ANGIOPLASTY AND STENTING OF THE CERVICAL CAROTID BIFURCATION UNDER FILTER PROTECTION: A PROSPECTIVE STUDY IN A SERIES OF 53 PATIENTS

G. BONALDI (1,2), L. AIAZZI (3), F. BARUZZI (2), F. BIORLI (4), A. FACCHINETTI (2), P. FACHINETTI (5), A. LUNghi (1), F. TERRANEO (3)

(1) Neuroradiology Department, Ospedali Riuniti, Largo Barozzi 1, 24128 Bergamo, Italy.
(2) Neuroradiology Department, Ospedale di Circolo e Fondazione Macchi, Viale L. Borri 51, 21100 Varese, Italy.
(3) Vascular Surgery Department, Ospedali Riuniti, Largo Barozzi 1, 24128 Bergamo, Italy.
(4) Neuroradiology Department, Ospedale di Circolo e Fondazione Macchi, Viale L. Borri 51, 21100 Varese, Italy.
(5) Neuroradiology Department, Ospedali Riuniti, Largo Barozzi 1 – 24128 Bergamo, Italy.

SUMMARY

The aim of this study is to assess safety, reliability, ease of use and usefulness of filter protection devices during angioplasty and stenting of stenotic lesions of the cervical carotid bifurcation. Over a period of 42 months, 53 patients harboring a cervical carotid bifurcation stenotic lesion were treated, by angioplasty and/or stenting using filter protection devices of different kinds. The stenosis was atherosclerotic in 48 cases, post-surgical in four and post-radiation in one case. In all cases, the treatment was successful, with good restoration of the luminal diameter. There were three major strokes (5.6%) and one minor stroke (1.9%).

Two of these (one major, one minor) occurred a few hours after the stenting procedure and both seemed by all evidence due to a hemorrhagic hyperperfusion syndrome. One hemiparesis and dysphasia occurred two days after the procedure, secondary to subacute thrombosis with occlusion of the stent. One patient complained of three episodes of decrease in visual acuity of the eye ipsilateral to the stenting in the two weeks following treatment. In conclusion, in our experience, use of the devices adds only few minutes to the procedure time; direct lesions of the arterial wall, such as dissections or intraluminal thrombi, related to the use of filters were never observed, and spasm of the distal I.C.A. also proved rapidly regressive. The content of all filters, if any, was histologically examined, but plaque material was found only in one case, probably owing to our primary stenting technique without use of pre-dilation. The major technical drawback is in-filter coagulation, which occurred in 16 cases, occluding the membrane of the filter and thus slowing or blocking intracranial flow. Such an event can be counteracted by a more aggressive anti-coagulation protocol, which could, however, be responsible for the two complications with hemorrhagic brain infarction. Furthermore, we observed two other major neurological events, which bring the incidence of neurological complications in this series as high as 7.5%. Therefore, it is our opinion that safety of filters is not yet proven, and consequently great care must be taken in their use.

Key words: angioplasty, carotid artery disease, stents.

RÉSUMÉ

Angioplastie et stenting de la bifurcation carotide cervicale avec filtre de protection : étude prospective de 53 patients

Le but de ce travail est d’évaluer la tolérance, la fiabilité, la facilité d’utilisation et l’intérêt des filtres de protection pendant l’angioplastie et/ou stenting des sténoses de la bifurcation carotide. Sur une période de 42 mois, 53 patients porteurs d’une sténose de la bifurcation carotide ont été traités par angioplastie et/ou « stenting » en utilisant des filtres de protection cérébrale de différents types. Les sténoses étaient d’origine athéroscléreuse (n = 48), post-chirurgicale (n = 4) et post-radiothérapique (n = 1). Les résultats anatomiques étaient dans tous les cas satisfaisants, avec une restauration satisfaisante du diamètre artériel. Il y eut trois cas d’accident cérébral majeur (5,6 %) et un cas mineur (1,9 %). Deux de ces accidents (un majeur et un mineur) sont survenus dans les heures suivant le traitement et correspondaient à un syndrome d’hyperperfusion hémorragique. Une hémiparésie avec dysphagie, survenue 2 jours après le traitement, était liée à un éthrombome subaigu avec occlusion du stent. Un patient présenta au cours des deux semaines qui suivirent le traitement, trois épisodes de cécité monocular homolatérale à l’endoprothèse. En conclusion, dans notre pratique, la mise en place de filtres de protection cérébrale ajoute quelques minutes à la durée totale du traitement ; des dissections de la paroi artérielle ou thrombi intraluminaux n’ont jamais été observés ; les spasmes distaux de l’artère carotide ont toujours été résoluts. Le contenu des filtres a été étudié ; du matériel de plaquage n’a été observé qu’une seule fois (présence probablement liée à notre technique initiale de stenting sans prédilatation). La principale limite technique est la survenue d’une coagulation au sein du filtre, observée dans 16 cas, responsable d’une occlusion de la membrane du filtre et d’un ralentissement ou d’un arrêt de la circulation. Un tel événement est prévenu par une anticoagulation plus importante qui pourrait être à l’origine de deux infarctus hémorragiques. De plus, nous avons observé deux autres complications majeures ce qui porte le taux de complications dans cette série à 7,5 %. À notre avis, la tolérance des filtres n’est pas aujourd’hui démontrée et de grandes précautions sont nécessaires lors de leur utilisation.

