folia medica

Folia Medica 65(6):902-908
DOI: 10.3897/folmed.65.e100100

Original Article

The Role of Periprocedural Hemodynamic Variables during Carotid Stenting for the Mid-Term General Mortality in Advanced Age Patients

Teodora Yaneva-Sirakova[1], Galina Zlatancheva[1], Kiril Karamfiloff [2], Latchezar Traykov[3], Ivo Petrov[1], Dobrin Vassilev[4]

[1] *Acibadem City Clinic Cardiovascular Center, Sofia University, Bulgaria*
[2] *Alexandrovska University Hospital, Sofia, Bulgaria*
[3] *Medical University of Sofia, Sofia, Bulgaria*
[4] *Medica Cor University Hospital, University of Ruse, Ruse, Bulgaria*

Corresponding author: Teodora Yaneva-Sirakova, Acibadem City Clinic Cardiovascular Center, Sofia University, Sofia, Bulgaria; Email: teodora.yaneva@gmail.com

Received: 14 Jan 2023 ♦ **Accepted:** 15 May 2023 ♦ **Published:** 31 Dec 2023

Citation: Yaneva-Sirakova T, Zlatancheva G, Karamfiloff K, Traykov L, Petrov I, Vassilev D. The role of periprocedural hemodynamic variables during carotid stenting for the mid-term general mortality in advanced age patients. Folia Med (Plovdiv) 2023;65(6):902-908. doi: 10.3897/folmed.65.e100100.

Abstract

Introduction: Carotid stenting may produce significant bradycardia and/or hypotension. This may have negative short- and long-term effects for the elderly high-risk patients. Their cerebral hemodynamics is with exhausted adaptive capacity because of the multiple cardiovascular risk factors, advanced age, and significant stenosis.

Aim: This was a retrospective study aimed at finding whether periprocedural hypoperfusion or hypotension at the time of carotid stenting had any significance for the acute neurological outcome and mid-term general mortality in advanced-age patients who were at high risk for surgical endarterectomy.

Materials and methods: We studied 138 consecutive patients with significant carotid stenosis from January 2015 to July 2019. The mean (SD) age was 67.41 (10.70) years. The mean follow-up period was 31 months (922 days). The patients were hemodynamically monitored periprocedurally according to a local protocol. Vasopressors were added if a prolonged hypotension was measured. Statistical data were analyzed using SPSS IBM v. 19 (p=0.05, CI 95%).

Results: The male patients were 94 (68%). The number of patients with hypotension periprocedurally or in the first 6 hours postprocedure was 55 (42%). The mean blood pressures were 135/83 mmHg before, 116/76 mmHg during, and 121/73 mmHg after the procedure. Kaplan-Maier analysis showed no significant differences in the mid-term general mortality rate between patients with and patients without transitory hypotension. There wasn't any difference in the postprocedural neurological outcome either.

Conclusions: The presence of hypotension during carotid stenting was not linked to a negative neurological outcome. It also did not increase mid-term all-cause mortality in elderly patients (mean age, 67 years). The finding could be attributed to the relatively brief period of hypotension, the prompt administration of vasopressors, or the prevention of brain edema and hyper-reperfusion during carotid stenting in terms of elevated blood pressure; however, this remains to be determined.

