Original articles

Endovascular treatment of carotid atherosclerotic disease: early and late outcome in a non-selected population

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Key words:
Carotid angioplasty; Stents.

Background. **Carotid angioplasty and stenting are still not widely accepted treatments for carotid stenosis. This registry was aimed at investigating the efficacy of endovascular therapy of the extracranial carotid arteries in a non-selected population.**

Methods. **One hundred nineteen consecutive patients (93 males and 26 females, mean age 70.4 ± 7.2 years) were enrolled to undergo percutaneous angioplasty and/or stenting of the extracranial carotid artery. The primary endpoint of this study was to evaluate the feasibility, safety and efficacy of carotid elective angioplasty and stenting in a "real life group" of patients (> 50 years, without strict target vessel selection or inclusion criteria) with carotid occlusive disease. During a 6-12 month follow-up period late major adverse events were evaluated, either related to the endovascular treatment or due to other pathological conditions.**

Results. **Percutaneous procedure was effective in 118/119 patients (99.16%). Procedural success rate was 99.16%, in-hospital major neurological symptomatic complications 0%, in-hospital minor neurological symptomatic complications 3.36% (two minor strokes, one transient ischemic attack and one subarachnoid hemorrhage). The follow-up observation time ranged from 6 to 36 months following the percutaneous procedure. Overall follow-up data showed absence of late major adverse events related to carotid disease (stroke, permanent neurological damage, death), in-stent restenosis 5.04%, balloon expandable stent crush 0.84%, stent migration 0.84%.**

Conclusions. **Our data confirm that percutaneous dilation and stenting of the carotid artery represent an effective method which, during the periprocedural phase and the follow-up, does not expose the patient to any greater risk of complications than traditional surgery.**

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Introduction

Severe atherosclerotic lesion of the carotid artery is the main cause of stroke and transient ischemic attack. Its incidence may reach 5-7% per year in patients with carotid artery stenosis > 70% with or without symptoms.

While time-honored carotid endoarterectomy (CEA) is still regarded as the gold standard therapy[1] for most of the patients, carotid angioplasty with stent may be at the moment the only alternative to this treatment, when CEA is too risky or unfeasible.

Although percutaneous angioplasty has been fully acknowledged as far as coronary or peripheral artery disease is concerned, the issue is still controversial on the endovascular treatment of carotid artery disease with regard to its indications, devices to be used, and long-term results.

The assessment of risk related to carotid angioplasty with stent, in comparison to CEA, continues to be difficult, since materials and methods for this approach have not been standardized yet.

Carotid angioplasty is ever more widely performed by interventional cardiologist, radiologist and vascular surgeon groups, and technical improvements enable us to widen indications, to reduce procedure-related complications and to face very complex anatomy and lesions.

This registry was aimed at investigating the efficacy of endovascular therapy (angioplasty and stenting) of the extracranial carotid arteries in a non-selected population by using strategies and devices for coronary angioplasty.

Methods

A total of 119 consecutive patients (93 males and 26 females, mean age 70.4 ± 7.2 years) were enrolled to undergo percutaneous angioplasty and/or stenting of the extracranial carotid artery by using combined coronary and vascular angioplasty devices and techniques.

The primary endpoint of this study was to evaluate the feasibility, safety and efficacy of elective angioplasty and stent deployment in a "real life group" of patients with carotid atherosclerotic disease. During a 6 to 36-month study period late major adverse events were evaluated, either related to the endovascular treatment or due to other pathological conditions.

Patient inclusion criteria were:
• age > 50 years;
• carotid critical stenosis (> 75% carotid lesion), monolateral and bilateral;
• patients symptomatic for homologous neurological symptoms;
• patients symptomatic for heterologous symptoms, but with a cerebral computed tomography (CT) scan positive for silent cerebral focal ischemia homolateral to the culprit carotid lesion;
• patients asymptomatic for homologous and heterologous symptoms, with a cerebral CT scan negative for cerebral focal ischemia, but with carotid echo-Doppler findings proving a severe complex stenosis with fast progression of atherosclerotic disease;
• patients refused by surgeons as high-risk surgical subsets.

