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RESEARCH ARTICLE

Neurological Outcome of Stenting in Carotid Occlusive Diseases.

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Abstract

Background: Carotid artery stenting (CAS)has become widely available and considered as an alternative to carotid endartectomy.
Objective: To compare CAS under protection device with medical treatment.
Material and method: fifty patients were enrolled in these study (30 patients treated with carotid stenting and 20 patients managed medically) with stenosis > 60%.with follow up at six months later clinically and radiologically using carotid duplex.
Result: After 6 months 4(13.4%) patients in CAS group develop TIA while in medically treated group 6(30%) patients, 1(3.3%) patient in CAS group develop stroke which was after procedure while 6(30%) patients in medically treated group develop stroke during follow up within 6 months, 1(3.3%) patient die in CAS group due to myocardial infarction, while in medically treated group 2(10%) patients died one of them due to large stroke and the other due to myocardial infarction. There was high significant difference between CAS and medical treated group after 6 months ($p<0.01$).
Conclusion: CAS under protection device is associated with better outcome than medical treatment.

Introduction:-

Stroke is estimated to be responsible for 5.5 million deaths worldwide annually. It is now the leading cause of disability in developed countries. Of those affected by first stroke approximately one third die, one third recover and one third are disabled (1).

Stroke risk increase with stenosis severity and plaque morphology. The aim of the treatment of carotid artery stenosis is to prevent neurological complications; this could be achieved by medical treatment, surgical or endovascular interventions(2).

Complications related to CEA which either early or late and Some them are devastating complications that involve the neurological system. These tend to occur early, within the first 30 days after surgery. (3).

Since the inception of carotid angioplasty over two decades ago, techniques and equipment for carotid artery stenting (CAS) have radically improved. Equipment with lower profile (e.g., smaller outer diameter sheaths with large inner lumen, 0.014" system balloon catheters and stent catheters) and targeted to carotid arteries (e.g., emboli protection devices, self-expanding stents) have evolved dramatically, leading to improved technical success and procedural safety of CAS especially for high risk surgical patients with low morbidity and mortality. (4).

MATERIALS AND METHODS:-

This prospective study was conducted on fifty patients with carotid stenosis more than 50% between 2011 and 2015 after obtaining a written informed consent from all patients.Thirty patient treated with carotid stenting and twenty patients were managed medically.**Exclusion criteria**includemajor functional impairment (modified Rankin Scale

≥3),Significant cognitive impairment,Major stroke within 4 weeks (new neurological event that persisted after7 days and increased NIH stroke scale score by ≥4,Contraindication to acetylsalicylic or dual antiplatelet treatment for 4 weeks., Inability to achieve safe vascular access,Intracranial aneurysm > 2mm or AVM requiring treatment,Concentric heavy lesion calcification,Visible thrombus in lesion ,Total occlusion and Long subtotal occlusion (string sign).

All patients were subjected to complete history taking,bed side examination including general examinations , full neurological examinations scales (NIHSS) scale(5) and modified Rankin Scale(6) at 4 points (Before CAS, within 3days after the procedure, one month, 6 months after the procedure, and when necessary).
Also carotid duplex ±MRA or CTA (neck vessels) at day zero and after 6 monthsand laboratory investigations (complete blood count, PT, PTT, liver and renal function tests, random blood sugar, lipid profile and serum uric acid), ECG and trans-thoracic echocardiography.

CAS Procedure:-
All patients will be treated with acetyl-salicylic acid (150 mg/d) associated with clopidogrel (75 mg/d) for one week or 300 mg clopidogrel at least 24 h before the procedure.

The procedures were performed with local anesthesia then percutaneous transfemoral access F8 with heparin to achieve an activated clotting time (ACT) longer than 200–250 sec. And atropine (0.51 mg IV) will be given in order to reduce the bradycardia and hypotension potentially associated with carotid dilation.

All patients will undergo an angiographic examination of the culprit carotid lesion in at least two different real-time projections and an angiographic examination of intracranial circulation in antero-posterior and lateral projection.

