

Safety of balloon pre-dilatation in the treatment of severe carotid artery stenosis

M. SAHIN, M.V. YAZICIOGLU, G. ACAR, S. DEMIR, M.E. KALKAN, B. OZKAN, G. ALICI, T. AKGUN, M. AKCAKOYUN, B. BOZTOSUN

Department of Cardiology, Kartal Kosuyolu Yuksek Ihtisas Heart-Education and Research Hospital, Istanbul, Turkey

Abstract. – **AIM:** To assess early outcome of predilatation prior stenting of severe carotid artery stenosis and to evaluate early major adverse cardiovascular and cerebral events (MACCE).

PATIENTS AND METHODS: The study group consisted of 265 consecutive patients (200 males, 65 female, mean age 66.7 ± 8.6 years) in whom 275 percutaneous transluminal angioplasty (PTA) procedures of carotid arteries were performed. Staged carotid stenting was performed in patients with bilateral carotid stenosis. Neuroprotection with a distal protection device was used in all cases. The patients were divided into two groups: direct carotid stent implantation without previous pre-dilatation was performed in 233 patients (direct stenting group) and predilatation was performed in 42 patients (predilatation group). Early events were recorded and analyzed subsequently.

RESULTS: We treated 275 carotid stenoses and the stent was implanted in all patients. Ten patients (3.7%) were treated by staged carotid artery stenting (CAS) due to bilateral carotid artery disease. The technical success rate was 97.1%. During 1-month follow-up, the prevalence of primary endpoint was 2.18%. The prevalence of MACCE at 30 days was higher in the predilatation group (2.4% vs. 2.1%; $p = 0.924$). Also periprocedural rate of hypotension was higher in predilatation group (7.1% vs. 1.7%; $p = 0.04$).

CONCLUSIONS: Balloon predilatation prior to stenting can be performed to treat severe carotid artery stenosis with acceptable periprocedural complication rate.

Key Words:

Carotid artery stenosis, Balloon pre-dilatation, Complication rate.

Introduction

In developed countries, stroke is the common cause of death¹. Every year, 1.5 million people in Europe and the United States suffer from stroke, and 30-40% of all cases are caused by athero-

sclerotic carotid artery disease². Severe stenoses ($\geq 80\%$) are probably associated with an increased risk of stroke when compared to moderate (60-79%) carotid artery stenosis and warrant much more aggressive revascularization³. Carotid artery stenting (CAS) has replaced surgical endarterectomy in many patients in recent years⁴. But fearing the risk of stroke, endarterectomy was generally preferred in severe carotid stenosis. However, the data are quite limited⁵ and the safety of CAS with predilatation has not been investigated systematically in large series. Therefore, we retrospectively analyzed the early outcomes of CAS with and without predilatation. The aim of this analysis was to evaluate the safety of CAS with predilatation in procedural and short-term follow-up.

Patients and Methods

Between May 2009 and June 2012, a total of 273 consecutive patients and 283 atherosclerotic carotid artery stenosis underwent CAS at the Kartal Kosuyolu Yuksek Ihtisas Heart-Education and Research Hospital Education. Patients were divided into 2 groups according to the method of stenting. Predilatation was performed (predilatation group) in forty-two patients and direct stenting was performed in the remaining patients ($n=233$) (direct stenting group). All the stenoses were quantified angiographically according to the NASCET (North American Symptomatic Carotid Endarterectomy Trial) criteria⁶. The criteria to qualify for invasive treatment included the presence of asymptomatic stenosis of carotid artery of $> 70\%$ or stenosis of $> 50\%$ in patients with a history of cerebrovascular events and/or critical stenosis of the contralateral carotid artery. Patients were excluded from the procedure only in case of total occlusion of carotid artery or ab-