Keywords

neurological outcome, systolic blood pressure drops

INTRODUCTION

Carotid artery stenting is associated with mechanical stretching at the time of balloon dilation and the respective stimulation of the carotid parasympathetic receptors, which leads to reflex bradycardia and hypotension.[1] This reaction is normal in young and healthy individuals[2], but it can be exaggerated and prolonged in advanced-age patients with multiple cardiovascular risk factors and clinically manifested cerebrovascular disease (CVD) and may have negative neurological effects. The patients indicated for carotid stenting have an elevated procedural mortality risk for carotid endarterectomy and high cardio-vascular risk.[3] Prolonged hypoperfusion due to hypotension and/or bradycardia can lead to compromise in the already stressed collateral cerebral circulation, which may compromise cerebral blood flow.[4] However, the significance of this reaction for the acute neurological outcomes and mortality rate after carotid stenting is not clear.[1,5] Risk factors for periprocedural hypotension are: the distance from carotid bifurcation and the use of proximal protective devices[6], age, the site with minimal lumen diameter[7], female sex[8], procedural aspects as dilation[1] and dilation percentage[9], use of balloon dilation compared to stenting alone[10]. Several researchers pointed out that periprocedural hypotension was associated with a significant risk for stroke, myocardial infarction, death, or length of stay.[11] The standard protocol in our clinic was invasive treatment of any significant coronary artery disease before the carotid stenting. This timing of the two procedures reduced the risk of periprocedural myocardial infarction at the time of carotid stenting with prolonged hypotension.[12] Permissive hypotension during carotid stenting, on the other hand, may be beneficial in preventing cerebral hyper-reperfusion and brain edema.

AIM

The aim of this retrospective study was to determine if periprocedural – intraprocedural, and postprocedural hypotension had a significant effect on the acute neurological outcome and mid-term mortality in patients with carotid artery stenting.

MATERIALS AND METHODS

All patients had their medical histories taken and physical status documented. Basic laboratory set for full blood count, coagulation status, electrolytes, renal and liver enzymes, CPK, MB, and high-sensitive troponin (hsTn). Glomerular filtration rate (GFR) was evaluated by MDRD and with the Cockcroft-Gault equation. The instrumental methods included ECG, echocardiography, and ultrasound assessment of the extracranial vessels.

Ethics

The procedure and follow-up were in accordance with the local ethical norms of Alexandrovska University Hospital. All patients had signed informed consent before the procedure of extracranial angiography and carotid stenting. The local ethics protocol for data management of hospitalized patients at Aleksandrovska University Hospital was followed, as were the recommendations of the Declaration of Helsinki from 1975. We did not require special ethical approval because the study was retrospective and observational in design, with no intervention on the subjects. All patients signed the Alexandrovska University Hospital's standard informed consent forms for hospitalization, data management in accordance with the GDPR, carotid stenting, blood sampling, and medical treatment (all of which absolutely necessary for the treatment of the patients).

All patients were either on dual antiplatelet therapy already or were loaded before the procedure. The standard local protocol included pretreatment of significant coronary artery disease, neurological assessment before and after carotid stenting, volume loading during the procedure, reduction of hypotensive and bradycardic medications before the procedure, and consequent step-up titration guided by the blood pressure.

Angiographic analysis

Quantitative angiographic analyses were performed using freely available software (Dicom Works version 3.1.5b, Paris, http://www.dicomworks.com/). Catheter calibration was used in all cases. The significance of the carotid stenoses was assessed in accordance with the current guidelines[3] and the resultant flow after stenting was assessed with the TICI flow classification.[13,14] Femoral access was used in most patients. Intravenous unfractionated heparin was used as an initial bolus of 5000 U and afterwards the dose was to keep activated clotting time (ACT) around 300 seconds. The precise procedural technique was implemented strictly according to the guidelines.[15] Self-expandable stents were used for the procedures.[15,16] Distal protection devices were used depending on the tortuosity of the vessel, place of the plaque, and percentage stenosis.[17] Balloon dilation was used by recommendations and personal experience of the operator.[18,19]

Hypotension was defined as a drop of systolic blood pressure below 90 mmHg or a drop of more than 30 mmHg in the mean arterial pressure, or such a drop of blood pressure (irrespective of values) that produces symptoms.[20]

Blood pressure values before the procedure were brachial. Intraprocedural blood pressure was invasively measured central aortic blood pressure. We had the intraprocedural hemodynamic curves of one-third of the stented patients available for the study. Blood pressure after the procedure and during the first 24 hours was brachially measured. Due to the inherent difference between central and brachial measurements[21,22], a significant hypotension was accept-

Folia Medica

ed only if the drop of blood pressure was registered by one and the same means of evaluation.

