Carotid Artery Stenting: Periprocedural Outcomes

Karotis Arter Stentleme: Periprosedural Sonuçlar

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Geliş Tarihi/*Received:* 27.10.2010
Kabul Tarihi/*Accepted:* 02.03.2011

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ABSTRACT Objective: Stroke is the third most common cause of death after cardiac diseases and cancer in western countries. Atherosclerosis is responsible for 90% of cerebral thromboembolic events and 90% of the atherosclerotic lesions of carotid artery system are observed in a 2 cm segment comprising the origin of internal carotid artery (ICA). In treatment of carotid stenosis, the aim is to prevent stroke. Carotid artery stenting (CAS) is an alternative of carotid artery endarterectomy (CEA). There are many studies comparing those two treatment options. In this study we determined the periprocedural neurologic complication rate on carotid artery stenting patients. **Material and Methods:** In this retrospective study, we analyzed the data of 96 cases/105 arteries (59 [61.4%] symptomatic and 37 [38.6%] asymptomatic cases) that were treated for carotid artery stenosis between June 2007 and April 2010. Patient's demographic variables and percentages of stenosis, the number and varieties of used stents and embolic protection devices with periprocedural 30 days stroke, myocardial infarction (MI) and death rate were investigated. Long-term outcomes were excluded from the study because of the limited number of cases and lack of a homogeneous distribution among the treatment lengths. **Results:** In our cases, death rate was 0.9%, minor stroke rate were 4.7%, and stroke/death rate was 5.7%. No transient ischemic attack (TIA), major stroke, or MI was observed. There was no significant difference betwen the symptomatic and asymptomatic patients. **Conclusion:** CAS has acceptable periprocedural complication rates and therefore can be applied in such cases. Currently, it is a good alternative to CEA. Further studies are required to determine the patients that would benefit more from CAS, rather than CEA.

Key Words: Atherosclerosis; carotid artery diseases; stroke; stents

ÖZET Amaç: İnme, batı toplumlarında kalp hastalıkları ve kanserden sonra üçüncü en sık ölüm nedenidir. Aterosklerozis serebral tromboembolik olayların %90'nından sorumludur ve karotis arter sistemindeki aterosklerotik lezyonların %90'ı internal karotid arter (İKA) orjinini içine alan 2 cm'lik segmentte gözlenir. Karotis darlıklarının tedavisinde amaç inmenin önlenmesidir. Karotis arter stentleme (KAS), karotis endarterektominin (KEA) bir alternatifidir. Bu iki tedavi seçeneğini karşılaştıran çok sayıda çalışma bulunmaktadır. Bu çalışmada, KAS uygulanan olgularda periprosedural dönem nörolojik komplikasyon oranlarını araştırdık. **Gereç ve Yöntemler:** Bu retrospektif çalışmada, Haziran 2007 ve Nisan 2010 tarihleri arasında karotid arter darlığı nedeniyle tedavi edilen 96 olgu/105 arterin (59 [%61,4] olgu semptomatik ve 37 [%38,6] olgu asemptomatik) verileri analiz edildi. Hastaların demografik özellikleri, darlık yüzdeleri, kullanılan stent ve emboli koruma cihazlarının sayı ve çeşitleri ile periprosedural 30 günlük miyokard infarktüsü (Mİ), inme ve ölüm oranları araştırıldı. Uzun dönem sonuçlar, böyle bir değerlendirme için olguların sayısının yetersiz olması ve tedavi süreleri arasında homojen dağılım bulunmaması gerekçeleriyle çalışma dışı bırakıldı. **Bulgular:** Olgularımızda, ölüm oranı %0.9, minör inme oranı %4.7, inme ve ölüm oranı %5.7 idi. Transient iskemik atak (TİA), majör inme ve Mİ görülmedi. Semptomatik hastalar ile asemptomatik hastalar arasında anlamlı farklılık yoktu. **Sonuç:** KAS kabul edilebilir periprosedural komplikasyon oranlarıyla uygulanabilir. Günümüzde KEA'ya iyi bir alternatiftir. Gelecekte, KEA'dan ziyade KAS'tan fayda görecek hastaların belirlenmesi için yeni çalışmalara ihtiyaç bulunmaktadır.