Patient exclusion criteria were:
• patients with thrombocytopenia, leukopenia, neutropenia and gastrointestinal bleeding in the previous 3 months;
• patients with an objective intolerance to the therapeutic association acetylsalicylic acid-ticlopidine.

Following these broad patient selection criteria, based more on carotid plaque characteristics and on the clinical patient status, both general and neurological, rather than on the percentage of carotid stenosis, the overall recruited patients were divided, from a neurological point of view, into symptomatic and asymptomatic subjects.

Symptomatic patients were defined those subjects symptomatic for homologous minor and/or major neurological symptoms (minor/major stroke, transient ischemic attacks, etc.).

Asymptomatic patients were defined as follows:
• subjects who did not experience homologous minor and/or major neurological symptoms (minor/major stroke, transient ischemic attacks, etc.), but with neurological history positive for heterologous symptoms (dizziness, dysarthria, disturbances of consciousness, memory loss, etc.) associated with a cerebral CT scan positive for silent cerebral focal ischemia homolateral to the culprit carotid lesion;
• subjects asymptomatic for homologous or heterologous symptoms, with a cerebral CT scan negative for cerebral focal ischemia, but with carotid echo-Doppler findings proving a severe complex stenosis with fast progression of atherosclerotic disease.

A written informed consent for intervention was obtained from all patients.

Prior to treatment, all patients underwent neurological examination (performed by an independent team), echo/color flow Doppler (lesion site and intracranial cerebral blood flow assessment), cerebral CT scan, and angiographic evaluation.

The clinical characteristics of the 119 study patients are summarized in table I.

Table I. Clinical characteristics of the study patients.

Clinical data	
No. patients	119
Sex (M/F)	93/26 (78.15%/21.85%)
Age (years)	70.4 ± 7.2
Cardiovascular risk factors	
Family history	30 (25.21%)
Hypertension	96 (80.67%)
Dyslipidemia	90 (75.63%)
Diabetes	21 (17.65%)
Smokers	24 (20.17%)
Ex-smokers	26 (21.85%)
Clinical history	
CAD	65 (54.62%)
Prior MI	19 (15.97%)
Prior PCI	14 (11.76%)
Prior CABG	29 (24.37%)
Severe heart valvular disease	7 (5.88%)
CMP	7 (5.88%)
Abdominal aneurysm	2 (1.68%)
Neurological history	
Symptomatic patients	
Homologous symptoms + positive cerebral CT	29 (24.37%)
Asymptomatic patients	
Heterologous symptoms + positive cerebral CT	70 (58.82%)
No symptoms and negative cerebral CT, but FP carotid atherosclerotic disease	20 (16.81%)

CABG = coronary artery bypass grafting; CAD = coronary artery disease; FP = fast progression; MI = myocardial infarction; CMP = cardiomyopathy; PCI = coronary intervention.

The indications to carotid endovascular angioplasty and stenting are shown in table II. Echo plaque characteristics and complexity in overall treated patients, symptomatic and asymptomatic patients are summed up in table III.

Within 24 hours of the procedure the patients underwent another neurological examination (performed by an independent team), and a complete echo/color flow Doppler evaluation.

Table II. Indications to carotid peripheral angioplasty/stenting.

Indications	No.	%
CS associated with severe CAD and/or polyvasculopathy	76	63.87
CS associated with LV function < 30%	3	2.52
Restenosis post-CEA	11	9.24
CS associated with contralateral carotid severe stenosis/occlusion	39	32.77
In-stent restenosis	6	5.04
Stent crush	1	0.84
Stent migration	1	0.84

CAD = coronary artery disease; CEA = carotid endoarterectomy; CS = carotid stenosis > 75%; LV = left ventricular.

A postprocedure cerebral CT scan was performed only in patients with documented neurological complications.