Statistical analysis

Mid-term mortality was defined as the mortality before the third year after stenting.

SPSS 19 was used for the statistical analysis. The qualitative data was presented with descriptive statistics, the number of cases, and a valid percent. Quantitative data was given as a mean and standard deviation value. All-cause mortality rate was assessed in the studied group. Because of crossing of the curves during the initial Kaplan-Mayer evaluation of hypotensive and non-hypotensive patients, the method was not good to study this group of patients. We used Cox regression model to find the significant predictors for mid-term survival. Correlation analysis was used to find the direction and strength of any correlation between the precise periprocedural blood pressure values and the mid-term mortality.

RESULTS

General characteristics of the patients with carotid stenting

The mean (SD) age of the studied group was 67.41 (10.70) years. The males were 94 (68%); the mean left (SD) ventricular ejection fraction was 58.84 (9.25%). Blood pressure values before the procedure were: 135/83 mmHg, mean (SD) 101 (15.34) mmHg, pulse pressure was 51.66 (16.17) mmHg, the mean double product (systolic blood pressure × heart rate) was 9606. The mean values of intraprocedural blood pressure were 115/56 mmHg, mean (SD) 91.05 (23.62) mmHg, pulse pressure mean (SD) 40 (17.12) mmHg. Postprocedural blood pressure: 121/73 mmHg, mean (SD) 89 (19.22) mmHg, pulse pressure mean (SD) 48 (15.96) mmHg.

Thirty-six patients (26%) had bilateral lesions. Total occlusion of the contralateral of the intervened carotid artery was found in 12 (8.7%) patients.

Most of the patients had arterial hypertension 132 (96%). Twenty-four patient (17%) were without a stable angina, and the majority were with second class according to the Canadian Classification – 79 (57%). Thirty-four patients (25%) had atrial fibrillation; the smokers were 62 (73% valid percent); dyslipidemia was found in 128 (94%) patients; 39 (28%) had diabetes mellitus; history of cancer was given by 17 (12%) patients; peripheral artery disease was found in 34 (25%) patients; a history of transitory ischemic attack was reported by 65 (47%), and previous ischemic stroke – by 72 (52%); only two patients (1%) reported having had a previous hemorrhagic stroke.

Periprocedural hypotension (as defined in the **Materials and methods** section) was found in 55 (41%) patients and postprocedural hypotension – in 42 (32%) patients. All patients received volume expansion with sodium chloride intraprocedurally.

Distal protection devices were used in 112 (91%) depending on the tortuosity of the vessel, the location of the plaque, and percentage stenosis.

Intravenous atropine was received by 8 (6%) patients, noradrenaline infusion – 15 (11%), dopamine infusion – 29 (22%), and dobutamine infusion – 28 (14%) patients. The main indication was hypotension and/or bradycardia below 40 beats per minute.

Complications, associated with the intervention

Complications associated with the intervention occurred in 11 (8%) patients: one patient (0.7%) had extravasation, three patients (2.2%) – intracranial hemorrhages, three patients (2.2%) had ischemic strokes, two (1.2%) patients had transitory ischemic attacks, and two patients (1.4%) had complications at the puncture site. We used the chi-square test to find that there was no correlation between periprocedural hypotension and periprocedural complications.

Acute neurological outcomes after carotid stenting

There was no correlation between periprocedural hypotension and acute neurological outcomes defined as stroke, TIA, or hemorrhagic stroke.

Concerning the medication of the patients: 119 (86%) were on clopidogrel; 17 (12%) were on ticagrelor; aspirin was given to 134 (97%) patients. At the time of the procedure, all antihypertensive predications were withheld and reintroduced into the therapy with titration in the first 72 hours with close monitoring of hemodynamics. Beta-blockers were administered to 85 (62%) patients, ACE inhibitors were given to 52 (38%) patients, ARB – to 35 (25%), calcium channel blockers were taken by 39 (28%) patients, statins – by 129 (94%) patients, aldosterone antagonists – by 13 (9%), anticoagulation – 11 (8%) with acenocoumarin and 11 (8%) with new oral anticoagulants.