Mots-clés : angioplastie, artère carotide, endoprothèse.

INTRODUCTION

Carotid artery angioplasty and stenting is becoming a more and more popular tool for treatment of stenotic lesions of the extracranial internal carotid artery (I.C.A.), as an alternative to carotid endarterectomy (C.E.A.), which has proved its efficacy and superiority over medical management for treatment of symptomatic and asymptomatic lesions in large, randomized trials [2, 6, 19]. However, endovascular percutaneous techniques, even if highly appealing for their potential as a safer, less trau-
motic alternative, remain controversial, owing to lack of such large, controlled, randomized trials and to excessive heterogeneity of patients’ cohorts in terms of pathologic processes, lesion sites, indications for treatment or recruitment criteria, and above all in terms of techniques and devices used. Several unresolved issues have prevented their development, the main one being the risk of neurological complications due to brain embolization by plaque fragments during the procedure.

Moreover, agreement on the best procedural technique from the numerous teams performing the procedure worldwide is still lacking. For instance, the use of different types of balloons arresting the flow during deployment of the stent and dilation to protect the brain from possible embolism has been advocated for many years by only a few [12, 29-31], so that the most frequent technical choice of different authors remains that of primary stenting without protection, as classically described [9, 10, 13, 14]. This was our technical choice until recently, when, as already reported [3, 4], we observed one major stroke from middle cerebral artery embolization by atherosclerotic material migration caused by balloon compression of the atherosclerotic plaque.

Since then, we have modified our attitude and we have been looking for a safe protection device. First, we tried a new system needing balloon I.C.A. occlusion, the GuardWire® (PercuSurge) [3, 4]. Only recently, have filter protection systems been developed and tested in ex vivo models [21]; thereafter the filters became available on the market. In July 2000, we started the present, prospective study on 38 patients also underwent CT angiography, mainly for confirmation of US findings and evaluation of I.C.A. plaque morphology and composition (above all in relation to ulcerations or calcifications).

Intra-procedural trans-cranial-Doppler was not available.

Subjects and Methods

Patient characteristics and lesion morphology

From July 2000 to December 2003, 53 patients with cervical carotid bifurcation stenotic lesions were treated, by P.T.A. and stenting (50 cases) or stenting alone (3 cases). The patients in this series represent 17.5% of the 303 patients treated since our first case in 1986 (114 P.T.A.s, 44 Palmaz stents, 145 self-expandable stents) [4, 18, 23].

Of the patients 35 were male, 18 were female; the mean age was 68 years (range 55-85 years).

All treated patients gave written consent; patients unable to give informed consent were excluded, as were patients disabled because of stroke or dementia.

The carotid bifurcation stenosis was due to atherosclerotic plaque in 48 cases, post-surgical restenosis in 4 cases, and post-radiation stenosis in one case.

38 patients were symptomatic, with symptoms being congruent with the lesion treated, and 15 were asymptomatic; in these cases, the choice of endovascular treatment was based either on a refusal of surgery by the patient, or to the fact that the patients were considered at high risk for surgery, both for poor general condition, local surgical anatomy (such as high retro-mandibular bifurcation or in the post-C.E.A. restenosis cases) or contralateral I.C.A. occlusion.