Anahtar Kelimeler: Ateroskleroz; karotid arter hastalıkları; inme, felç; stentler

Turkiye Klinikleri J Cardiovasc Sci 2011;23(1):40-7

Carotid artery stenosis is a prevalent disease, caused predominantly by atherosclerosis. Other causes are rare and include fibromuscular dysplasia, trauma, and carotid dissection. The presence of carotid artery stenosis is associated with an increased risk of stroke and other ischemic manifestations of systemic atherosclerosis (e.g., myocardial infarctions and vascular deaths). The primary goal of revascularization of significant carotid artery stenosis is to prevent strokes.[1] Carotid artery stenting (CAS) is the alternative of carotid artery endarterectomy (CEA) for prevention of stroke.[2] In this study, we investigated the periprocedural neurologic complication rate on carotid artery stenting patients.

MATERIAL AND METHODS

PATIENT POPULATION

In this study, patients who underwent treatment due to atherosclerosis in the extracranial segments of the carotid artery system between June 2007 and April 2010, were included. The entire medical data on those patients were evaluated retrospectively. Patient's demographic variables and percentages of stenosis, the number and varieties of used stents and embolic protection devices with perioperative 30 days stroke, myocardial infarction (MI) and death rate were investigated. Long-term outcomes were excluded from the study because of the limited number of cases and lack of a homogeneous distribution among the treatment lengths. This study was performed as part of institutional review board approved clinical trials.

Medical management of cases indicating endovascular treatment and diagnosed as carotid stenosis, was performed by radiologist and neurologist physicians. Diagnostic radiological examinations of the patients were with Doppler ultrasonography (US), magnetic resonance (MR) angiography, computed tomography (CT) angiography, and digital subtraction angiography (DSA). Plaque morphologies in all the patients were also evaluated with Doppler US. All patients have provided signed informed consent and agreed to undergo regular follow-up evaluation. Endovascular treatment was performed on 105 arteries of 96 cases (Table 1).

Each case was treated with primary or secondary stenting, depending on the lesion type. Six-

TABLE 1: Distribution of the treated cases relative to lesions.

Vessel	Number of lesions
ICA	103 (98.2%)
Right (41)	41 (39.1%)
Left (46)	46 (43.9%)
Bilateral (8)	16 (15.2%)
*ECA (right)	1 (0.9%)
CCA (right)	1 (0.9%)

ICA: internal carotid artery, ECA: external carotid artery, CCA: common carotid artery
(*) Same-session treatment with the left ICA lesion.

teen (16.7%) of the cases were female, whereas 80 were male (83.3%). Age range was 41-84 years and mean age was 66±9 years. Fifty-nine of the cases (61.4%) were classified as symptomatic because the patients presented a transient ischemic attack or stroke, and 37 (38.6%) of the patients classified as asymptomatic. Demographic data of the patients, including the major risk factors for atherosclerosis, were shown in Table 2.

ENDOVASCULAR TECHNIQUE

Pre-procedural therapy consisted in the administration in the 5 days prior to the intervention of aspirin (300 mg/day) or clopidogrel (75 mg/day) in all cases. All treatments were performed in the angiography unit. There was an anesthesiologist in the treatment team. Electrocardiography (ECG), blood pressure, and oxygen saturation were monitored during the whole procedure. Procedures were performed under local anesthesia, except two cases. While brachial artery was used in one patient who was Lerich syndrome, femoral artery was used as access site in all other patients. A vascular sheath, matching the diameter of the stent and technique to be used, was inserted into the femoral artery. We performed arcus aorta, selective carotid, and selective cerebral angiography to patients who had not received diagnostic DSA examination previously, to investigate tandem lesions or additional pathologies. Percentage of the stenosis was measured by NASCET (North American Symptomatic Endarterectomy Trial) method on diagnostic angiograms (Table 3).

During the procedure, intra-arterial heparin (5000 IU) was administered to raise the activated clot-

TABLE 2: Demographic data.