A clinical and echo/color flow Doppler follow-up was also planned at 3 and 6 months following the procedure and later at least once a year.

Medical treatment. All patients were treated with acetylsalicylic acid at a mean dosage of 125 mg/die and ticlopidine at a mean dosage of 500 mg/die at least 4-5 days prior to admission.

The mean dosage of sodium heparin utilized during the procedure was 100 IU/kg.

Ticlopidine (500 mg/die) was continued for at least 30 days after the interventional procedure (hemochrome and white blood count were checked up 7-10 days following the percutaneous intervention).

Antiplatelet therapy with aspirin was continued indefinitely.

Materials. Guiding catheter inventory, guidewire inventory and stent types regarding the 119 study patients are summarized in table IV.

Intravascular ultrasonography (IVUS) using an Ultra Cross 3.2 probe (Boston-Scimed, Maple Grove, MN, USA) was performed in 3 cases.

Description of the procedure. One hundred fifteen procedures (96.64%) were carried out via puncture of the right and/or left femoral artery. In 4 patients (3.36%), the procedure was conducted by puncture of the right and/or left radial artery.

Once the common carotid artery was selected, all patients underwent an angiographic examination of the intracranial circulation in the antero-posterior and/or lateral projection. The same angiographic check-up was

Table IV. Material inventory.

	No.	%
Guiding catheters		
Multipurpose 1-2 6F	12	10.08
Multipurpose 1-2 7F	2	1.68
Multipurpose 1-2 8F	24	20.17
Multipurpose 1-2 9F	9	7.56
Multipurpose Hokey Stick 6F	8	6.72
Multipurpose Hokey Stick 7F	1	0.84
Multipurpose Hokey Stick 8F	51	42.86
Multipurpose Hokey Stick 9F	5	4.20
Judkins Right 6F	1	0.84
Judkins Right 8F	4	3.36
Judkins Right 9F	1	0.84
Internal mammary 8F	1	0.84
Guidewires		
BMW 0.014 in	51	42.86
Hannibal 0.014 in	16	13.45
Platinum Plus 0.014 in	3	2.52
Choice PT Plus 0.014 in	15	12.61
Mailman 0.014 in	3	2.52
ATW 0.014 in	1	0.84
Platinum Plus 0.018 in	30	25.21
Stents		
Palmaz-Schatz	7	5.88
Easy Wallstent	48	40.34
Carotid Wallstent	26	21.85
Magic Wallstent	24	20.17
Expander	8	6.72
RX Ultra	4	3.36
SITO	1	0.84
NIR Primo	1	0.84

Table III. Echo plaque characteristics and complexity in the study group.

	Overall patients	Symptomatic patients Homologous symptoms Positive cerebral CT	Asymptomatic patients	
			Heterologous symptoms Positive cerebral CT	No symptoms Negative cerebral TC
Characteristics				
Soft-homogeneous	35 (29.41%)	9 (7.56%)	20 (16.81%)	6 (5.04%)
Hard-homogeneous	21 (17.65%)	3 (2.52%)	14 (11.76%)	4 (3.36%)
Heterogeneous with soft components	50 (42.02%)	16 (13.45%)	27 (22.69%)	7 (5.88%)
Unvaluable	13 (10.92%)	1 (0.84%)	9 (7.56%)	3 (2.52%)
Complexity				
Severe calcifications	25 (21.01%)	4 (3.36%)	8 (6.72%)	13 (10.92%)
Erosion/ulcer	30 (25.21%)	12 (10.08%)	11 (9.24%)	7 (5.88%)

CT = computed tomography.

performed at the end of the procedure in order to determine if there was any variation in the intracranial blood flow due to embolic phenomena or related to the procedure itself.

The left internal carotid artery was treated in 66 cases (55.46%), the right internal carotid artery in 42 cases (35.29%), the left common carotid artery in 7 cases (5.88%), and the right common carotid artery in 5 cases (4.20%).