solute contraindications for double anti-platelet therapy (active gastrointestinal bleeding, recent hemorrhagic stroke, allergy to aspirin), nonatherosclerotic carotid artery stenosis such as Takayasu's arteritis and fibromuscular dysplasia. Patients with a history of incomplete data were also excluded. In order to minimize the risk of thromboembolic complications, asymptomatic and symptomatic patients received aspirin (300 mg/d) and clopidogrel (75 mg/d) at least 2 and 5 days prior to the procedure respectively. In case of angiographic and ultrasonographic evidence of unstable atherosclerotic plaque containing clots, patients were pretreated with anti-platelet agents and anticoagulants (aspirin, clopidogrel, low-molecular-weight heparin) for 2-4 weeks. The proximal balloon protection device (e.g., MOMA) was used or endarterectomy was performed in patients with persistent thrombus despite the treatment. In addition, patients received their regular antihypertensive medications on the morning of the procedure. Sedatives were not applied prior to the procedure to facilitate accurate neurological assessment during angioplasty. The CAS procedures were all performed under local anesthesia via femoral access. An 8F introducer sheath was positioned and heparin (UFH 100 IU/kg of body weight) was administered to provide prolongation of ACT (activated clotting time) to 250-300 s. Then continuous arterial pressure and electrocardiographic monitoring were performed during the procedure. Then a guiding catheter 8F JR4 (Cordis Corporation, Miami, FL, USA) was placed proximal to the target lesion. A distal embolic protection with a vascular filter was used in all patients during CAS procedures. Following placement of the protection devices procedures were performed with predilation (2 mm to 3 mm), stent deployment, and postdilation (4.0 mm-6.0 mm). Pre-dilatation was performed only in case of critical stenosis (> 95%) or when severe calcifications were seen under fluoroscopy to facilitate the stent's passage of the lesion. Self-expanding stents were implanted into the stenosed carotid arteries. If needed, the lesion was post-dilated to achieve a residual stenosis \leq 30% after stent deployment. To prevent bradycardia and hypotension, 0.5-1.0 mg of atropine was routinely administered intravenously prior to balloon inflation. During the procedure, atropine (0.5-1.0 mg) was given again for patients who developed a > 20 beats/min drop in their heart rate during balloon dilatation and stent deployment. In case of profound hypotension (systolic blood pressure < 80

mmHg) infusion of inotropic agents (dopamine 5-15 μ g/kg/min) in addition to fluids was used. Before retrieval of the protection device, final biplane angiogram of the stented lesion and intracranial views were obtained. The procedure was considered successful when the stenotic segment of the carotid artery was effectively dilated (residual stenosis < 30% with adequate blood flow). After the procedure the anti-platelet therapy was continued (clopidogrel for the first 4-6 weeks in asymptomatic patients, 3 months in symptomatic patients and aspirin indefinitely). All patients were followed up in intensive care unit (ICU) for the first 3 hours after procedure. All the patients were repeatedly examined by physicians (including neurological examination) and all symptomatic patients were examined by board-certified neurologists. Post-procedural intracranial examination with magnetic resonance imaging (MRI) or computed tomography (CT) was performed if the neurological status of the patient changed. Meticulous blood pressure control was performed to maintain the systolic blood pressure between 100 mmHg and 130 mmHg or lower the blood pressure to 10% to 20% below baseline. All cardiovascular, neurological and local complications occurring during the index hospitalisation and after discharge, up to 30 days following the procedure, were recorded. Major adverse cardiac and cerebrovascular events (MACCE) such as myocardial infarctions (MI), stroke and death were also evaluated in this time period. Stroke was defined as a neurological deficit that persisted more than 24 h. Transient cerebral ischaemia following the procedure was defined as occurrence of new or exacerbation of previously existing neurological symptoms with total recovery within 24 hours. The diagnosis of Q-wave MI was based on the presence of new Q waves on the ECG and an elevated creatine kinase at least 2 times the upper limit of the normal range with an elevated level of MB isoenzyme. A non Q-wave MI had no new pathologic Q-waves on ECG and a total creatine kinase of greater than two times the normal with an elevated MB isoenzyme. The ECG was performed persistently during the first 24 hours and the remaining period of hospitalization in case of unexplained chest pain.