The guidewire will be used to cross the carotid-artery stenosis first, and then the filter will be expanded before the stent deployment.

Carotid stenting will be carried out by using self-expandable crush resistant stents (Smart Precise - Cordis or Carotid Wallstent -bostonscientific).

The pre-dilation balloons will be routinely undersized (artery: balloon ratio = 2:1.5) in order to reduce vessel dissection and/or distal embolization.

Stent placement will be optimized through single or multiple postdilatations by using suitably sized balloons based on angiographic quantitative analysis of the vessel.

Procedural success was defined as; to achieve a less than 30% residual diameter stenosis of the treated lesion in at least two matched views on angiography and finally absence of distal embolization.

STATISTICAL ANALYSIS:-
Data was analyzed on an IBM personal computer, using Statistical Package for Special Science (SPSS) software computer program version 15. Then data described as mean ± standard deviation (SD) for quantitative (Numerical) variables and as frequency and percentage for qualitative (Categorical) variables. Chi-square test (or Fisher's exact test when appropriate) used for comparison of distribution of qualitative variables among different group.

Results:-
The study was conducted between 2011 and 2015 on 50 patients with (mean age± standard deviation) (66±7.92), 27(54%) of them were male 23(46%) were female.(table 1).stenosis was in 17(56.6%) male patients in CAS while female 13 (43.4%) patients. In medically treated group 10 (50%) patients were male and 10(50%) were female .with no statically difference between male and female (p>0.05).(table 2).All the patient were symptomatic but 12(24%) patients were presented with TIA, 38(76%) presented with stroke. Left carotid stenosis in 20(40%) patients and right carotid stenosis in 29(58%) patients with (mean stenosis ± standard deviation) (74.7±10.517).(table 3).

As regard NIHSS at presentation , 15(50%) patients in CAS group were mild stroke (NIHSS <5), while in medical treated group 11(55%) patients, 15(50%) patients were moderate stroke (NIHSS 5-15) in CAS group and 9(45%)

patients in medically treated group and no sever stroke (NIHSS >15) in both groups.With no statically difference between CAS and medical treated group (p>0.05).(table 4)

According to disability as regard MRS at presentation in each group, MRS was 0 in 8(26.6%) patients in CAS while 4 (20%) patients in medically treated group.1-2 in 22 (73.4%) patients in CAS and 116(60%) patients in Medically treated group ,with no statically difference between CAS and Medical treated group (p>0.05).(table 5)

Outcome and correlative analysis:-

Among 30 patients, stenting of ICA bifurcation was done under general anesthesia in 2 cases (6%) and other cases under local anesthesia, stenting was done using trans-femoral approach in 29 cases (96.7 %) and one case (3.3%) through brachial approach due to occlusion of the abdominal aorta.

Prestenting balloon dilatation of lesion used in 5 (16.6%) patients, and 25 (83.4%) patients without prestenting balloon dilatation.poststenting balloon dilatation of the lesion done in 9 (30%) patients, and 21 (70%) patients with no poststenting balloon dilatation.

The mean stenosis before the procedure is 74.7, procedural success rate was 100 % with mean residual stenosis 17.7 %. One patient (3.3%) developed ipsilateral stroke with National Institutes of Health Stroke Scale (NIHSS score = 6), three(10%) patients developed TIA immediately after stenting and symptoms resolve within 3 hours, the periprocedural vascular complications increased with increased degree of stenosis .(table 6)

After 6 months 4(13.4%) patients in CAS group develop TIA while in medical treated group 6(30%) patients, 1(3.3%) patient in CAS group develop stroke which was after procedure while 6(30%) patients in medical treated group develop stroke during follow up within 6 months, 1(3.3%) patient die in CAS group due to myocardial infarction, while in medical treated group 2(10%) patients died one of them due to large stroke and the other due to myocardial infarction. There was high significant difference between CAS and medical treated group after 6 months (p<0.01).(table 7).