Once the guide catheter was positioned at the right or left common carotid artery, the guidewire was placed directly in the intracranial portion of the ipsilateral internal carotid artery. In the patients who presented a pathology limited to the common carotid artery, the distal third of the guide (Platinum Plus 0.018, Boston-Scimed) was placed at the ipsilateral external carotid artery.

The cerebral protection with PercuSurge GuardWire[2] (PercuSurge Inc., Sunnyvale, CA, USA) was performed in 6 cases (5.04%). Peripheral and carotid angioplasty or stenting without cerebral protection was performed in 113 cases (94.96%)

In 61 cases (51.26%) stent delivery was carried out without pre-dilation (direct stenting). In the remaining 58 cases (48.74%) a pre-dilation of the tight lesion was performed by using coronary angioplasty dilation catheters.

The pre-dilation balloons were routinely undersized (artery/balloon ratio 1.8-1.5), in order to reduce vessel dissection and/or distal embolization. In 99 cases (83.19%) (either performed with pre-dilation or done by using direct stenting technique), stent placement was optimized through multiple dilation by using suitably-sized balloons based on quantitative analysis of the vessel. All post-stenting dilations lasted for just a few seconds (5-8 s), with a mean inflation pressure of 5 ± 2 bars.

Two cases were treated contemporarily by combined peripheral and carotid angioplasty and stenting. The first patient underwent angioplasty and stenting of the left common iliac artery and of the right internal carotid artery. In the second patient, angioplasty and stenting of the left renal artery and the right internal carotid artery were performed in the same endovascular session.

Of the 6 cases (5.04%) with severe in-stent restenosis, 5 patients were treated with balloon angioplasty and/or stent implantation. One patient decided to undergo surgical treatment.

In 3 cases, an IVUS examination was performed following stent placement due to the angiographic presence of severe wall calcification and/or suspected residual stenosis. Following the IVUS evaluation, an intra-stent post-dilation with a larger balloon was performed in 2 cases; in 1 case, IVUS showed a complete stent apposition to the arterial wall, and we did not modify this satisfactory final result with further post-dilation.

During the procedure, all the patients were constantly asked about overall conditions, and the function of the contralateral upper limbs was continuously monitored.

A mean dosage of 1 mg of intravenous atropine was administered to all the patients before the peripheral angioplasty/stenting procedure, in order to reduce bradycardia and hypotension potentially associated with carotid dilation.

At the end of the procedure, the arterial introducer was immediately removed and hemostasis of the femoral artery was achieved by using an Angio-Seal hemostatic device[3] (St. Jude Medical Daig, Minnetonka, MN, USA).

Results

Pre-procedure echo-analysis of carotid plaques. Looking at the relationship between echo-analysis of treated plaques, neurological symptoms and cerebral CT findings before the endovascular carotid procedure, the overall data were divided into three subgroups: data regarding all the admitted study patients, data regarding the symptomatic patient subset, and data regarding the asymptomatic patient subset (Table III).

In particular, some figures have to be outlined:

• 85 patients (71.43%) showed plaque echo-analysis patterns compatible for high embolization risk (Table III); 55 patients (46.22%) showed echo patterns of plaque complexity;

• 86.2% of the subjects with homologous neurological symptoms and positive cerebral CT showed plaque echo-analysis patterns compatible for high embolization risk (Table III); in this subset 55.17% of the subjects showed echo patterns of plaque complexity;

• 67.14% of the subjects with neurological history positive for heterologous symptoms and cerebral CT scan positive for silent cerebral focal ischemia homolateral to the culprit carotid lesion showed plaque echo-analysis patterns compatible for high embolization risk (Table III); in this subset 27.14% of the subjects showed echo patterns of plaque complexity;

• 65.0% of the subjects asymptomatic for homologous and heterologous symptoms, with a cerebral CT scan negative for cerebral focal ischemia, but with carotid echo-Doppler findings proving a severe complex stenosis with fast progression of atherosclerotic disease, showed plaque echo-analysis patterns compatible with high embolization risk (Table III); in this subset 100% of the subjects showed echo patterns of plaque complexity.