Statistical Analysis

The data was analyzed using the Statistical Package for the Social Sciences (SPSS) software for Windows version 13.0 (SPSS Inc., Chicago, IL, USA). Parametric variables were reported as

means and standard deviations, non-parametric as absolute numbers and percentages. Parametric variables were compared between groups using Student's *t*-test and Mann Whitney U test. Comparisons of the frequency of the non-parametric variables between subgroups were carried out using χ^2 test. $p < 0.05$ was considered statistically significant.

Results

In 273 patients, 283 CAS procedures were performed in our Hospital. Of these, 275 (97.1%) (206 men and 69 women; mean age 66.7 ± 8.6 years) procedures were successful. We did not succeed in 8 cases due to diffuse carotid stenosis, severe distortions in the internal carotid artery and anatomic abnormality of aortic arch. Direct carotid stent implantation without previous pre-dilation was performed in 233 patients, and predilatation was performed in 42 patients. 10 patients (3.7%) had a bilateral carotid artery disease. These patients were treated by staged CAS. Simultaneous bilateral carotid artery stenting was not performed in such patients. Table I shows the baseline characteristics that, in general, were similar in both groups. However, the predilatation group had a higher percentage of renal insufficiency (12.9% predilatation versus 3.9% direct stenting; $p = 0.032$). The percentage of patients who smoke (33.6% vs. 14.3%; $p = 0.012$), previous myocardial infarction (22.8% vs. 7.1%; $p = 0.024$) and history of coronary artery bypass grafting (24.1% vs. 7.1%; $p = 0.014$) was higher in direct stenting

group compared predilatation group. Interventional and angiographic characteristics of the study population are summarized in Table II. Predilatation group were associated with a greater degree of angiographic stenosis ($96.7\% \pm 2.3\%$ vs. $82.9\% \pm 11.1\%$; $p < 0.001$), and a greater proportion contralateral stenosis (35.7% vs. 15.9%; $p = 0.003$). The detailed data of all early events observed in both groups are presented in Table III. Postprocedural strokes occurred in 4 patients (1 and 3 strokes in predilatation group and direct stenting group, respectively). Two deaths occurred in direct stenting group. One died as a consequence of periprocedural ischemic stroke. The other patient underwent coronary stenting 15 days after the CAS procedure. This patient died as a result sudden death 2 days after the coronary stenting. There was no periprocedural myocardial infarction. The incidence of MACCE at 30 days was higher insignificantly in the predilatation group (2.4% vs. 2.1%; $p = 0.924$). Transient ischemic attacks incidence at 30 days was higher direct stenting group (0% vs 5.1%; $p = 0.044$). On the other hand, periprocedural rate of hypotension was higher in predilatation group (7.1% vs. 1.7%; $p = 0.04$).

Discussion

The choice of revascularization in carotid stenosis is still uncertain. The risk of embolism is higher in patients with severe carotid stenosis. Embolus can occur when the guide wire and filter passes through or during the predilatation. In this study, we compared the short-term outcomes

Table I. Baseline clinical characteristics of the patients.

Variables	Predilatation group n (%)	Direct stenting group n (%)	<i>p</i>
Age, years, mean \pm SD	67.9 \pm 7.4	66.4 \pm 8.7	0.286
Sex, men	34 (81)	172 (73.8)	0.326
Hypertension	34 (81)	200 (85.8)	0.413
Diabetes mellitus	14 (34.1)	82 (35.3)	0.882
Hyperlipidemia	19 (45.2)	130 (55.8)	0.206
Renal insufficiency	4 (12.9)	8 (3.9)	0.032
Current smokers	6 (14.3)	78 (33.6)	0.012
Coronary artery disease	25 (59.5)	173 (74.2)	0.057
Previous myocardial infarction	3 (7.1)	51 (22.8)	0.024
History of coronary artery bypass grafting	3 (7.1)	56 (24.1)	0.014
Previous coronary artery intervention	7 (18.4)	74 (32.9)	0.074
Peripheral arterial disease	8 (19.5)	31 (13.5)	0.316
Symptomatic	23 (54.8)	98 (42.1)	0.127
Hospitalization time, mean \pm SD	1.7 \pm 1.0	2.2 \pm 2.3	0.103

SD: standard deviation.

Table II. Interventional and angiographic characteristics of the study population.