As regard NIHSS after 6 months , 14(46.7%) patients in CAS group were mild stroke (NIHSS <5), while in medical treated group 9(45%) patients, 15(50%) patients were moderate stroke (NIHSS 5-15) in CAS group and 9(45%) patients in medically treated group and sever stroke (NIHSS >15) were 1(3.3%) in CAS group while 2(10%) patients in medical treated group.With no statically difference between CAS and medical treated group (p>0.05).(table 8).

According to disability as regard MRS after 6 months in each group, MRS was 0 in 8(26.6%) patients in CAS while 2(10%) patients in medically treated group.MRS 1-2 in 21 (70%) patients in CAS and 10(50%) patients in Medically treated group ,MRS 3 in 2(10%) patients in medically treated group and no patients in CAS group.MRS 4 in 3(15%) patients in medically treated group, no patients in CAS group, MRS 5 in 1 patient in medically treated group, MRS 6 ion 1(3.3%)patient in CAS group and in 2(10%) patients in medically treated group. With statically difference between CAS and Medical treated group (p<0.05).(table 9).

Table 1:- Age and sex distribution :

	No	**%**
Sex		
Male	27	54%
Female	23	46%
Age:- Mean±SD	66.24±7.92	
Range	46-82	

Table 2:- Sex distribution within groups:

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
Sex	Male	17	56.6	10	50	27	54	>.05
	Female	13	43.4	10	50	23	46	
Total		30	60	20	40	50	100	

Table 3:- Presentation and site of stenosis :

	No	**%**
Symptoms		
Stroke	38	76%
TIA	12	24%
Site		
Right	29	58%
Left	20	20%
Bilateral	1	2
Stenosis		
Mean±SD	(74.7±10.517)	

Table 4:- Clinical presentation as regard NIHSS:

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
NIHSS	Mild	15	50	11	55	26	52	>.05
	Mod	15	50	9	45	24	48	
	Sever	0	0	0	0	0	0	
Total		30	60	20	40	50	100	

Table 5:- Disability as regard MRS at presentaion:

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
MRS	0	8	26.6	4	20	12	24	>.05
	1-2	22	73.4	16	80	38	76	
	3	0	0	0	0	0	0	
Total		30	60	20	40	50	100	

Table 6:- Procedural Results and Complications :

Procedural Results and Complications		
	No	%
Transient bradycardia	2	6.6%
Stroke	1	3.3%
TIA	3	10%

Table 7:- Follow up after 6 months

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
Follow up after 6 months	TIA	4	13.4	6	30	10	20	<.01
	Stroke	1	3.3	6	30	7	14	
	Death	1	3.3	2	10	3	6	
	No new event	24	80	6	30	30	60	
Total		30	60	20	40	50	100	

Table 8:- Clinical presentation as regard NIHSS after 6 months:

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
NIHSS	Mild	14	46.7	9	45	23	46	>.05
	Moderate	15	50	9	45	24	48	
	Sever	1	3.3	2	10	3	6	
Total		30	60	20	40	50	100	

Table 9:- Disability by MRS after 6 months:

		Carotid Stenosis				Total		
		CAS		Med treatment				
		No	%	No	%	No	%	P
MRS	0	8	26.7	2	10	10	20	<.05
	1-2	21	70	10	50	31	62	
	3	0	0	2	10	2	4	
	4	0	0	3	15	3	6	
	5	0	0	1	5	1	2	
	6	1	3.3	2	10	3	6	
Total		30	60	20	40	50	100	

Discussion:-

Atherosclerotic disease of the carotid artery is responsible for 20%–30% of all strokes **(7)** and significant clinically relevant stenosis >50% was detected in(2.5%) of the atherosclerotic symptomatic subjects**(8).**

Patient selection to determine the most appropriate means of carotid stenosis management, whether CAS, surgical CEA, or lone pharmacologic management, is a complex one and decision regarding the optimal mode of revascularization (CEA or CAS) must be determined on an individual patient by patient basis. **(9).**

The aim of this study is to assess the value and consequences of extracranial carotid artery stenting immediately and at short term follow-up using clinical assessment and carotid Doppler ultrasonography.