Comparison of the characteristics of the dead and alive patients

We used independent sample t-test with null hypothesis that the two population (dead/alive) means were equal. Levene's test for equality of variances tested the homogeneity of variance in the studied groups. We considered as significant only the results with confidence interval of the difference which does not contain 0.

Table 1 shows only the significant differences for which the $p<\alpha$ ($\alpha=0.05$) and for which we could reject the null hypothesis for equality between dead and alive. There were no significant differences in the frequencies of smoking, CCS functional class, hypertension, atrial fibrillation, dyslipid-

Table 1. Differences between dead and alive patients after carotid stenting

Characteristics	Dead	Alive	*p*	CI
Age	71.64±8.69	66.61±10.89	0.043	−9.9, −0.16
NYHA class	95%	22%	0.02	−1.35, −0.125
Previous MI	32%	9%	0.037	−0.379, −0.085
Renal function GFR (Cockcroft and Gault)	57.63±25.7	77.74±30.63	0.006	−1.04, −0.14
LVEF	49.62±12.57	60.54±7.4	0.01	5.07, 16.78

emia, diabetes mellitus, peripheral artery disease frequency, hemoglobin and high-sensitive troponin levels, blood pressure variables – systolic and diastolic, pulse pressure, mean, pre-, intra- and post-procedurally.

Comparison of the characteristics of the patients with and without periprocedural hypotension

There was no statistically significant difference between the patients with and the patients without periprocedural hypotension in terms of mean characteristics and frequency of distribution of risk factors. We used the independent sample t-test with level of significance 0.05 and the corresponding confidence interval and test for equality of variance (Levene's test).

We applied the chi-square test for the association between periprocedural hypotension and mortality. The null hypothesis stated that mortality was independent of periprocedural hypotension. The test statistics returned p=0.26 (α=0.05). Thus, we did not reject the null hypothesis, there is not enough evidence to suggest any association between periprocedural hypotension and mid-term death at follow-up.

Survival analysis

For the survival analysis, we first checked the Kaplan-Meier survival curves for periprocedural or postprocedural hypotension. There was no significant difference between the survival time of patients both with periprocedural or procedural hypotension and the patients without hypotension. The results are shown in **Fig. 1**.

Factors for mid-term mortality after carotid stenting

Cox regression analysis was used to investigate how different factors affected when a death would occur. We worked with CI for exp (B) 95% (HR), entry 0.05, removal 0.10. Only tests analysis with Omnibus test <0.05 was used for explanation of the model, because Cox regression was good to fit the given data.