Procedural success of the carotid angioplasty and stenting. Percutaneous procedure was effective in 118/119 patients (99.16%). One case (0.84%) was ineffective because of spiral dissection of the left internal carotid artery distal to the stenosis site due to PercuSurge occlusive balloon vessel injury.

A single stent was successfully implanted in 112 cases (94.12%). In 2 cases (1.68%) was necessary to use another stent because of a residual dissection distal to the first implanted stent. In 1 complex case (0.84%) (severe tandem lesion involving both the left common and left internal carotid arteries) the two lesions were treated contemporarily by implanting three stents. In 5 patients (4.20%) with severe intra-stent restenosis after previous angioplasty and stenting a balloon dilation was used in 3 cases, and balloon angioplasty and re-stenting in 2 cases.

Procedural success of carotid peripheral angioplasty/stent deployment was defined as:
• angiography: < 30% quantitative coronary angioplasty residual diameter stenosis of all treated lesions, without alterations in the intracranial circulation at the post-procedural angiographic examination (the assessment of the residual diameter stenosis was performed by averaging at least two matched views on quantitative angiography);
• echo color Doppler: absence of significant residual stenosis and pathological acceleration in blood flow (< 1.5 m/s).

Procedural angiographic and echo color Doppler success was achieved in 99.16% of all the treated lesions.

In-hospital adverse events. The neurological examination performed within 24 hours of the procedure was comparable to the pre-procedural check-up in 115 patients (96.63%).

No patient experienced major neurological complications (major stroke and death 0%). One patient (0.84%) showed, 12 hours after the procedure, symptoms characterized by transient episodes of subjective dizziness with moderate spatial disorientation. These symptoms regressed completely 72 hours after the procedure, and CT and magnetic resonance imaging examinations excluded the presence of any brain lesion. This patient was discharged asymptomatic 7 days after the procedure.

One case (0.84%) was complicated by subarachnoid hemorrhage 6 hours after the procedure, probably related to the anticoagulation treatment. A complete resolution of symptoms and radiological findings (CT and magnetic resonance) was achieved within 3 days.

Two patients (1.8%) showed, between 4 to 6 hours following the procedure, weakness of the arm and some mild deficit of speech output. In both patients CT scan demonstrated a small ischemic infarct area in the hemisphere supplied by the treated carotid artery. All these patients had a complete recovery of the neurological status in a few days, and were discharged within 6 to 10 days after carotid stenting without neurological deficit.

We have to signal, as asymptomatic procedural complication, 1 case (0.84%) of spiral dissection of the left internal carotid artery distal to the stenosis site due to PercuSurge occlusive balloon vessel injury. Although we were unable to cross the spiral dissection and the intracranial left internal carotid artery completely occluded, the patient did not complain of any symptom. The neurological examination did not show any neurological deficit neither in the cath lab nor in ward. CT and magnetic resonance imaging evaluation excluded the presence of any lesion. We can explain this behavior reminding that the hemisphere supplied by the occluded carotid artery received a sufficient blood flow by the contralateral carotid circulation and intracranial posterior circulation.

Follow-up data. At present time complete clinical follow-up data on all the 119 patients are available.

The follow-up observation time ranges from 6 to 36 months following the percutaneous procedure: in 76 patients (63.86%) complete data on 1-year follow-up were collected, and in the remaining 43 patients (36.14%) complete data on the 6-month follow-up are now available.

Follow-up data were collected during serial non-invasive post-peripheral angioplasty examinations:
• neurological examination at 3 and 6 months after the procedure;
• Doppler of the epiaortic vessels and intracranial blood flow at 3 and 6 months after the procedure, and later once a year.