Degree of stenosis, measured according to the NASCET criteria [20], was not less than 70% for asymptomatic patients, 50% or more in symptomatic patients [4].

Imaging evaluation

The pre-treatment angiographic examination always includes a 4-vessel study with intracranial circulation. Furthermore, for all patients a carotid ultrasound (US) examination was performed before and immediately after the procedure [7-17].

Immediately after P.T.A. and stenting, angiographic runs are performed both of the treated cervical bifurcation and of the homolateral intracranial circulation.

Pre-treatment brain M.R.I. and/or C.T. were available for all patients. Before intervention 38 patients also underwent CT angiography, mainly for confirmation of US findings and evaluation of I.C.A. plaque morphology and composition (above all in relation to ulcerations or calcifications).

Drug regimen

All patients but one (see below) received for at least 48 hours before the procedure aspirin (100mg twice daily) and one among either ticlopidine (250mg twice daily) or clopidogrel (75mg daily). At the beginning of the study, we used to administer heparin during the procedure, to achieve an activated clotting time of at least two and a half times the baseline; this practice was subsequently modified, as discussed later. From a practical point of view, once the Arrow® sheath or guiding catheter are positioned in the common carotid artery, we start injecting 100 I.U./kg of heparin, subsequently we control ACT and add 2.000 I.U. at a time until the desired ACT value is obtained.

1mg atropine was administered just before balloon dilation of the stenosis for possible bradycardia due to carotid glomus stimulation. Blood pressure and neurological status were carefully monitored during the whole procedure.

Administration of low molecular weight heparin was maintained for at least 48 hours after treatment (more frequently for 5-6 days, at decreasing doses), ticlopidine or clopidogrel were discontinued after 1 month, while aspirin is maintained life long.

Angioplasty and stenting technique

The endovascular treatment was performed under local anesthesia and mild sedation in most cases, under general anesthesia, owing to lack of cooperation by the sedated patient, in 5 cases. Percutaneous access was obtained through the femoral route, and a long, armed sheath (Arrow® by Arrow International) or a guiding catheter was posi-
tioned in the common carotid. In all cases, stents and balloons with monorail architecture were used.

The closed filter first has to pass through the stenosis and in all but one case, this happened without need of pre-dilation; in one case, a pre-dilation with a three-mm. balloon was necessary owing to a pre-occlusive stenosis with a curved shape (figures 1a and 1b). After opening of the filter, the stent is mounted on it, placed across the bifurcation, covering the whole plaque and not only the stenotic segment, and also involving the origin of the external carotid artery, and deployed. A control angiography is done and, if deemed necessary, the balloon is mounted for dilation.

Of the 53 overall treatments, 50 were stenting followed by angioplasty, with balloon sizes ranging from 4.5 to 6mm, while three lesions underwent only stenting without P.T.A.

In 47 cases a Carotid Wallstent® (Boston Scientific) was used, with an unconstrained diameter of 10mm and length between 3 and 5cm, tailored to plaque’s and bifurcation’s morphology; in 6 cases an Acculink® (Guidant) stent was used, with a conic shape and a 7 to 10mm unconstrained diameter.

The femoral sheath is immediately removed; vascular occlusion devices (Angioseal® by St. Jude Medical, except 1 case – see below) are always used to control femoral bleeding.

Patients are usually discharged the third day after the procedure.

Filter devices

We used the AngioGuard® and AngioGuard Xp® systems (Cordis) in 36 cases, the NeuroShield® system (MedNova) in three cases, the Trap® system (Microvena) in two cases, the Spider® system (an evolution of the Trap, ev3) in seven and the Filter-Wire EX® system (Boston Scientific- EPI) in five cases.

All of these systems are designed for emboli and debris capture while maintaining a valid flow to the distal carotid territory. All the systems, except the Spider®, cross the lesion closed in a sheath, subsequently retrieved for opening; a second, larger sheath is mounted at the end of the procedure for retrieval of the filter with possible debris inside. The Spider® system provides crossing the stenosis, using whichever angiographic 0.014” wire, with a delivery catheter, through which the filter is subsequently inserted. All filters are “built-in” on a guide wire, which constitutes their proximal part, on which stent and balloon are mounted, thus allowing a single passage through the stenosis. The distal tip of the filter system is a short, floppy, shapeable and steerable tract of wire.