Male	80 (83,3%)
Female	16 (16,7%)
Age (years)	41–84
Mean (years)	66±9
Symptomatic patient	59 (61,4%)
Major stroke	23 (38,9%)
Minor stroke	23 (38,9%)
TIA	13 (22%)
Asymptomatic patient	37 (38,6%)
Ipsilateral post-CEA restenosis	4 (4,2%)
Contralateral carotid occlusion	16 (16,6%)
Dislypidemia	27 (28,1%)
Diabetes mellitus	37 (38,5%)
Smoking	34 (35,4%)
Hypertension	64 (66,6%)
Peripheral vascular disease	13 (13,5%)
Coronary artery disease	43 (44,8%)
Acute myocardial infarction	7 (7,3%)

TIA: Transient ischemic attack, CEA: Carotid artery endarterectomy.

TABLE 3: Treated lesions.

Stenosis %	Number and percentage of lesions
90-99	41 (39)
70-89	57 (54.2)
50-69	7 (6.8)

ting time (ACT) above 250 second. Diagnostic catheter was replaced with the guiding catheter or guiding sheath through the guidewire with 0.035 inch diameter and proper length. We preferred 8F Envoy (Cordis Neurovascular Corporation, Florida, USA), March 1 (Boston Scientific, Massachusetts USA) guiding catheters and 90 cm 6-7 F Destination (Terumo Medical Corporation, Tokyo, Japan) guiding sheaths. The filter at the end of the 0.014 inch guidewire extended through the guiding catheter or sheath as a coaxial system, was released at the petrous segment of the internal carotid artery (ICA). Filter protection device was used in all cases, whereas in three cases the procedure was performed without protection.

To prevent bradycardia and hypotension during balloon inflation and stent deployment, prophylactic atropine (0.5-1 mg) administration was performed in all patients 1min before stent deployment. In cases with a percent stenosis ≥ 90%, predilatation was applied with a low-profile (3x20 mm) PTA balloons. The self-expanding stent was implanted after comprising the entire stenotic segment. In all cases, following stent implantation, postdilatation was performed with the PTA balloons (5x20 mm and 6x20 mm). For stenoses of ICA and CCA, 97 (92.3%) open-cell and 8 (7.7%) closed-cell self-expandable carotid stents were used. In one case with CCA stenosis was placed 2 stents due to the length of the stenotic segment. The stents we used were shown in Table 4.

ECA stenosis was treated in the same session with the contralateral ICA stenosis. Following the treatment of ICA stenoses, additional stent therapy was performed on the stenoses of subclavian artery in 1, renal artery in 1, and vertebral artery in 2 patients. In 2 different patients, ipsilateral parophtalmic and posterior communicating artery aneurisms were treated by endovascular method after the management of ICA stenoses.

In all cases, after stent deployment, a post-procedural angiography was performed to evaluate the eventual residual stenosis and the intracranial circulation. Following the procedure, all patients were prescribed Aspirin (100 mg/day) plus clopidogrel (75 mg/day) for the first 3-6 months and lifelong aspirin therapy were suggested.

DEFINITIONS

Periprocedural neurologic complications were defined based on the classification of Mathur et al. as follows;[3]

TIA: any neurological deficit that completely resolved within 24 h

Minor stroke: any new neurological deficit that either persisted after 24 h but completely resolved within 7 days

TABLE 4: Stents used in the treatment.

Stents	Cell type	Number	%
Protege (EV3, Minnesota, USA)	Open	87	82,8
Precise RX (Cordis, New Jersey, USA)	Open	10	9,5
Xact (Abbott Laboratories, Illinois, USA)	Closed	8	7,7

Major stroke: any new neurological deficit that persisted after 30 days

RESULTS

The case with preocclusive eksternal carotid artery (ECA) had an occluded ipsilateral ICA and a 70% stenotic contralateral ICA. This case has been complaining of transient vision loss which was ipsilateral to the ECA lesion, for the last two months showing daily recurrence. In the same session, both ECA and contralateral ICA stenoses were successfully treated (Figure 1). No recurrence was reported postoperatively.