Follow-up data are reported in table V. The overall in-stent restenosis rate was 5.04%. Looking at a more detailed analysis regarding the correlation between kind of implanted stents, type of lesion and restenosis, we cannot at the moment make any definitive statement. The only interesting element emerges from a clinical observation, and it is not actually related to technical or procedural data: in 4 (80%) of the 6 patients who experienced this kind of late complication the clinical history was positive for a particularly aggressive and accelerated carotid atherosclerosis associated with untreatable severe hyperlipidemia.

The reported carotid stent crush occurred in a diseased 72-year-old man (previous anterior myocardial infarction, previous coronary angioplasty on the left anterior descending coronary artery, previous minor stroke, right internal carotid severe ulcerated stenosis, left internal carotid total occlusion, reduced vasomotor cere-

Table V. Clinical follow-up data.

	No.	%
Neurological death	0	0
Overall in-stent restenosis	6	5.04
Stent crush	1	0.84
Stent migration	1	0.84
Neurological event free patients	111	93.28
Minor + major neurological events*	8	6.72
Major neurological events**	0	0

* restenosis + *de novo* lesions + neurological late events + neurological death; ** permanent neurological damage + neurological death.

bral reserve), refused by the surgical team, who underwent a carotid angioplasty and stenting by using a coronary balloon expandable NIR Primo 4.0/16 mm 9 cells.

The echo and quantitative angiography evaluation showed a very small reference vessel diameter (4.0 mm) and, for this reason, we decided to implant a premounted coronary stent designed for vessels till 4.5 mm in diameter. The procedure phases were conducted under IVUS guidance, with an optimal immediate result.

Three months later, in the absence of symptoms, the non-invasive evaluation showed a stent crush involving the distal segment of the device, due to the poor hoop strength of this coronary stent. The case was refused for the second time by surgeons as a too high-risk patient, and consequently was re-treated with an endovascular procedure which consisted in Magic Self-expandable Wallstent implantation, with a proper immediate result and a stable 1-year follow-up.

The reported carotid stent migration occurred in a 63-year-old man, treated for a severe ostial left internal carotid artery lesion not involving the bifurcation, by implanting a self-expandable Easy Wallstent 6.0/28 mm. The stent was delivered just at the origin of the left internal carotid artery, leaving free the bifurcation. Six months later, in the absence of symptoms, the non-invasive evaluation showed a stent migration of about 1 cm towards the distal left internal carotid artery, with a pathological blood flow acceleration (2.9 m/s) at the proximal edge of the device.

The case was re-treated with an endovascular procedure which consisted in Easy Wallstent 8.0/41 implantation from the carotid bifurcation to the mid part of the first stent, with a proper immediate result and a stable 1-year follow-up.

Discussion

Stroke is the third major cause of death in Western countries and its complications lead to significant socio-economic problems connected with extended hospitalization and rehabilitation of patients with neurological damage.

Between 20 and 30% of strokes are caused by critical pathology of the carotid artery.

Various prospective studies in symptomatic patients (NASCET[1] and ECST[4]) and in asymptomatic patients (ACAS[5]) have shown that surgery is better than medical therapy in patients with critical carotid stenosis.

CEA is a surgical procedure that continues to represent the treatment of choice for this type of pathology. Nevertheless, various retrospective and prospective studies have demonstrated that this method is not entirely free from risk[6-10].

NASCET and ECST studies showed an equivalent percentage of post-CEA strokes, with an incidence of 5.8 and 7.5% respectively. Moreover in the ACAS group there was a lower incidence (2.3%) of ischemic events in asymptomatic patients treated with CEA.

The overall risk of post-CEA neurological damage is up to now around 7.7%[11] according to the literature data, based on a review of numerous surgical cases.

Separate mention must also be made of patients with associated ischemic heart disease. In these patients, combined CEA and bypass surgery is associated with a 4.5-7.1% incidence of cerebral ischemic events and a high mortality[12].

Moreover, traditional surgery must take other potential complications into account. They are represented mainly by possible cranial nerve damage, with percentages ranging from 7 to 25%, and by hematoma and/or local infection at the site of the surgical incision, with an incidence of 3.5 to 5%[1,4,5,11,13-16].