![Figure 1](image1.png)

**FIG. 1.** – Tight left I.C.A. stenosis (a), in b after pre-dilation with a 3mm coronary balloon and positioning of an Angioguard filter (arrow). In c the lesion after p.t.a. and stenting. Four hours later the patient underwent a stroke with aphasia and hemiplegia; the C.T. scan showed a haemorrhagic temporo-parietal lesion, with mass effect (d), probably related to a hyperperfusion syndrome. See text for discussion.

**FIG. 1.** – Sténose serrée de l’artère carotide interne gauche (a), après dilatation par un ballonnet coronaire de 3 mm et mise en place d’un filtre Angioguard (flèche) (b). Aspect après dilatation et endoprothèse (c). Quatre heures après le traitement, survenue brutale d’une hémitéligie et aphatie ; le scanner montre une lésion hémorragique temporo-pariétale (d), probablement en rapport avec un syndrome d’hyperperfusion.
The AngioGuard® has a distal umbrella-shaped capturing polyurethane membrane, with laser-drilled microholes, assembled on nitinol expandable arms.

Also the NeuroShield® filter entraps the embolic material by means of an expandable microporous membrane, whose design differs in its shape, which is similar to a capsule with proximal large triangular flow inlet ports and distal holes.

The FilterWire EX® also has a capturing basket made of a microporous plastic membrane, but characterized by the shape of a windsleeve, held opened in the arterial lumen by a single, proximal metallic ring.

The Trap® system, no longer available, and the Spider® differ from the other filters for the fact that they are composed of a nitinol mesh basket coated with heparin, instead of a plastic membrane with microholes; the Spider® is characterized by a windsleeve shape, similar to the previously described FilterWire EX®.

Histopathologic evaluation

Filters were examined after retrieval in the angiographic suite using a 3X magnifying glass with neon light; filters with evidence of solid material on them were sent for examination.

The filters with entrapped particles were fixed in buffered formalin (formaldehyde 4% w/v and acetate buffer 0.005M) for 24 hours, and then the collected particles were routinely processed to paraffin wax and 5µm thick sections were stained with hematoxylin-eosin for histopathologic examination.

RESULTS

Procedural and morphological results

The procedure was performed in all attempted cases and a good restoration of the lumen, with no more than 20% residual stenosis, was always obtained. Local complications, such as wall dissec-

tions or intraluminal thrombi were never angiographically evident; intracranial, post-P.T.A. angiographic runs never showed evidence of cerebral embolism.

A more or less relevant spasm of the distal I.C.A., at the level where the filter was positioned, was observed just after filter retrieval in every case, though without any angiographically evident slowing of flow; such a spasm was severe in only four cases and always proved to be rapidly regressive (figure 2).

The opened filters never caused an angiographically evident slowing of flow before or after primary deployment of the stent. In nine cases a more or less severe slowing of flow was noted, and in six other cases a complete arrest of flow was present, always and only after balloon inflation for angioplasty. In all cases, a normal flow rate was restored after filter retrieval.

In 17 cases (32%) small, yellowish, translucent fragments were present on the inner surface of the filter membrane. In every case where a slowing of flow was noted after P.T.A., debris was present on the filter membrane, and the amount of such material was large (more than 2/3 of the membrane surface) and relatively non-homogeneously thick in the six cases where arrest of flow was present.

Clinical results

There were three major strokes (5.6%): — one major aphasic and haemiplegic stroke, that persisted for months post-procedure and regressed only partially. The stroke occurred four hours after a stenting procedure, which developed without any problem; all evidence indicated it was due to a hyperperfusion syndrome and not to cerebral embolism (figure 1). Blood pressure parameters were in the normal range for the patient’s age after the procedure. Before treatment, the patient was symptomatic for congruent hemiparetic T.I.A.s, the last episode having occurred more than 3 months before the procedure;

![Fig. 2. – After p.t.a. and stenting of a right I.C.A. stenosis and before filter retrieval (E.P.I. filter, short arrow), the control angiogram shows severe spasm of the artery (a-long arrow). Few minutes after filter retrieval the spasm had completely resolved (b).](image-url)