In the present study the mean age of patients is (66±7.92 years) which is younger than most studies as **(SAPPHIRE)**Stenting and angioplasty with protection in patients at high risk for endarterectomy population(**10)**, **(EVA-3S)**Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis (**11)**, **(CAVATAS)**Carotid and Vertebral Artery Transluminal Angioplasty Study**(12)**, **(SPACE)**Stent Protected Angioplasty versus Carotid Endarterectomy (**13)** and the study done by **Naylor et al., 1998(14)** in them the mean age by years are 72.6, 69.7,67, 67.9 and 71 respectively.

The presence of patients with younger age in the current study may be explained by the difference in vascular risk factors between the current study and the other studies, the frequency of diabetes mellitus is more in this study than that in other studies, the frequency of other risk factors as hypercholesterolemia, hypertension, ischemic heart disease and smoking is equal or slightly higher than other studies**.**This leads to early atherosclerosis and subsequent early occurrence of carotid stenosis in young age (**15).**

In this study the frequency of carotid stenosis is more common in male than female 54% versus 46% ,this finding is congruent with **SAPPHIRE**(**10)**study in whichthe prevalence of male is more than female (62%)**(16) .De Weerd et al .,2009** (17)found that Prevalence of moderate stenosis increases with age in both men and women, but men at all ages have the higher prevalence estimates.

Sex difference in carotid stenosis is now widely accepted. Sex hormones seem to play a fundamental protective role in women through widespread actions, affecting endothelial function, lipid homeostasis, and cardiovascular risk factor reduction (**18).** Moreover, some authors claim that estrogens might have plaque stabilization properties and effects on inflammatory status (**19).**

In the present study the occurrence of the periprocedural vascular complications (stroke and TIA) is correlated with the degree of stenosis, this is consistent with **Mathur and his colleagues (20)**who found that CAS performed in lesions with angiographic severity > 90% stenosis were associated with higher 30-day stroke rate of 14.9% compared with lower rate of 3.5% in patients with lesion severity < 90% stenosis, however other studies have found no difference in the mean severity of stenosis [50%–69% versus 70%–99%].(**21).**

The cause of difference may be explained by the difference in pathology of the plaque more than the degree of the stenosis as the cases in the current study are symptomatic which characterized by presence of plaques with fissures, intramural microthrombi or inflammation so stenotic lesions are more vulnerable to vascular complications(**22).**
The results of this study showed 0% restenosis rate after 6 months follow up with 0% ipsilateral strokes due to restenosis. These results compare well with results of **Powell et al (2004)(23)** who studied 74 patients for 6 months

follow-up and reported 2.7% restenosis rate with 0% stroke. **Cernetti et al (2003)**(24)studied 104 patients for 31 months follow-up and reported 3.9% restenosis rate with 0% stroke. **Mudra et al (2003)(25)** studied 100 patients for 10 months follow-up and reported 2.4% restenosis rate with 0% stroke. **Henry et al (2002)(26)** who studied 180 patients for 6 months follow- up and reported 0.6% restenosis rate with 0% stroke. **Gupta et al (2000)(27)** studied 105 patients for 18 months follow-up and reported 1.9% restenosis rate with 0% stroke. **Gross et al (1999)(28)** studied 89 patients for 13 months follow-up and reported 3.7% restenosis rate with 0% stroke. Also the results of the largest multicenter survey to date by **Wholey et al (2003) (29)** included over 11243 patients reported 2.7% restenosis rates after 12 months, 2.6% after 24 months and 2.4% after 36 months, the rate of neurologic events after stent placement was 1.2%, 1.3%, and 1.7% at 1, 2, and 3 years, respectively. All these studies used the ≥50% restenosis threshold.