A case who was treated in the same session due to bilateral ICA stenosis, occurred left hemiplegia within 30 minutes of the postprocedural period. Diffusion MRI showed small lesions consistent with acute infarction in the right parietal and occipital lobes. Another case who treated because of a right ICA lesion, occurred transient vision loss after therapy. Diffusion MRI was determined an acute infarction of 2x1 cm size in the right occipital lobe. Both patients were followed-up with heparin and support therapies. Following resolution of their complaints, our patients were discharged without any neurological deficit.

After procedure (in 30 days), 3 of our cases developed neurological complications (2 ipsilateral minor and 1 contralateral minor ischemic strokes). In one of these cases, the procedure was performed without protection, and additional stent therapy was performed on the renal artery in the same session.

No transient ischemic attack (TIA), major stroke, or MI was observed. In a patient who presented with acute ischemic stroke treated due to high-grade stenosis of the ICA, ipsilateral large cerebral hematoma communicating with the lateral ventricle occured 2 days after the procedure (Figure 2). This patient died 26 days after the procedure. Results of the periprocedural period were shown in Table 5.

DISCUSSION

Stroke is the third most common cause of death after cardiac diseases and cancer in western countries. According to the data of American Heart Association, 87% of all strokes are associated with ischemia, whereas 10% and 3% are due to intracerebral hemorrhage and subarachnoid hemorrhage, respectively.[4] Atherosclerosis, a systemic disease, is responsible for 90% of cerebral thromboembolic events in the industrialized nations.[5] 90% of the atherosclerotic lesions of carotid artery system are observed in a 2 cm segment comprising the origin of ICA and it is generally of unifocal character.[6] In cases where diagnosis of carotid stenosis is established, the degree of the stenosis bears great importance in choosing the proper therapeutic method. The superiority of CEA over medical therapy has been shown for symptomatic patients with a stenosis level ≥50% by NASCET and ECST (European Carotid Surgery Trial), and for

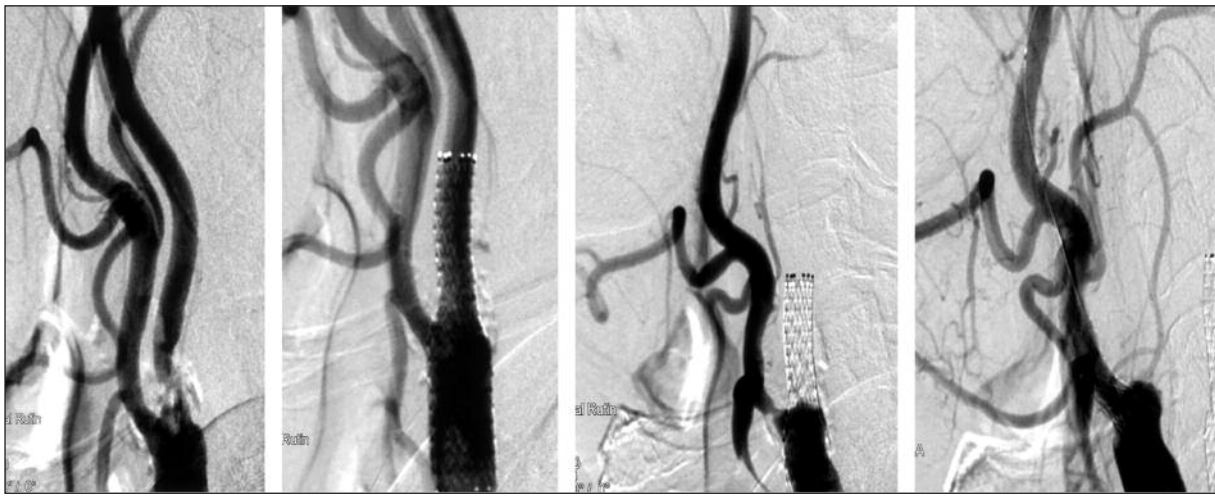

FIGURE 1: Periprocedural angiograms of the case who was treated for left ICA and contralateral ECA stenosis. A and B. Left ICA, before and after procedure. C and D. Right ECA, before and after procedure. Right ICA was occluded.