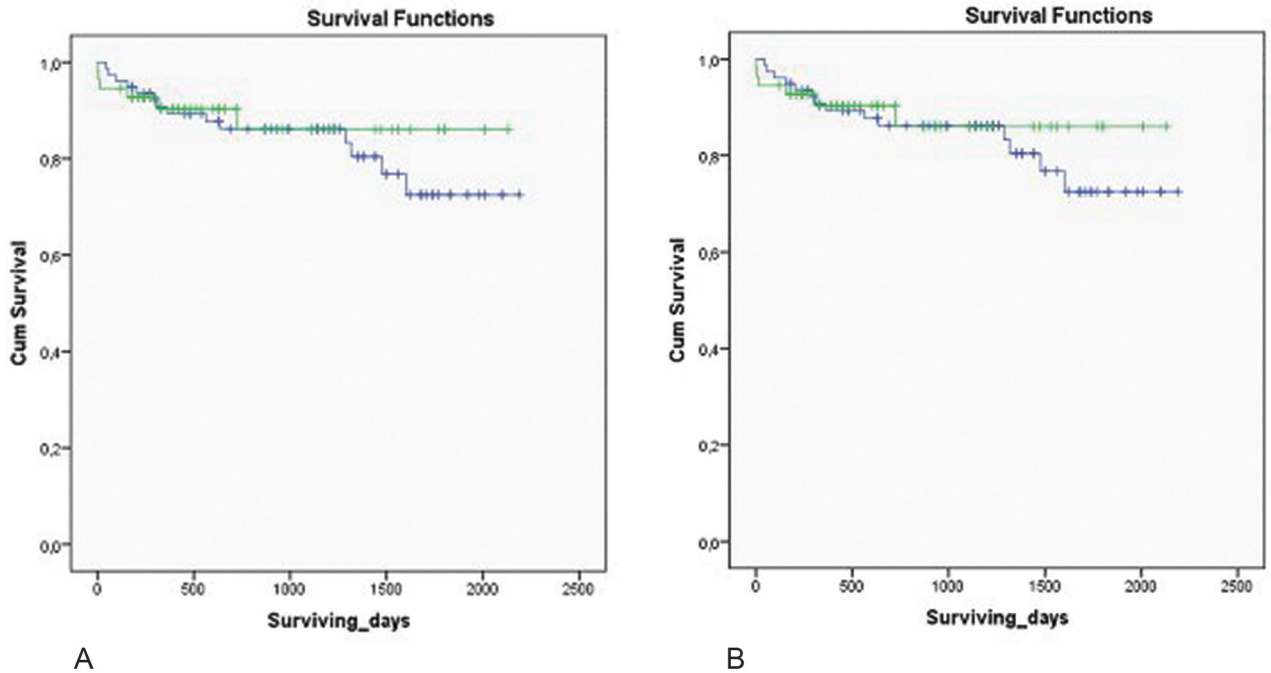

Figure 1. Survival function - hypotension post carotid stenting (**A**); survival function for hypotension periprocedurally (**B**).

Folia Medica

The analysis showed that hypotension during and after carotid stenting had no significant effect on the mid-term all-cause mortality. The same was valid for the precise values of periprocedural blood pressure. These parameters were not associated with significant hazard in any Cox regression mode. These results are shown in **Table 2**.

DISCUSSION

This study population was one of a very high cardiovascular risk. About 96% of the study group were with arterial hypertension, 39% with diabetes, and around 71% with renal dysfunction. These three groups had an elevated risk for maladaptive and impaired acute blood pressure response. [23] Forty-one percent of the patients in the study group had periprocedural hypotension, but this was not associated with elevated all-cause mid-term mortality. Several relatively small studies have found the periprocedural hypotension correlated with periprocedural neurological findings[24] but not with periprocedural mortality.[1,6,25] Data for the long-term effect of periprocedural hypotension is scarce. Some studies find postprocedural (at 6 hours) hypotension to be a positive prognostic factor for prolonged up to one-year beneficial antihypertensive effect.[26]

A postprocedural change in the neurological status was observed only in patients with complications as stroke and transitory ischemic attack, irrespective of periprocedural hypotension. This was a confirmative result compared to other studies.[27]

The results from the Cox regression analysis showed that periprocedural hypotension was not a factor for elevated mid-term all-cause mortality. In the regression model, a certain value gained: previous myocardial infarction, low, ejection fraction, periprocedural complications (the majority of which transitory ischemic attacks). These factors play a respective role for the mortality rate in the very high cardiovascular risk group patients.[28,29]

Our results do not contradict any of these findings. They rather shed light on the part of periprocedural hypotension and mid-term all-cause mortality. We could not prove periprocedural hypotension to be a risk factor for elevated mid-term all-cause mortality after carotid stenting. Even if we consider that the cerebral circulation of such patients with very high cardiovascular risk and significant carotid stenosis has repetitively been put under low-flow conditions, the compensatory mechanisms are strained to their limit, the autoregulation – partly dysfunctional, the clinical result of excessive hypotension, expressed in elevated mortality rate, was not proved. It is evident, also from the diverse studies' findings, that a much larger, complicated and well pre-defined study with functional neuroimaging is needed to elucidate the precise connection between periprocedural findings, neurological, hemodynamic findings, and mortality. Our explanation for the result is that the hypotension was relatively short in duration because immediate counteractions were undertaken to resolve it. Another possible explanation could be that hypotension prevents brain edema at the time of reperfusion.[30]