Variable	Predilatation group n (%)	Direct stenting group n (%)	p
Lesion located in LICA/RICA	25/17 (59.5/40.5)	125/108 (53.6/46.4)	0.481
Stenosis at baseline (%)	96.7 ± 2.3	82.9 ± 11.1	< 0.001
Contralateral occlusion	1 (2.4)	17 (7.2)	0.236
Contralateral stenosis > 50%	15 (35.7)	37 (15.9)	0.003
Mean stent length [mm]	37.7±6.9	37.9 ± 7.5	0.982
Application of embolism protection device	42 (100)	233 (100)	NS
Implantation of stents	42 (100)	233 (100)	NS
Rate of post-dilations	40 (95.2)	207 (88.8)	0.207

LICA: left internal carotid artery; RICA: right internal carotid artery.

of patients treated by direct CAS and CAS with predilatation. Our study suggested that, in our center carotid stenting is performing with acceptable frequency of major cerebral events. MACCE was similar in CAS with predilatation and direct stenting group. However, the rate of hypotension and bradycardia was higher in predilatation group. Extracranial carotid atherosclerosis with the resulting atherothromboembolism may account for up to 20% of ischemic strokes⁷. The enlargement of a narrowed cerebral blood vessel in patients with stenosis of ≥ 70% reduces the absolute five-year risk of ischemic stroke by 16%⁸. Multiple treatments have been shown efficacious in treating carotid disease. Carotid endarterectomy (CEA) has been shown to be effective in significantly reducing the risk of recurrent stroke emanating from that pathological nidus⁹. On the other hand, CAS as a novel method for carotid revascularization has been widely used in patients with significant carotid artery stenosis as an alternative to CEA in the past ten years¹⁰. There are many studies comparing CAS and CEA. EVA-3S (Endarterectomy Versus Angioplasty in Patients with Severe Symptomatic

Carotid Stenosis) study¹¹ suggested that the 30-day appearance of any stroke or death was higher in the stenting group (9.6% vs. 3.9%). There was no significant difference in the rates of the primary end point (stroke, death and myocardial infarction) between CAS and CEA (7.2% vs. 6.8%; $p < 0.51$) in CREST (Carotid Revascularization Endarterectomy versus Stenting Trial) trial¹². But in the same study, stroke rate was higher in the CAS group; myocardial infarction rate was higher in CEA group (stroke 4.1% vs. 2.3%, $p < 0.012$; and myocardial infarction 1.1% vs. 2.3%, $p < 0.032$). In general, 30 day adverse outcome (stroke/death rate) for CAS ranged from 1.2% to 7%¹³. Current recommendations of the American Heart Association concerning procedures on the carotid arteries describe the acceptable frequency of major cerebral events (stroke, death) occurrence at the level of 3% for asymptomatic patients and 6% for patients with symptomatic occlusion of the carotid artery¹⁴.

In our series, the 30 day composite rate of any stroke or death was 2.18% in all patients. These results are considerably below the maximum complication rates recommended in the American

Table III. Thirty-day incidence of complications in groups.

Type of complication	Predilatation group n (%)	Direct stenting group n (%)	p
Death	0 (0)	2 (0.9)	0.547
Stroke	1 (2.4)	3 (1.3)	0.586
Myocardial infarction	0 (0)	0 (0)	NS
Transient ischaemic attacks	0 (0)	12 (5.1)	0.044
Acute renal failure	0 (0)	8 (3.4)	0.223
Hypotension requiring prolonged (> 12 h administration of catecholamines)	3 (7.1)	7 (2.5%)	0.04
MACCE	1 (2.4)	6 (2.18%)	0.924
		5 (2.1)	

MACCE: major adverse cardiac and cerebrovascular events.