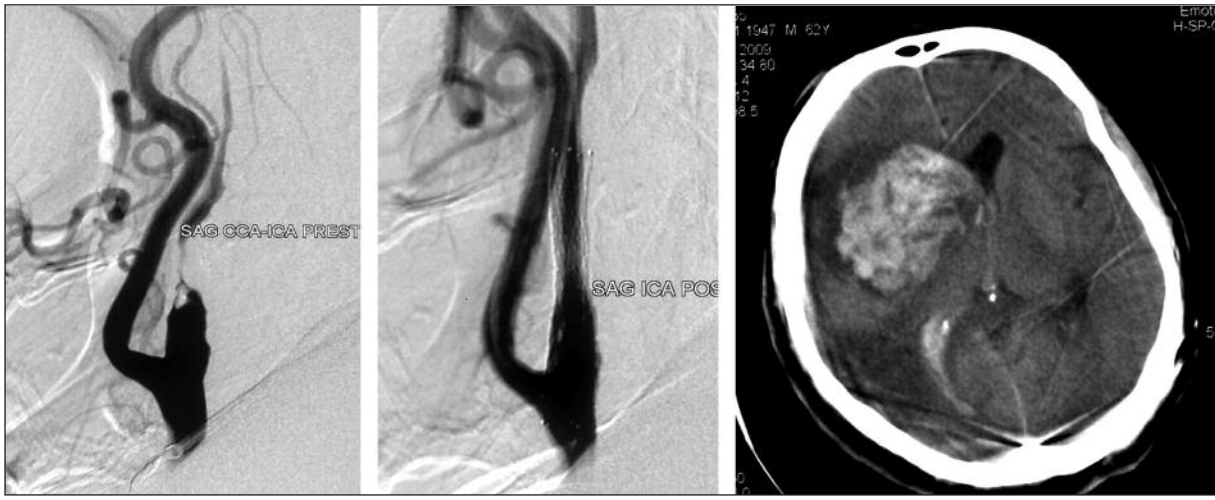

FIGURE 2: The case who occurred intracerebral hemorrhage after procedure A and B. Right ICA angiograms, before and after procedure. C. CT view of the large hematoma communicating with the ventricle in the right cerebral hemisphere.

TABLE 5: Periprocedural results.

Patients	Number of arteries	TIA	Minor stroke	Major stroke	Mortality	Any stroke and mortality
Asymptomatic	39 (37,2%)	-	2 (5,1%)	-	-	2 (5,1%)
Symptomatic	66 (62,8%)	-	3 (4,5%)	-	1 (1,5%)	4 (6%)
Total	105 (100%)	-	5 (4,7%)	-	1 (0,9%)	6 (5,7%)

asymptomatic patients with a stenosis level ≥ 60% by ACAS (Asymptomatic Carotid Atherosclerosis Study) and ACST (Asymtomatic Carotid Surgery Trial) studies.[7-12] CAS has been developed as an alternative for CEA. Compared to CEA, it has the following advantages: usually patients do not receive general anesthesia and therefore do not experience the associated complications, neck incision and the resultant possible cranial and cutaneous damage are avoided, length of recovery and hospital stay are shorter, and it is almost the only treatment option for patients with additional medical problems or cases under high risk for surgery. Presence of a risk for stroke and local complications are the disadvantages of the CAS.[13]

There are many randomized studies comparing those two treatment options. However, initial studies have some negative aspects such as inadequate length of time required for the accumulation of enough experience, technological handicaps of the early stents, and lack of routine usage of embolic protection devices.[14-16]

SAPPHIRE (Stenting and Angioplasty With Protection In Patients at High Risk for Endarterectomy) study, published in 2004, is the only randomized clinical trial in high-risk patients that compared contemporary CAS with embolic protection device against CEA.[17] Perioperative stroke, MI, or death rates were determined as 4.8% in the CAS group and 9.8% in the CEA group. At the end of a 1 year period, primary end-point rates including the perioperative results, were 12.1% for the CAS group and 20.1% for the CEA group. When MI was removed, there was no statistically significant difference between the two groups (5.5% vs. 8.4%). Authors concluded that CAS with embolic protection device was not an inferior technique compared with CEA. However, there was absence of a medical control in this study. Moreover, high surgical risk does not mean high risk for stroke while under medical therapy. Thus, further studies are needed to better define the patients under high risk and determine the patients that could benefit from CAS.