**FIG. 2.** – Après dilatation et endoprothèse d’une sténose de l’artère carotide droite et avant retrait du filtre (filtre EPI, flèche), le contrôle angiographique montre un spasme sévère (a). Régression complète du spasme quelques minutes après le retrait du filtre.
ANGIOPLASTY AND STENTING OF THE CERVICAL CAROTID BIFURCATION

— one hemiparesis and dysphasia occurring two days after the procedure, secondary to subacute thrombosis with occlusion of the stent (figure 4) and still lasting months later (see discussion); the patient was asymptomatic before stenting;

— one patient (who presented with several hemiparetic T.I.A.s, the most recent a few days before treatment) complained, in the two weeks following the procedure, of three episodes of decrease in visual acuity of the eye homolateral to the stenting (which occurred without any problem and with very good morphological result), despite medical therapy (aspirin, ticlopidine and low molecular weight heparin); the third episode was close to unilateral total blindness. Multiple follow-up US examinations showed patency of the stent, without evidence of intraluminal thrombi; fluoroscopy showed thrombosis of the central retinal artery. Clinical episodes subsided when the patient was put under Coumadin, but one year after the last episode, the patient has only partially recovered visual acuity. An angiographic control performed eight months after the procedure confirmed patency of the stent.

There was one minor, hemiparetic stroke (1.9%), again probably due to a hyperperfusion syndrome (figure 3), in a patient who had had two episodes of amaurotic T.I.A.s., the most recent 4 months before the stenting procedure. The patient developed symptoms 2 hours after the procedure, with blood pressure parameters in the normal range for the patient’s age, and a C.T. immediately performed showed slight hypodensity in the subcortical frontal region, where a small haematoma was found the day after. The hemiparesis regressed almost completely over a few weeks.

We also observed other less relevant, completely regressive clinical events:

— 2 losses of consciousness, lasting no more than twenty seconds, after balloon inflation, due to severe bradycardia/asystolia caused by carotid glomus stimulation and despite use of atropin before balloon inflation;

— one unstable bradycardia/hypotension lasting for 2 days (with an episode of loss of consciousness for few seconds 1.5 hours after stenting) that required intravenous dopamine infusion;

— one hemiparetic transient ischemic attack (T.I.A.) 3 days after stenting, which lasted only 5 minutes;

— one groin haematoma, without any sequela; a percutaneous suture device was used only in this case for femoral haemostasis;

— one loss of consciousness during arrest of flow due to filter filling with solid material; the loss of consciousness lasted for the two minutes needed for filter retrieval;

— one asymptomatic, immediate post-stenting occlusion of the external carotid artery.

Histopathological results

In all cases but one the material inside the filters, sent for histological examination, proved to be fibrin filaments, in six cases mixed with platelets. In only one case, tiny fragments of plaque material, mixed with cholesterol crystals, were found on the filter’s membrane.

DISCUSSION

Crossing of the stenosis and positioning of the filtering basket in the distal I.C.A. have been fast and easy, thanks to the good shapeability and torqueability of all the devices tested, with low or no frictions on the plaque surface. In only one case we did decide, owing to the angiographic aspect of the stenosis, to pre-dilate as a first step, not trying to...
push the closed filter through a pre-occlusive, curved lumen (Figure 1). Filters are becoming more and more flexible, and their profiles are improving constantly as well; this is an important safety feature, reducing the aggressiveness of the device crossing the non-protected plaque.

The support provided by the main wire body of the devices to the catheter systems carrying the stent and the balloon was good with no need for dangerous tractions on the filter basket once positioned in the distal cervical carotid artery. We found that these characteristics were common to all devices to a similar extent, the learning curve is short and technical skills acquired with one device can be transferred to another. During endovascular procedures, every waste of time potentially adds to the complication rates, but it is our opinion that a procedure performed with a filter only slightly differs from a classical primary stenting and angioplasty: the overall increase in the average procedure times with use of filters is, in experienced hands, less than 10 minutes, and we do not think that this really adds to the complication rates.

The present study is not aimed to detect differences between filters. However, from our evidence and experience the filters we tested seem very similar in their use and performance; the most frequent side effect, i.e. spasm, is equally spread in a similar way among the different filters, as far as such a consideration can be made with our relatively small numbers. We think that it would be scientifically incorrect and commercially unfair to make a comparison between the different devices, in some cases used only twice, and thus we do not at the moment offer any such comparison.

Events related to carotid glomus stimulation maintained the same frequency as before use of filters.