Relevant data are represented by the incidence of post-CEA restenosis that is extremely variable, since percentages ranging from 2 to 36% have been reported. The onset of clinically-observable restenosis occurs in 8-10% of cases[16,17]. In this respect, it is important to point out that surgical treatment of restenosis is subject to a higher perioperative risk.

Although percutaneous balloon angioplasty has been used on the carotid artery as far back as the ’80s, it has never gained approval, above all because of the high risk of distal embolization. The advent of the endovascular stent has undoubtedly led a number of operators to reconsider the option of percutaneous treatment of this vascular district.

There are now numerous data in the literature reporting extensive case studies done on patients treated for critical carotid stenosis, with good immediate results. The most important studies[18-24] have demonstrated that percutaneous treatment of carotid pathologies is correlated with a risk of cerebral ischemic events and, more generally, with a rate of complications that is no higher than that reported with traditional surgery.

If we look at our registry data, compared to the recent literature data[18-23,25,26], the percutaneous approach to obstructive carotid pathology is very interesting in terms of procedural success, in-hospital complications, and late follow-up results.

In our broad inclusion criteria population, percutaneous procedure was effective in the 99.16% of patients. The immediate results are encouraging: procedural success rate 99.16%, in-hospital major neurological symptomatic complications 0%, in-hospital minor neurological symptomatic complications 3.36% (two minor strokes, one transient ischemic attack and one subarachnoid hemorrhage).

The importance of the reported immediate results is even more interesting if we consider that they were obtained in a non-selected group of patients with high degree of complexity both in terms of clinical status and carotid stenosis.

Summing up the previously reported data on clinical conditions and on carotid plaque characteristics, we can observe that:

• from a clinical standpoint, the majority of enrolled patients (63.87%) were high risk patients affected by severe carotid lesion(s) associated with severe coronary artery disease and/or polyvasculopathy;
• from a neurovascular standpoint, a large number of enrolled patients (32.77%) had a severe carotid stenosis associated with contralateral carotid severe stenosis/occlusion;
• 85 patients (71.43%) showed plaque echo-analysis patterns of high embolization risk (soft homogeneous plaques in 35 patients, heterogeneous plaques with soft components in 50 patients);
• 55 patients (46.22%) showed angiographic and/or echo-analysis patterns of plaque complexity (heavy calcifications in 25 patients, plaque erosion/ulceration in 32 patients).

Although this patient setting, what has to be outlined is the absolute absence of major neurological complications, even if only 5.04% of the endovascular carotid procedures were conducted under cerebral protection. The reasons why we decided to unprotect the most of the carotid procedures are various and complex.

First of all, we started our experience on carotid endovascular treatment in 1997, when no protection devices were commercially available. Moreover, both the cerebral protection techniques described at that time by Theron et al.[27-29] and Kachel[30] reported a high cerebral complication rate related to the use of protecting device itself.

Actually, only in the late 1999 major technical progress allowed the introduction of the GuardWire temporary occlusion balloon and the Aspiration system (PercuSurge, Inc.)[2,31].

In second instance, since the beginning we thought that at least three kinds of cerebral protections can be carried out during carotid angioplasty and stenting. The most known and generally accepted one can be defined "passive cerebral protection", and consists in the use of devices which allow the operator to capture and remove embolic material generating during the procedure. The other two technical ways to reduce cerebral complications can be grouped under the concept of "active cerebral protection", which means any method and/or work strategy to minimize the probability to generate big particles of embolic material during the endovascular procedure.

On the one hand, stent implantation by itself may contribute to stabilize the plaque, by fixing the plaque materials to the artery wall (intrinsic antiembolic stent property). By the light of this assumption, when technically feasible we brought on a direct stenting procedure without pre-dilation. In our series direct stenting was performed in 61 patients (51.26%).