Heart Association guidelines for carotid stenting. Our better results may be due to better pre-procedural preparation and proper selection of patients. All patients received dual antiplatelet therapy a few days prior to the procedure. In case of evidence of unstable atherosclerotic plaque containing fresh clots, patients were pretreated with antiplatelet agents and anticoagulants for a few weeks. Statin therapy was also given to all patients. There was no periprocedural myocardial infarction in our study. In case of severe stenosis of the coronary artery, stenting procedures were performed before CAS. The lack of MI may be due to this preparation. In severe carotid stenosis, embolism is a major concern with endovascular treatment and delayed carotid artery intervention compared with the peripheral and coronary interventions¹⁵. This risk can be at any stage of endovascular treatment¹⁶. Numerous investigations have shown procedure-related strokes by using MR imaging and transcranial Doppler monitoring^{17,18}. Embolic signals have been detected during balloon predilatation, stent deployment, and post-stent balloon dilatation¹⁹. One study found that the highest embolic loads occurred during balloon predilatation²⁰. Another research has found that the highest embolic counts during stent deployment²¹. On the other hand, some reports showed that most emboli are produced by post-stent dilatation^{22,23}. In addition to being a high risk of embolism, the success rates are lower in severe carotid stenosis. Generally, the success rate of direct stenting in severe stenosis is about 80%. In the remaining 20%, predilatation is needed²⁴.

Until recently, CEA was the preferred strategy for these patients. We can hypothesize that the predilatation before stenting of critical carotid stenoses is safe. In a small-scale work by Zhang C et al⁵, it was shown that after balloon pre-dilatation, CAS was a safe and effective treatment of carotid artery subtotal occlusion. In our report, the risk of adverse event occurrence in the early 30-day observation period was 2.4% in the predilatation group and 2.1% in the direct stenting group. Symptomatic status, stenosis severity, contralateral occlusion and contralateral stenosis are commonly used to estimate stroke risk among patients with carotid stenosis²⁵. In our study the degree of baseline angiographic stenosis, the rate of contralateral stenosis and the ratio of symptomatic patients were more in predilatation group compared to direct stenting group. Despite the higher risk profile for stroke, the short-term result was acceptable in predilatation group.

Carotid sinus reaction including bradycardia, asystole, and hypotension is one of the most common complications of internal carotid artery angioplasty. It can occur during or after CAS due to over stretching of the carotid sinus baroreceptors by the balloon or the stent²⁶. Hypotension and bradycardia, in our series, occurred in 2.54% of the cases during the procedure, and was found to be lower than other studies^{27,28}. Major predictors for hemodynamic instability were a stenosis over 90% (25), aggressiveness of dilation²⁹, fibrous tissue is composed of more than 60% of the whole plaque volume, the prior use of beta-blockers and the absence of diabetes mellitus³⁰. In our case load, the rate of hypotension and bradycardia was higher in predilatation group. In predilatation group, angiographic stenosis was more severe and the lesions were more calcific. Moreover, postdilatation was more common in this group. These may contribute to higher rate of hypotension in predilatation group.

Conclusions

Our results show that balloon pre-dilatation, with the distal embolic protection device, can be performed to treat severe carotid artery stenosis with acceptable safety. Large-scale prospective trials are required for the optimal therapeutic strategy for severe carotid stenosis.

Disclosure of Interest

The Authors have no conflict of interest to declare.

References

- 1) DE WEERD M, GREVING JP, HEDBLAD B, LORENZ MW, MATHIESEN EB, O'LEARY DH, ROSVALL M, SITZER M, BUSKENS E, BOTS ML. Prevalence of asymptomatic carotid artery stenosis in the general population: an individual participant data meta-analysis. *Stroke* 2010; 41: 1294-1297.
- 2) SUDLOW CL, WARLOW CP. Comparing stroke incidence worldwide: what makes studies comparable? *Stroke* 1996; 27: 550-558.
- 3) CHAMBERS BR, NORRIS JW. Outcome in patients with asymptomatic neck bruits. *N Engl J Med* 2003; 315: 860-865.
- 4) LEVY E, YAKUBOVITCH D, RUDIS E, ANNER H, LANDSBERG G, BERLATZKY Y, ELAMI A. The role of combined carotid endarterectomy and coronary artery bypass grafting in the era of carotid stenting in view of long-term results. *Interact Cardiovasc Thorac Surg* 2012; 15: 984-988.