**Figure 4.** Severe atherosclerotic I.C.A. stenosis (a), with homolateral stenosis of the siphon (b); after stenting and balloon angioplasty the flow in the distal internal carotid is blocked by debris collected by the filter; the distal and proximal markers of the latter are indicated by the arrows (c). The flow is normally restored after filter retrieval (d). Two days later the patient developed contralateral disphasia and hemiparesis; the stent proved to be occluded at a C.T.-angio examination (e-f/arrows). See text for discussion.

**Fig. 4.** Sténose athéroscléreuse sévère de l’artère carotide interne (a), avec sténose du siphon homolatéral (b). Après angioplastie et stenting, le flux distal carotid est bloqué par des débris collectés dans le filtre ; les marqueurs proximal et distal sont indiqués par des flèches (c). Le flux est restauré après retrait du filtre (d). Deux jours plus tard, le patient présente une dysphasie avec hémiparésie ; l’occlusion du stent est démontré par angioscanner (e, f).
After positioning and opening the filters and before or after primary deployment of the stents, blood flow in the distal I.C.A. never showed a perceivable slowing at fluoroscopy, allowing the operators to perform the subsequent phases of the procedure easily, with no need for risky haste, as is required with balloon protection systems blocking I.C.A. flow with balloons; moreover, with use of filters the carotid flow and the intracranial circulation pattern can be checked at any time. In nine cases we observed a slowing of flow in the I.C.A. distally to the filter, and in six more cases the flow was blocked; interestingly, we observed such slowing or arrest of flow only immediately after balloon inflation for angioplasty. In all these fifteen cases (of which only one, described in figure 4, with a subsequent clinical complication) variable amount of debris was present on the inner surface of the filter membrane after retrieval; thus, we surmise this condition to be due to partial or total occlusion of the filter’s holes by the debris itself. Therefore, when considering the histopathology of the debris (see further), it is our present opinion that the onset of fibrin and platelets on the membrane is provoked or highly favored by blood stagnation during balloon inflation. For this reason, after our initial experiences, we decided to use a more aggressive anti-coagulation protocol during the procedure, never starting balloon dilation of the stent unless the activated clotting time was at least 3 times the baseline (i.e., usually more than 400 seconds); doing so, in the last, consecutive 36 procedures we had only three cases with a small amount of fibrin and platelets on the filters’ surface, without slowing of flow, one case with a complete arrest of flow after balloon inflation due to presence of plaque fragments and cholesterol crystals on the filter’s inner surface and one more case with arrest of flow due to presence of a large amount of fibrin filaments inside the filter. However, this latter case deserves some remarks. First of all, in this case, shown in figure 4, a severe slowing of flow was present in the intracranial circulation at the pre-stenting diagnostic angiography, owing to the presence of both a tight stenosis of the bifurcation and of a tandem, less severe stenotic lesion of the homolateral carotid siphon; for these reasons, surmising that the patient could be at risk for a post-stenting hyperperfusion syndrome, we did not use antplatelets agents before and during the procedure, and this could partially explain the in-filter coagulation. Besides, in all probability the presence of the tandem intracranial stenosis could have aggravated the slowing of flow during the procedure, and consequently the thrombi formation on the filter membrane. The concomitance of the tandem lesion and of the non-aggressive anti-coagulation treatment (before, during and after the procedure) could have been the cause of the subacute occlusion of the stent with subsequent clinical complication. We consider this case a possible procedural mistake of ours: we should have considered the patient protected from a hyperperfusion by the presence of the stenosis of the siphon, and thus use aggressive anticoagulative and hypertensive treatment to prevent stent thrombosis, or should have treated by dilation and stenting also the tandem intracranial lesion to favor the outflow from the stent. In any case, we now keep the presence of a second, stenotic lesion on the same artery as a risk factor for subacute occlusion of the proximal stent owing to poor outflow from the stent.