SPACE (Stent-Protected Percutaneous Angioplasty of the Carotid versus Endarterectomy) trial whose results were published in 2006, included 1183 symptomatic patients.[18] 30-day ipsilateral stroke or

death rates were 6.8% for the CAS arm and 6.3% for the CEA arm; there was no statistically significant difference between the groups. Another study which was published in 2006, EVA-3S (Endarterectomy Versus Angioplasty in Severe Symptomatic Carotid Stenosis), reported 30-day stroke and death rates as 9.6% for the CAS arm and 3.9% for the CEA arm.[19] This study was discontinued due to problems concerning reliability and disutility after enrollment of 527 patients. The results of the ICSS (International Carotid Stenting Study) were published in 2010.[20] The trial, which included 1713 symptomatic patients, found the 120-day stroke, mortality or procedural MI rate for CAS and CEA groups as 8.5% and 5.2%, respectively. Although authors underscored completion of long-term follow-up is needed to establish, they declared carotid endarterectomy should remain the treatment of choice for patients suitable for surgery. CREST (Carotid Revascularization Endarterectomy versus Stent Trial) study included 2502 patients (symptomatic/asymptomatic) and initial outcomes were published in 2010.[21] The outcomes for CAS and CEA groups were as follows, respectively: mortality 0.7% vs 0.3%, stroke 4.1% vs 2.3%, and MI 1.1% vs 2.3%. Authors reported high risk for stroke in CAS patients and for MI in CEA patients during the periprocedural period.

In our study, periprocedural any stroke and death rate was 5.7% in 105 interventions. None of our cases demonstrated TIA, major stroke, or MI. All the procedures, except the treatment applied on the ECA lesion and the therapies on ICA lesions in 2 different cases, were performed with filter-type embolic protection device. The case wasn't received no embolic protection device, developed contralateral minor stroke. Using no embolic protection device was thought to have no influence over this outcome because the lesion was located in the contralateral hemisphere and occurred during the postprocedural period. Distal microembolizations generally develop during stages of stent insertion or balloon inflation.[22] There is a correlation between the sizes of the embolic particles and incidences of neurologic deficit and cerebral infarction.[23] Embolic protection devices play an important role in prevention of distal embolization, however, since distal embolic protection devices should pass through the stenotic segment, inattentive use of them may lead to complications. The other causes of complications are as follows, respectively; poor adaptation of the device to the wall, filter-type devices failing to retain the small particles by their pores, slow-flow phenomenon, collateral embolization (eg. Ophtalmic artery), vasospasm, and vascular dissection.[24,25] Late embolic events may occur hours to days after CAS. These infrequent late events probably arise from detachment of atherosclerotic fragments protruding through stent struts. In soft and heterogeneous plaques, this risk is high. Closed-cell stents usage is a more suitable option for such lesions.[26]

Our case which ended with death due to intracranial hemorrhage, had been diagnosed as acute infarction prior to the procedure. Emergency revascularization was performed due to ipsilateral preocclusive carotid stenosis. The main therapeutic aim of emergency carotid stent placement is not removal of an ongoing embolic source, but restoration of blood flow to rescue the ischemic penumbra in the affected hemisphere.[27] Imai et al. showed that emergency stenting improved the 7-day neurological and 90-day clinical outcome in 17 selected patients with ipsilateral carotid occlusion or severe stenosis.[27] Emergency CAS, may cause hyperfusion syndrome, secondary embolization and hemorrhage in the infarction area.[28,29]

There was no significant difference betwen the symptomatic and asymptomatic patients in our periprocedural outcomes. Stroke and death rate was found to be 3.3% in 808 procedures by Reimers et al., 2.5% in 200 procedures by Lin et al., 2.9% in 698 patients by Reiter et al., 2.1% in 1.092 procedures by Simonetti et al., whereas Eskandari et al. found the TIA/stroke and death rate as 2.3% in 388 procedures.[30-34] Our outcomes were in agreement with literature data of the randomized studies, prospective studies and case series.

