over 90% (48 patients). 16 patients with stenosis over 90% developed hemodynamic instability and 32 patients with stenosis over 90% had hypotension and/or bradycardia. We observed that a stenosis over 90% was associated with an increased risk for having hemodynamic instability (Table 2) (OR=2.95% CI=0.95-4.18; p=0.0657). We also noticed that 47 patients had a length of the maximum stenosis over 6 mm (61%). 15 patients with a maximum stenosis over 6 mm had hemodynamic instability and 32 patients didn't. A length of the maximum stenosis over 6 mm was associated with an increased risk for having hemodynamic instability (Table 2) (OR=1.87, 95% CI=0.88-3.96; p=0.099). The charts were analyzed to see if the medication received by the patient influenced the development of hemodynamic instability. We analyzed the use of ACE inhibitors, diuretics, beta-blockers and we found that only the use of beta-blockers was associated with an increased risk of hemodynamic instability (OR=2.85, 95% CI=1.45-5.58; p=0.0021). To see if the associations we observed had a statistical significance we used logistical regression and found that a stenosis over 90%, maximum length of stenosis over 6 mm, and the use of beta-blockers were independent factors associated with the appearance of hemodynamic instability after CAS (Table 1). The hemodynamic instability that occurs both during and after CAS is mediated through the baroreceptors, which are located in the adventitia at the carotid bifurcation. ❑

	OR	95% CI	p
Stenosis over 90%	2	0.956-4.18	0.0657
Use of betablockers	2.85	1.45-5.58	0.0021
Stenosis over 6 mm	1.87	0.88-3.96	0.099
Neurologic incident	0.4	0.049-3.2668	0.3925
Prehidration	0.28	0.52-1.91	1

TABLE 2. Statistical analysis of the factors involved in hemodynamic instability

DISCUSSION

The baroreceptors are stimulated by the stretch of balloon angioplasty, initiating a reflex arch to the nucleus tractus solitarius via the sinus nerve of Hering, a branch of the glossopharyngeal nerve. This in turn causes a reciprocal change in peripheral sympathetic and vagal neural activity, resulting in a fall in blood pressure and bradycardia. Hemodynamic instability represented by hypotension and/or bradycardia appeared in a relative small proportion of our patients and manifested for maximum 24 hours after the procedure. None of the patients required aggressive resuscitation with atropine or inotropic support and none needed a pace-maker. In our study the hemodynamic instability didn't associate with neuro-

logic events after carotid artery stenting. Major predictors for hemodynamic instability were a stenosis over 90%, a maximal length of stenosis over 6 mm and the prior use of beta-blockers. The characteristics of the stenosis were the most important predictors of hypotension and/or bradycardia, a long maximal stenosis and a stenosis over 90% required extensive manipulation of the artery witch increases the chance for changes in neural sympathetic and vagal activity. We also found that pre administration of IV isotonic fluids to fill the vascular bed didn't influence the development of hemodynamic instability.

The limitations of this study comes from the relatively small patient sample, the insufficient data collected from the patients' charts, the fact that we didn't have any data regarding blood pressure and heart rhythm during the procedure. Also there was no data to compare if stopping or continuing the administration of beta-blockers influences the apparition of hemodynamic instability. In addition, since the majority of patients presented with significant co-morbidities, it may make distinguishing these at-risk factors more difficult. We didn't have data to confirm that discontinuation of the antihypertensive medication could prevent hemodynamic instability. This study reemphasizes the importance of close hemodynamic monitoring during and after CAS and is consistent with the previous studies witch showed that the instability didn't translate into major neurologic complications (5-7). ❑

CONCLUSIONS

Hemodynamic instability due to hypotension and bradycardia in response to carotid artery stenting occurs in a relatively low proportion of patients. Patients who had a long (over 6 mm) stenosis situated near the common carotid artery's bifurcation and therefore underwent extensive carotid manipulation or those who were on beta-blockers were at high risk for hypotension or bradycardia. Preadministration of intravenous fluids didn't prevent the periprocedural hypotensive response.

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