- 5) ZHANG C, WANG Z, ZHENG H, QIU Y, HE X, YANG H, LIU W. Self-expanding stents in the treatment of carotid artery subtotal occlusion: a clinical study on the patients of Hubei and Sichuan in China. *Biomed Mater Eng* 2012; 22: 27-33.
- 6) U-KING-IM JM, TRIVEDI RA, CROSS JJ, HIGGINS NJ, HOLLINGWORTH W, GRAVES M, JOUBERT I, KIRKPATRICK PJ, ANTOUN NM, GILLARD JH. Measuring carotid stenosis on contrast-enhanced magnetic resonance angiography: diagnostic performance and reproducibility of 3 different methods. *Stroke* 2004; 35: 2083-2088.
- 7) PETTY GW, BROWN RD JR, WHISNANT JP, SICKS JD, O'FALLON WM AND WIEBERS DO. Ischemic stroke subtypes: a population-based study of incidence and risk factors. *Stroke* 1999; 30: 2513-2516.
- 8) WAUWER A, VAN LEEUWEN MS, VAN OSCH MJ, VAN DER WORP BH, MOLL FL, LO RT, MALI WP, PROKOP M. Changes in cerebral perfusion after revascularization of symptomatic carotid artery stenosis: CT measurement. *Radiology* 2007; 245: 541-548.
- 9) TAYLOR DW, BARNETT HJM. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991; 325: 445-453.
- 10) WHOLEY MH, AL-MUBARAK N, WHOLEY MH. Updated review of the global carotid artery stent registry. *Catheter Cardiovasc Interv* 2003; 60: 259-266.
- 11) MAS JL, CHATELLIER G, BEYSSSEN B, BRANCHEREAU A, MOULIN T, BECQUEMIN JP, LARRUE V, LIÈVRE M, LEYS D, BONNEVILLE JF, WATELET J, PRUVO JP, ALBUCHER JF, VIGUIER A, PIQUET P, GARNIER P, VIADER F, TOUZÉ E, GIROUD M, HOSSEINI H, PILLET JC, FAVROLE P, NEAU JP, DUCROCO X; EVA-3S INVESTIGATORS. Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. *N Engl J Med* 2006; 355: 1660-1671.
- 12) HOBSON RW 2ND, HOWARD VJ, ROUBIN GS, BROTT TG, FERGUSON RD, POPMA JJ, GRAHAM DL, HOWARD G; CREST INVESTIGATORS. Carotid artery stenting is associated with increased complications in octogenarians: 30-day stroke and death rates in the CREST lead-in phase. *J Vasc Surg* 2004; 40: 1106-1111.
- 13) GOODNEY PP, SCHERMERHORN ML, POWELL RJ. Current status of carotid artery stenting. *J Vasc Surg* 2006; 43: 406-411
- 14) MOORE WS, BARNETT HJ, BEEBE HG, BERNSTEIN EF, BRENER BJ, BROTT T, CAPLAN LR, DAY A, GOLDSTONE J, HOBSON RW 2ND, ET AL. Guidelines for carotid endarterectomy: a multidisciplinary consensus statement from the ad hoc committee, American Heart Association. *Stroke* 1995; 26: 188-201.
- 15) LOWNIE SP, PELZ DM, LEE DH, MEN S, GULKA I, KALAPOS P. Efficacy of treatment of severe carotid bifurcation stenosis by using self-expanding stents without deliberate use of angioplasty balloons. *AJNR Am J Neuroradiol* 2005; 26:1241-1248.
- 16) AYDINER O, BOZTOSUN B, SIRVANCI M, AKÇAKOYUN M, KARAMAN K, AKSOY T, ONAT L. Early and late outcomes of carotid artery stenting. *Anadolu Kardiyol Derg* 2007; 7: 152-157.
- 17) JAEGER HJ, MATHIAS KD, DRESCHER R, HAUTH E, BOCKISCH G, DEMIREL E, GISSLER HM. Diffusion-weighted MR imaging after angioplasty plus stenting of arteries supplying them brain. *AJNR Am J Neuroradiol* 2001; 22: 1251-1259.