Contrary to results of the present study higher restenosis rates were reported by different studies. **Schillinger et al (2004)(30)** studied 100 patients for 23 months follow-up and reported 14% restenosis rate with 2% stroke. **Bowser et al (2003)(31)** studied 52 patients for 26 months follow-up and reported 15.4% restenosis rate with 0% stroke. **Bonaldi et al (2002)**(32)who studied 50 patients for 12 months follow-up and reported 8% restenosis rate with 0% stroke. **Chakhtoura et al (2001)(33)** who studied 49 patients for 18 months follow-up and reported 12.2% restenosis rate with 0% stroke. **New et al (2001)(34)** who studied 319 patients for 14 months follow-up and reported 7.2% restenosis rate with 0.6% stroke. **Groschel et al (2005)(35)** conducted a systematic analysis of all peer-reviewed studies reporting on the rate of restenosis (≥50%) after carotid artery stenting based on duplex ultrasound or angiography that were published between January 1990 and July 2004. They identified 34 studies that reported on a total of 4185 patients with a follow-up of 3814 arteries over a median of 13 months (range, 6 to 31 months). The cumulative restenosis rates after 1 and 2 years were ≈6% and 7.5% in those studies, which used a lower restenosis threshold ≥50% to 70%.

This difference can be explained by that all cases included in the current study carotid stenosis due to atherosclerosis while other studies carotid in-stent restenosis occurs [mostly in non- atherosclerotic carotid stenosis lesions (radiation induced carotid stenosis and post-endarterectomy restenosis) which have more liability to restenosis.

According to results of the present study and that of previous studies the short-term restenosis rates after CAS is promising, thus there is a need for further research on the long-term durability of CAS procedures which showed a higher restenosis rates.

Finally, there still is the need for identifying specific risk factors for the development of restenosis after CAS. Some studies have identified advanced age **(36)**, female gender **(36)**, hyperglycemia **(37),** previous treatment with a CEA **(38)**, and increased serum levels of acute-phase reactants **(39)** as potential risk factors for the development of a restenosis after CAS; however, the definitive role of these factors remains to be elucidated in larger trials.Also, CAS under protection device is associated with better outcome than medical treatment and it appears to be a safe procedure.

Conclusion:-

Patient selection to determine the most appropriate means of carotid stenosis management, whether CAS, surgical CEA, or lone pharmacologic management, is a complex one and decision regarding the optimal mode of revascularization (CEA or CAS) must be determined on an individual patient by patient basis.also low rate of neurological complications in multiple randomized trials and registries, should encourage us to keep working in this field with good training and to increase the learning curve**.**