Major limitation of our study was the inadequate number of cases for evaluation of mid- and long-term outcomes. Mid- and long-term outcomes are needed in order to reveal the efficiency of CAS in treatment of carotid stenosis. Among other limitations of our study, we can mention single-center

feature, exclusion of causes of carotid artery disease other than atherosclerosis, and absence of a randomized investigation including surgery.

CONCLUSION

Periprocedural 30 days outcomes of stenting therapy for carotid stenoses are acceptable. CAS outcomes are not less efficient than the outcomes of CEA reported in the literature. By the use of embolic protection devices and advancing equipment technology, CAS will produce even better results in all patient groups. Further studies are required to determine patients that would benefit more from CAS rather than CEA in the future.

REFERENCES

1. Anthony FY, Saw J. Epidemiology and significance of carotid artery stenosis. In: Saw J, Exaire JE, Lee DS, Yadav JS, eds. Handbook of Complex Percutaneous Carotid Intervention. 2nd ed. New Jersey: Humana Press; 2007. p.3-11.
2. Ozturk V, Men S, Ugurel B, Goktay Y, Yaka E, Kaya D, et al. Management of carotid artery stenosis: experiences in Dokuz Eylül University Hospital. J Neurol Sci Turk 2008;25(1):25-31.
3. Mathur A, Roubin GS, Iyer SS, Piamsonboon C, Liu MW, Gomez CR, et al. Predictors of stroke complicating carotid artery stenting. Circulation 1998;97 (13):1239-45.
4. Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, et al. Heart disease and stroke statistics--2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009;119(3):e21-181.
5. Osborn AG. Atherosclerosis. Diagnostic Neuroradiology. 1st ed. St. Louis: Mosby; 1994. p.330-41.
6. Mohr JP, Gautier J. Internal carotid artery disease. In: Mohr JP, Choi DW, Grotta JC, Weir B, Wolf PA, eds. Stroke: Pathophysiology, Diagnosis, and Management. 4th ed. Philadelphia: Churchill Livingstone; 2004. p.75-100.
7. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1991;325(7):445-53.
8. Barnett HJ, Taylor DW, Eliasziw M, Fox AJ, Ferguson GG, Haynes RB, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1998;339(20):1415-25.
9. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. European Carotid Surgery Trialists' Collaborative Group. Lancet 1991;337(8752):1235-43.
10. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST). Lancet 1998;351(9113):1379-87.
11. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. JAMA 1995;273(18): 1421-8.
12. Halliday A, Mansfield A, Marro J, Peto C, Peto R, Potter J, et al. Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms: randomised controlled trial. Lancet 2004;363(9420): 1491-502.
13. Touzé E, Calvet D, Chatellier G, Mas JL. Carotid stenting. Curr Opin Neurol 2008;21(1):56-63.
14. Naylor AR, Bolia A, Abbott RJ, Pye IF, Smith J, Lennard N, et al. Randomized study of carotid angioplasty and stenting versus carotid endarterectomy: a stopped trial. J Vasc Surg 1998;28(2): 326-34.
15. Brooks WH, McClure RR, Jones MR, Coleman TL, Breathitt L. Carotid angioplasty and stenting versus carotid endarterectomy for treatment of asymptomatic carotid stenosis: a randomized trial in a community hospital. Neurosurgery 2004;54(2):318-24.
16. Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial. Lancet 2001;357(9270):1729-37.
17. Yadav JS, Wholey MH, Kuntz RE, Fayad P, Katzen BT, Mishkel GJ, et al.; Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy Investigators. Protected carotid-artery stenting versus endarterectomy in high-risk patients. N Engl J Med 2004;351(15):1493-501.
18. Ringleb PA, Allenberg J, Brückmann H, Eckstein HH, Fraedrich G, Hartmann M, et al.; SPACE Collaborative Group. 30 day results from the SPACE trial of stent-protected angioplasty versus carotid endarterectomy in symptomatic patients: a randomised non-inferiority trial. Lancet 2006;368(9543):1239-47.
19. Mas JL, Chatellier G, Beyssen B, Branchereau A, Moulin T, Becquemin JP, et al.; EVA-3S Investigators. Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Engl J Med 2006;355(16):1660-71.
20. Ederle J, Dobson J, Featherstone RL, Bonati LH, van der Worp HB, de Borst GJ, et al.; International Carotid Stenting Study investigators. Carotid artery stenting compared with endarterectomy in patients with symptomatic carotid stenosis (International Carotid Stenting Study): an interim analysis of a randomised controlled trial. Lancet 2010;375(9719):985-97.
21. Brott TG, Hobson RW 2nd, Howard G, Roubin GS, Clark WM, Brooks W, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med 2010;363(1):11-23.
22. Al-Mubarak N, Roubin GS, Vitek JJ, Iyer SS, New G, Leon MB. Effect of the distal-balloon protection system on microembolization during carotid stenting. Circulation 2001;104(17):1999-2002.
23. Tübler T, Schlüter M, Dirsch O, Sievert H, Bösenberg I, Grube E, et al. Balloon-protected carotid artery stenting: relationship of periprocedural neurological complications with the size of particulate debris. Circulation 2001;104(23):2791-6.
24. Atkins MD, Bush RL. Embolic protection devices for carotid artery stenting: have they made a significant difference in outcomes? Semin Vasc Surg 2007;20(4):244-51.
25. Casserly IP, Abou-Chebl A, Fathi RB, Lee DS, Saw J, Exaire JE, et al. Slow-flow phenomenon during carotid artery intervention with embolic protection devices: predictors and clinical outcome. J Am Coll Cardiol 2005;46(8):1466-72.
26. Henry M, Polydorou A, Henry I, Polydorou Ad, Hugel M. Carotid angioplasty and stenting under protection: techniques, indications, results, and limitations. In: Heuser RR, Henry M, eds. Textbook of Peripheral Vascular Interventions. 2nd ed. London: Informa Healthcare; 2008. p.300-35.