- 18) GERRATY RP, BOWSER DN, INFELD B, MITCHELL PJ, DAVIS SM. Microemboli during carotid angiography: association with stroke risk factors or subsequent magnetic resonance imaging changes. *Stroke* 1996; 27: 1543-1547.
- 19) VAN HEESEWIJK HP, VOS JA, LOUWERSE ES, VAN DEN BERG JC, OVERTOOM TT, ERNST SM, MAUSER HW, MOLL FL, ACKERSTAFF RG; CAROTID PTA AND STENTING COLLABORATIVE RESEARCH GROUP. New brain lesions after carotid angioplasty and stent placement. *Radiology* 2002; 224: 361-365.
- 20) ORLANDI G, FANUCCHI S, FIORETTI C, ACERBI G, PUGLIOLI M, PADOLECCHIA R, SARTUCCI F, MURRI L. Characteristics of cerebral microembolism during carotid stenting and angioplasty alone. *Arch Neurol* 2001; 58: 1410-1413.
- 21) AL-MUBARAK N, ROUBIN GS, VITEK JJ, IYER SS, NEW G, LEON MB. Effect of the distalballoon protection system on microembolization during carotid stenting. *Circulation* 2001; 104: 1999-2002.
- 22) MARTIN JB, PACHE JC, TREGGIARI-VENZI M, MURPHY KJ, GAILLOUD P, PUGET E, PIZZOLATO G, SUGIU K, GUIMARAENS L, THÉRON J, RÜFENACHT DA. Role of the distal balloon protection technique in the prevention of cerebral embolic events during carotid stent placement. *Stroke* 2001; 32: 479-484.
- 23) VITEK JJ, ROUBIN GS, AL-MUBAREK N, NEW G, IYER SS. Carotid artery stenting: technical considerations. *AJNR Am J Neuroradiol* 2000; 21: 1736-1743.
- 24) VESELKA J, ZIMOLOVÁ P, MARTINKOVI OVÁ L, TOMAŠOV P, HÁJEK P, MALÝ M, SPA EK M, ZEMÁNEK D, TESAŘ D. Comparison of mid-term outcomes of carotid artery stenting for moderate versus critical stenosis. *Arch Med Sci* 2012; 8: 75-80.
- 25) ARONOW HD, GRAY WA, RAMEE SR, MISHKEL GJ, SCHREIBER TJ, WANG H. Predictors of neurological events associated with carotid artery stenting in high-surgical-risk patients: insights from the Cordis Carotid Stent Collaborative. *Circ Cardiovasc Interv* 2010; 3: 577-584.
- 26) NII K, TSUTSUMI M, AIKAWA H, HAMAGUCHI S, ETOU H, SAKAMOTO K, KAZEKAWA K. Incidence of hemodynamic depression after carotid artery stenting using different self-expandable stent types. *Neurol Med Chir* 2011; 51: 556-560.
- 27) POPESCU D, MERGEANI A, BAJENARU OA, ANTOCHI FA. Hemodynamic instability after elective carotid stenting: frequency and risk factors. *Maedica (Buchar)* 2011; 6: 258-261.
- 28) BALDI S, ZANDER T, RABELLINO M, GONZALEZ G AND MAYNAR M. Carotid Artery Stenting without Angioplasty and Cerebral Protection: A Single-Center Experience with up to 7 Years' Follow-Up. *AJNR Am J Neuroradiol* 2011; 32: 759-763.
- 29) GUPTA R, ABOU-CHEBL A, BAJZER CT, SCHUMACHER HC, YADAV JS. Rate, predictors, and consequences of hemodynamic depression after carotid artery stenting. *J Am Coll Cardiol* 2006; 47: 1538-1543.
- 30) TSURUMI A, MIYACHI S, HOSOSHIMA O, IZUMI T, OHSHIMA T, MATSUBARA N, KINKORI T, NAITO T, WAKABAYASHI T. Can periprocedural hypotension in carotid artery stenting be predicted? A carotid morphologic autonomic pathologic scoring model using virtual histology to anticipate hypotension. *Interv Neuroradiol* 2009; 15: 17-28.