Limitations

This study is retrospective. Some patients were included in the study by chance at a later point in time, and thus not all alive patients were followed for an equal amount of time. However, these were real-life results, and they did not follow our prescribed protocols. Blood pressure variability was not assessed. It is possible that exaggerated blood pressure variability at the time around carotid stenting plays an important prognostic role with straining the compensatory mechanisms to the full.

CONCLUSIONS

Hypotension during carotid stenting was not associated with a negative neurological outcome as well as with elevation in mid-term all-cause mortality, despite the advanced age of the patients and the impaired from cardio-vascular risk factors cerebral circulation in this symptomatic group. The finding can be due to the relatively short period of hypotension and to the quick administration of vasopressors and volume substitution.

Table 2. Variables in the equation for Cox regression analysis of the factors for all-cause mid-term mortality in patients with carotid stenting. Hypotension was excluded from this final table because its significance level was above the threshold of 0.05

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
MI	1.226	0.511	5.759	1	0.016	3.408	1.252	9.278
EF	–0.083	0.022	13.598	1	0.000	0.920	0.881	0.962
PAD	–2.275	0.807	7.944	1	0.005	0.103	0.021	0.500
TIA	0.970	0.502	3.736	1	0.053	2.638	0.987	7.051
Complications	1.419	0.701	4.091	1	0.043	4.132	1.045	16.340

MI: myocardial infarction; EF: left ventricular ejection fraction; PAD: peripheral artery disease; TIA: transitory ischemic attack

Acknowledgements

The authors have no support to report.

Funding

The authors have no funding to report.

Competing Interests

The authors have declared that no competing interests exist.