27. Imai K, Mori T, Izumoto H, Watanabe M, Majima K. Emergency carotid artery stent placement in patients with acute ischemic stroke. AJNR Am J Neuroradiol 2005;26(5):1249-58.
28. Jovin TG, Gupta R, Uchino K, Jungreis CA, Wechsler LR, Hammer MD, et al. Emergent stenting of extracranial internal carotid artery occlusion in acute stroke has a high revascularization rate. Stroke 2005;36(11):2426-30.
29. Geisler BS, Röther J, Kucinski T, Zeumer H, Eckert B. Emergency carotid artery stenting in persistent hemodynamic deficit associated with severe carotid stenosis. AJNR Am J Neuroradiol 2005;26(3):549-52.
30. Reimers B, Schlüter M, Castriota F, Tübler T, Corvaja N, Cernetti C, et al. Routine use of cerebral protection during carotid artery stenting: results of a multicenter registry of 753 patients. Am J Med 2004;116(4):217-22.
31. Lin PH, Bush RL, Peden EK, Zhou W, Guerrero M, Henao EA, et al. Carotid artery stenting with neuroprotection: assessing the learning curve and treatment outcome. Am J Surg 2005;190(6):850-7.
32. Reiter M, Bucek RA, Effenberger I, Boltuch J, Lang W, Ahmadi R, et al. Plaque echolucency is not associated with the risk of stroke in carotid stenting. Stroke 2006;37(9):2378-80.
33. Simonetti G, Gandini R, Versaci F, Pampana E, Fabiano S, Stefanini M, et al. Carotid artery stenting: findings based on 8 years' experience. Radiol Med 2009;114(1):95-110.
34. Eskandari MK, Usman AA, Garcia-Toca M, Matsumura JS, Kibbe MR, Morasch MD, et al. Eight-year institutional review of carotid artery stenting. J Vasc Surg 2010;51(5):1145-51.