On the other hand, the incidence of cerebrovascular accidents induced by angioplasty may be reduced by using very low profile wires, balloons and stents, and by carrying on procedures as short as possible, in order to reduce trauma incurred at the unstable plaque site. Moreover, the post-stenting dilation phase has to be managed with care, avoiding aggressive balloon inflation and any other maneuver which may cause a plaque shifting or protrusion through the stent meshes.

Nevertheless, we have to underline that this "active cerebral protection" strategy consists in the process of tailoring the endovascular procedure to a specific patient and a specific kind of carotid lesion. Consequently, the "active cerebral protection" strategy bases its rationale mostly on a deep knowledge of the patient clinical status, vascular anatomy, carotid plaque characteristics and complexity and, not less important, technical features of the materials (guiding catheters and sheaths, wires, balloon, stents, etc.) at disposition of the operator.

In this context a key role is played by echo-Doppler evaluation of the characteristics and complexity of the carotid plaque, which allowed us to identify the high risk carotid bifurcation lesions in terms of plaque echolucency.

Actually, all the patients admitted to this study were treated under the "active cerebral protection" strategy, and only few of them (n = 6, 5.04%) at high risk of periprocedural stroke underwent the procedure, when PercuSurge system was commercially available, under "passive cerebral protection" strategy too.

The overall follow-up data showed the absence of major adverse late events related to carotid disease (stroke, permanent neurological damage, death), in-stent restenosis 5.04%, balloon expandable stent crush 0.84%, and stent migration 0.84%. Despite these outstanding data, we are aware of the small number of patients we have enrolled and treated, and many doubts remain about the real short- and long-term results evaluated on significant numbers.

Prospective results are still unknown, not only about the real restenosis rate and/or the recurrence of symptoms, but also in terms of the behavior of the stent in that particular district.

Nevertheless, our data confirm that percutaneous dilation and stenting of the carotid artery is an effective method which, during the periprocedural phase and follow-up, does not expose the patient to any greater risk of complications than traditional surgery.

In the broadest case studies, the present restenosis rate is about 2.8%[32] at 6 months following percutaneous carotid stenting. In our series the restenosis rate following stent implantation was 5.04%.

About the problem of deformation of balloon expandable stents, we had in our series only one case of stent crush (NIR Primo 9 cells 16 mm). We must here highlight several reported cases of stent deformation and/or compression following the implant[33,34].

In conclusion, despite the well-established advantages of surgical therapy to treat carotid pathologies, new endovascular methods with a percutaneous approach and the use of stents are being used more and more extensively. The advantages of the endovascular procedure,

whose periprocedural embolic risks are comparable to those of surgery, include reduced system invasiveness, the possibility of treating patients with a high level of general impairment, and the patient's rapid discharge from the hospital, thereby decreasing hospitalization time and lowering healthcare costs. Our experience demonstrates that the use of coronary angioplasty material is effective in treating carotid stenoses, minimizing the risks of intracranial embolization even without the use of cerebral protection systems, i.e.Theron's technique, Kachel's technique, PercuSurge technique[2,24,27-31].

Despite the fact that the immediate peri- and postprocedural results are encouraging, we feel that careful long-term clinical follow-up and examinations are necessary in order to assess these good results and the long-term patency of the arterial segments treated percutaneously.

Up to now, the accepted strict indications to carotid percutaneous treatment with stent are: restenosis on previous CEA; carotid stenosis at the base of the skull whose surgical approach entails subluxation of the mandible; stenosis involving the common carotid artery difficult to access with a cervical surgical approach; stenoses of a non-atherosclerotic etiology (fibromuscular dysplasia, Takayasu's arteritis, radiation pathology); critical bilateral carotid stenosis associated with reduced vasomotor cerebral reserve; critical carotid stenosis associated with contralateral carotid occlusion; and critical carotid stenosis in surgical high-risk subsets.

The future spread of these indications to treatment must move ahead with caution until the long-term results can be verified[35].

Nevertheless, by the light of the present results and technical developments, we could reasonably review and extend the previous indications, looking more at the feasibility of the procedure than the refusal by the surgeon to treat the carotid lesion itself.

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