REFERENCES

1. Lavoie P, Rutledge J, Dawoud M, et al. Predictors and timing of hypotension and bradycardia after carotid artery stenting. Amer J Neuroradiol 2008; 29(10):1942–7.
2. Andani R, Khan YS. Anatomy. Head and Neck, Carotid Sinus. [Updated 2020 Feb 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554378
3. Aboyans V, Ricco J, Bartelinf M, et al. 2017 ESC Guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS): Document covering atherosclerotic disease of the extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries. Endorsed by: the European Stroke Organization (ESO), The Task Force for the Diagnosis and Treatment of Peripheral Arterial Diseases of the European Society of Cardiology (ESC) and of the European Society for Vascular Surgery (ESVS). Eur Heart J 2018; 39(9):763–816.
4. Zarrinkoob L, Wahlin A, Ambarki K, et al. Blood flow lateralization and collateral compensatory mechanisms in patients with carotid artery stenosis. Stroke 2019; 50:1081–8.
5. Lin P, Ahou W, Kougias P, et al. Factors associated with hypotension and bradycardia after carotid angioplasty and stenting. J Vasc Surg 2007; 46:846–54.
6. Nanto M, Goto Y, Yamamoto H, et al. Complications and predictors of hypotension requiring vasopressor after carotid artery stenting. Neurol Med Chir (Tokyo) 2017; 57(3):115–21.
7. Mylonas S, Moulakakis K, Antonopoulos C, et al. Carotid artery stenting-induced hemodynamic instability. J Endovasc Ther 2013; 20(1):48–60.
8. Wu T, Ham S, Katz S. Predictors and consequences of hemodynamic instability after carotid artery stenting. Ann Vasc Surg 2015; 29(6):1281–5.
9. Bussiere M, Lownie S, Lee D, et al. Hemodynamic instability during carotid artery stenting: the relative contribution of stent deployment versus balloon dilation. J Neurosurg 2009; 110(5):905–12.
10. Nonaka T, Oka S, Miyata S, et al. Risk factors of postprocedural hypotension following carotid artery stenting. Interv Neuroradiol 2006; 12:205–10.
11. Arhuidese I, Ottinger M, Shukla A, et al. Hemodynamic events during carotid stenting are associated with significant periprocedural stroke and adverse events. J Vasc Surg 2020; 71(6):1941–53.
12. Boulanger M, Touze E. Periprocedural risk of myocardial infarction after carotid endarterectomy and carotid angioplasty and stenting. Arch Cardiovasc Dis 2016; 109:159–62.
13. Tomsick T. TIMI, TIBI, TICI: I came, I saw, I got confused. Am J Neuroradiol 2007; 28(2):382–4.
14. Bonati LH, Kakkos S, Berkefeld J, et al. European Stroke Organisation guideline on endarterectomy and stenting for carotid artery stenosis. Eur Stroke J 2021; 6(2):I-XLVII. Available from: https://journals.sagepub.com/doi/pdf/10.1177/23969873211012121, retrieved on 06.07.2021
15. Liu Y, Qin H, Zhang B, et al. Efficacy of different types of self-expandable stents in carotid artery stenting for carotid bifurcation stenosis. J Huazhong Univ Sci Technolog Med Sci 2016; 36(1):95–8.
16. Castriota F, Liso A, Biamino G, et al. Technical evolution of carotid stents. Interv Cardiol Rev Available from: https://www.icrjournal.com/articles/technical-evolution-carotid-stents (Retrieved on 06.07.2021).
17. Tallarite T, Rabinstein A, Cloft H, et al. Are distal protection devices "protective" during carotid angioplasty and stenting? Stroke 2011; 42:1962–6.
18. Ahn S, Prince E, Dubel G. Carotid artery stenting: review of technique and update of recent literature. Semin Intervent Radiol 2013; 30(3):288–96.
19. Ogata A, Sonobe M, Kato N, et al. Carotid artery stenting without post-stenting balloon dilatation. J Neurointerv Surg 2014; 6:517–20.
20. Unger T, Borghi C, Charchar F, et al. 2020 International Society of Hypertension global hypertension practice guidelines. Hypertension 2020; 75(6):1334–57.
21. McEniery C, Cockcroft J, Roman M, et al. Central blood pressure: current evidence and clinical importance. Eur Heart J 2014; 35(26):1719–25.
22. Mitchell G. Central pressure should not be used in clinical practice. Artery Res 2015; 9:8–13.
23. De Boer I, Bangalore S, Benetos, et al. Diabetes and hypertension: a position statement by the American Diabetes Association. Diabetes Care 2017; 40(9):1273–84.
24. Goksal E, Niftaliyev E, Deniz C, et al. Prolonged hypotension after carotid artery stenting: incidence, predictors and consequences. Acta Neurochir (Wien) 2017; 159(11):2081–7.
25. Mylonas S, Moulakakis K, Antonopoulos C, et al. Carotid artery stenting-induced hemodynamic instability. J Endovasc Ther 2013; 20(1):48–60.
26. Chang A, Hung H, Hsieh F, et al. Beneficial effects of prolonged blood pressure control after carotid artery stenting. Clin Interv Aging 2017; 12:103–9.
27. Mlekusch W, Schillinger M, Sabeti S, et al. Hypotension and bradycardia after elective carotid stenting: frequency and risk factors. J Endovasc Ther 2003;10(5):851-859. doi:10.1177/152660280301000501
28. Arnett D, Blumenthal R, Albert M, et al. 2019 ACC/AHA Guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidliens. Circulation 2019; 140;e596–e646.
29. Pirpoli M, Hoes A, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. Eur Heart J 2016; 37:2315–81.
30. Farooq M, Goshgarian C, Min J, et al. Pathophysiology and management of reperfusion injury and hyperperfusion syndrome after carotid endarterectomy and carotid artery stenting. Exp Transl Stroke Med 2016; 8(1):1–8. doi.org/10.1186/s13231-016-0021-2