References :-

1. The global burden of disease: 2004 update. Geneva, World Health Organization, 2008 .
2. **Liapis CD, Avgerinos ED, Chatziioannou A:** The Aortic Arch: Markers, Imaging, and Procedure Planning for Carotid Intervention, Vascular Disease Management,2009; Jan/Feb, Volume 6, Available from URL: http://www .vascular disease management.com.
3. Bailes JE, Medary MB. Carotid endarterectomy. In: Winn HR, ed. Youmans Neurological Surgery, 5th Ed. Saunders, Philadelphia; 2004, pp 1621–1649.
4. **Saw J, Walsh S:** Aortic Arch and Cerebrovascular Anatomy and Angiography. Carotid artery stenting :The Basics, part 2009;4,10:149-169.
5. Www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.
6. Bonita R, Beaglehole R. "Modification of Rankin Scale: Recovery of motor function after stroke."Stroke 1988 Dec;19(12):1497-1500.
7. Timsit SG, Sacco RL, Mohr JP, Foulkes MA, Tatemichi TK, Wolf PA, Price TR, Hier DB: Early clinical differentiation of cerebral infarction from severe atherosclerotic stenosis and cardioembolism. Stroke 1992;23:486– 491.
8. Foad Abd Allah ,Essam Baligh and Magdy Ibrahim:Clinical Relevance of Carotid Atherosclerosis among Egyptians: A 5-Year Retrospective Analysis of 4,733 Subjects. Neuroepidemiology 2010;35:275–279.
9. Yadav JS, Wholey MH and Kuntz RE; Protected carotid-artery stenting versus endarterectomy in high-risk patients. N Engl J Med2004; 351:1493–501.
10. Jacqueline Saw, Davis S. Lee. Indications for carotid artery stenting. IN: Handbook of complex percutaneous carotid intervention. New Jersy: Humana Press, 2007; 111-127.
11. Mas JL, Trinquart L, Leys D, Albucher JF, Rousseau H, Viguier A, Bossavy JP, Denis B, Piquet P, Garnier P, Viader F, Touzé E, Julia P, Giroud M, Krause D, Hosseini H, Becquemin JP, Hinzelin G, Houdart E, Hénon H, Neau JP, Bracard S, Onnient Y, Padovani R,. Endarterectomy Versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) trial: results up to 4 years from a randomised, multicentre trial.. Lancet Neurol 2008;7:885-92.
12. Brahmanandam S, Ding EL, Michael S,: Clinical results of carotid artery stenting compared with carotid endarterectomy J Vasc Surg 2008:47:343-9.
13. Ringleb PA, Allenberg J, Brückmann H, Eckstein HH, Fraedrich G, Hartmann M, Hennerici M, Jansen O, Klein G, Kunze A, Marx P, Niederkorn K, Schmiedt W, Solymosi L, Stingele R, Zeumer H, Hacke W. 30 day results from the SPACE trial of stent-protected angioplasty versus carotid endarterectomy in symptomatic patients: a randomised non-inferiority trial.. Lancet 2006;368:1239-47.
14. Naylor AR, Bolia A, Abbott RJ,: Randomized study of carotid angioplasty and stenting versus carotid endarterectomy: a stopped trial. J Vasc Surg 1998; 28:326 34.
15. J¨arvisalo MJ, Raitakari M, Toikka JO,: Endothelial dysfunction and increasedarterial intima-media thickness in children with type 1 diabetes. Circulation2004;109: 1750–1755.
16. Massop D, Dave R, Metzger C, Bachinsky W, Solis M, Shah R, Schultz G, Schreiber T, Ashchi M, Hibbard R; SAPPHIRE Worldwide Investigators. Stenting and angioplasty with protection in patients at high-risk for endarterectomy: SAPPHIRE Worldwide Registry first 2,001 patients.Catheter Cardiovasc Interv. 2009 Feb 1;73(2):129-36.
17. De Weerd M1, Greving JP, de Jong AW, Buskens E, Bots ML :Prevalence of asymptomatic carotid artery stenosis according to age and sex: systematic review and metaregression analysis.Stroke. 2009 Apr;40(4):1105-13.
18. Agriniern., M. Cournot, J. Dallongeville, D. Arveiler, P. Ducimetière, J.B. Ruidavets, et al.Menopause and modifiable coronary heart disease risk factors: a population based studymaturitas, 65 (2010), pp. 237–243.
19. Iemolof., A. Martiniuk, D.A. Steinman, J.D. spencesex differences in carotid plaque and stenosisstroke, 35 (2004), pp. 477–481.
20. Mathur A, Roubin GS, Iyer SS,: Predictors of stroke complicating carotid artery stenting. Circulation1998 : 97(13):1239–45.
21. Chaturvedi S, Matsumura JS, Gray W,; Carotid artery stenting in octogenarians: periprocedural stroke risk predictor analysis from the multicenter Carotid ACCULINK/ACCUNET Post Approval Trial to Uncover Rare Events (CAPTURE 2) clinical trial. Stroke2010: 41(4):757–64.
22. Gray WA, Yadav JS, Verta P,: The CAPTURE registry: predictors of outcomes in carotid artery stenting with embolic protection for high surgical risk patients in the early post-approval setting. Catheter Cardiovasc Interv2007: 70(7):1025–33.