Роль перипроцедуральных гемодинамических переменных во время стентирования сонных артерий в среднесрочной общей смертности у пациентов пожилого возраста

Теодора Янева-Сиракова[1], Галина Златанчева[1], Кирил Карамфилов[2], Лачезар Трайков[3], Иво Петров[1], Добрин Василев[4]

[1] *Аджибадем Сити Клиник, Сърдечно-сосудистый центр, Софийский университет, Болгария*

[2] *УМБАЛ "Александровска", София, Болгария*

[3] *Медицинский университет – София, София, Болгария*

[4] *УМБАЛ „Медика Кор", Русенский университет, Русе, Болгария*

Адрес для корреспонденции: Теодора Янева-Сиракова, Аджибадем Сити Клиник, Сърдечно-сосудистый центр, Софийский университет, Болгария; Email: teodora.yaneva@gmail.com

Дата получения: 14 января 2023 ♦ **Дата приемки:** 15 мая 2023 ♦ **Дата публикации:** 31 декабря 2023

Образец цитирования: Yaneva-Sirakova T, Zlatancheva G, Karamfiloff K, Traykov L, Petrov I, Vassilev D. The role of periprocedural hemodynamic variables during carotid stenting for the mid-term general mortality in advanced age patients. Folia Med (Plovdiv) 2023;65(6):902-908. doi: 10.3897/folmed.65.e100100.

Резюме

Введение: Стентирование сонной артерии может вызвать значительную брадикардию и/или гипотонию. Это может иметь негативные краткосрочные и долгосрочные последствия для пожилых пациентов из группы высокого риска. У них церебральная гемодинамика находится в исчерпанном адаптационном потенциале из-за множественных сердечно-сосудистых факторов риска, пожилого возраста и выраженного стеноза.

Цель: Это было ретроспективное исследование, целью которого было выяснить, имеет ли перипроцедурная гипоперфузия или гипотензия во время стентирования сонной артерии какое-либо значение для острого неврологического исхода и среднесрочной общей смертности у пациентов пожилого возраста, которые подвергались высокому риску хирургической эндартерэктомии.

Материалы и методы: Мы обследовали 138 последовательных пациентов со значительным стенозом сонной артерии с января 2015 г. по июль 2019 г. Средний возраст составил 67.41 (10.70) года. Средний период наблюдения составил 31 месяц (922 дня). Пациенты находились под гемодинамическим контролем перипроцедурно в соответствии с местным протоколом. Вазопрессоры добавляли, если отмечалась длительная гипотония. Статистические данные анализировали с использованием SPSS IBM v. 19 (p=0.05, CI 95%).

Результаты: Пациентов мужского пола было 94 (68%). Число пациентов с артериальной гипотензией перипроцедурно или в первые 6 часов после процедуры составило 55 (42%). Среднее артериальное давление составляло 135/83 mmHg. до, 116/76 mmHg. во время и 121/73 mmHg. после процедуры. Анализ Kaplan-Maier не выявил существенных различий в среднесрочной общей смертности между пациентами с транзиторной гипотензией и пациентами без неё. Не было никакой разницы и в постпроцедурном неврологическом исходе.

Заключение: Наличие гипотонии во время стентирования сонной артерии не было связано с отрицательным неврологическим исходом. Он также не увеличил среднесрочную смертность от всех причин у пожилых пациентов (средний возраст 67 лет). Это открытие можно объяснить относительно коротким периодом гипотонии, немедленным назначением вазопрессоров или предотвращением отёка мозга и гиперреперфузии во время стентирования сонных артерий в условиях повышенного артериального давления; однако это ещё предстоит определить.

Ключевые слова

неврологический исход, падение систолического артериального давления