23. Powell RJ, Schermerhorn M, Nolan B: Early results of carotid stent placement for treatment of extracranial carotid bifurcation occlusive disease. J Vasc Surg 2004. 39:1193–1199.
24. Cernetti C, Reimers B, Picciolo A,: Carotid artery stenting with cerebral protection in 100 con- secutive patients: immediate and two-year follow-up results. Ital Heart J 2003:4:695,.
25. Mudra H, Ziegler M, Haufe MC, Hug M, Knape A, Meurer A, Pitzl H, Büchele W, Spes C.Percutaneous carotid angioplasty with stent implantation and protection device against embolism--a prospective study of 100 consecutive cases. Dtsch Med Wochenschr. 2003 Apr 11;128(15):790-6. German.
26. Henry M, Henry I, Klonaris C,: Benefits of cerebral protection during carotid stenting with the Percu-Surge Guard Wire system: midterm results. Jendovasc Ther2002;9:1–13.
27. Gupta A, Bhatia A, Ahuja A, Shalev Y, Bajwa T.Carotid stenting in patients older than 65 years with inoperable carotid artery disease: a single-center experience.Catheter Cardiovasc Interv. 2000 May;50(1):1-8; discussion 9.
28. Gross CM, Krämer J, Uhlich F, Tamaschke C, Vogel P, Friedrich I, Dietz R, Waigand J.Treatment of carotid artery stenosis by elective stent placement instead of carotid endarterectomy in patients with severe coronary artery disease.Thromb Haemost. 1999 Sep;82 Suppl 1:176-80.
29. Wholey MH, Al-Mubarek N: Updated review of the global carotid artery stent registry. Catheter Cardiovasc Interv;2003 60:259–266.
30. Schillinger M, Exner M, Sabeti S, Amighi J, Wagner O, Ahmadi R, Minar E.Excessive carotid in-stent neointimal formation predicts late cardiovascular events.J Endovasc Ther. 2004 Jun;11(3):229-39.
31. Bowser AN, Bandyk DF, Evans A, Novotney M, Leo F, Back MR, Johnson BL, Shames ML.Outcome of carotid stent-assisted angioplasty versus open surgical repair of recurrent carotid stenosis.J Vasc Surg. 2003 Sep;38(3):432-8.
32. Bonaldi G. Angioplasty and stenting of the cervical carotid bifurcation: report of a 4-year series.Neuroradiology. 2002 Feb;44(2):164-74.
33. Chakhtoura EY, Hobson RW 2nd, Goldstein J, Simonian GT, Lal BK, Haser PB, Silva MB Jr, Padberg FT Jr, Pappas PJ, Jamil Z.In-stent restenosis after carotid angioplasty-stenting: incidence and management.J Vasc Surg. 2001 Feb;33(2):220-5; discussion 225-6.
34. New G,Roubin GS, Iyer SS,: mmediate and late clinical outcomes of carotid artery stenting in patients with symptomatic and asymptomatic carotid artery stenosis: a 5-year prospective analysis. Circulation2001; 103(4):532–7.
35. Gröschel K, Riecker A, Schulz JB, Ernemann U, Kastrup A.Systematic review of early recurrent stenosis after carotid angioplasty and stenting.Stroke. 2005 Feb;36(2):367-73.
36. Khan MA, Liu MW, Chio FL, Roubin GS, Iyer SS, Vitek JJ.Predictors of restenosis after successful carotid artery stenting. Am J Cardiol. 2003 Oct 1;92(7):895-7.
37. Willfort-Ehringer A, Ahmadi R, Gessl A, Gschwandtner ME, Haumer A, Lang W, Minar E, Zehetmayer S, Ehringer H.Neointimal proliferation within carotid stents is more pronounced in diabetic patients with initial poor glycaemic state.Diabetologia. 2004 Mar;47(3):400-6.
38. Setacci C, Pula G, Baldi I, de Donato G, Setacci F, Cappelli A, Pieraccini M, Cremonesi A, Castriota F, Neri E.Determinants of in-stent restenosis after carotid angioplasty: a case-control study. J Endovasc Ther. 2003 Dec;10(6):1031-8.
39. Schillinger M, Exner M, Mlekusch W, Rumpold H, Ahmadi R, Sabeti S, Lang W, Wagner O, Minar E.Acute-phase response after stent implantation in the carotid artery: association with 6-month in-stent restenosis.Radiology. 2003 May;227(2):516-21.