Histopathologic evaluation of the filters’ content showed fibrin filaments in 16 cases, seven of which with entrapped platelets. We think that such material is a consequence of in-filter coagulation, probably mainly related to the microporous surface. We are greatly surprised to have found atheromatous plaque material on the filter’s membrane after retrieval in only one case. In a recent article [1], analyzing from a histopathologic point of view the material collected inside the filters in 38 endovascular treatments, solid fragments were found on microscopic evaluation in 31 out of 37 specimens, and all detected material was identifiable as of atheromatous nature, mainly of lipid-rich macrophages, cholesterol clefts, calcium and platelets entrapped in fibrin. Fragments were present at macroscopic evaluation in 27 filters (72.9%). Other authors report presence of plaque fragments in 53% out of 83 filters, and these authors use pre-dilation after filter positioning, before stent deploying [25]. In our experience, macroscopic evidence of fragments is reported only in 17 out of 53 cases (32%), and such material proved to be composed of fibrin and platelets in all but one case. We hypothesize that this could be due to a different procedural technique: the authors of the two quoted articles [1-25] used a pre-dilation after filter positioning in 60.5% and 83.7% of cases respectively, while we always deploy a stent without pre-dilation (in our only case with pre-dilation the filter was positioned after it). This therefore seems to confirm that fragmentation of the plaque and debris dislodging is consequent to compression by balloon of the bare plaque, while the bulk of an already deployed stent protects plaque surface and at least partially prevents fragments from dislodging and embolizing. For this reason, and thanks to the very low profile and size of the stents now available, which easily pass through even tight stenoses, we nearly always perform primary stenting without predilation, and consider this a safer technique.

In all cases, we observed a mild degree of I.C.A. spasm after filter positioning. The spasm has been more intense in four cases (figure 2); however, the spasm did not cause any apparent slowing of the intracranial flow and it rapidly resolved after retrieval of the filter without drug administration. Spasm can be reduced by undersizing the filters, thus avoiding an excessively hard contact of the filter basket against the carotid wall; however, we are more concerned by the risk that emboli could pass around an undersized filter. Despite this, we never observed lesions of the carotid inner wall, such as dissections or floating thrombi, after filter retrieval and spasm regression.

Regarding safety of the devices, the four strokes are worth analyzing.

One is due to subacute stent thrombosis, and has already been discussed; this case, like the one in which the patient complained of three episodes of decrease in visual acuity probably due to emboli in the retinal artery, do not seem clearly related to the use of filters. However, theoretically, in these cases
too it cannot be excluded that intimal lesions provoked by the filters, even not detected angiographically (during the procedure) or by US or C.T. (after the procedure), could have contributed to the thrombotic condition and subsequent complication. Such considerations could apply also in the patient who presented a T.I.A. three days after the procedure.

The other two strokes could very likely be due to a hyperperfusion syndrome and not to a hemorrhagic infarction from embolism; ultrasound studies performed immediately after the onset of the stroke showed a good patency of stent and I.C.A., with no evidence of arterial wall dissection. The two patients had suffered the most recent symptoms at least three months earlier, thus they were apparently not at greater risk for reperfusion hemorrhage because of a recent ischemic brain lesion with altered blood brain barrier. Figure 3b shows lack of apposition of the stent to the anterior wall of the common carotid, and this could contribute to thrombo-embolic events; however, in favor of a hyperperfusion are the very early onset of symptoms, with the patient still under heavy anti-coagulation, the immediate C.T. evidence of a brain edematous lesion, the normal sonographic patency of the stent, the hemorrhagic transformation. Hyperperfusion syndrome is more and more frequently reported in the neuroradiological endovascular literature, and such a complication seems to be more frequent after stenting rather than carotid surgery [8, 11, 15, 16, 22, 24, 27, 28], probably because of the heavier anticoagulation medical therapy needed after stent implantation rather than after endarterectomy; thus, it is noteworthy that in the present series the incidence of cerebral hemorrhagic complications post-stenting is as high as 3.8%, among the highest in the literature, while in a previous series of one of the authors [4] no intracranial hemorrhagic complication was observed in 71 treatments, in which only three filters were used, with a standard anticoagulation protocol. It is therefore our opinion that the hemorrhagic complications in this series could be related to the stronger anticoagulation protocol used to prevent in-filter coagulation; this must be confirmed by other observations, however great care is needed until everything is clarified or filters improved. Manufacturers will probably have to combine, developing new devices, more effectiveness and more safety, looking both for the ideal material for the filtering membrane and for the more suitable size of the micropores: a larger size carries the risk of permeability to potentially dangerous emboli, while too small a size carries the risk of in-filter coagulation and consequent arrest of flow (and need of heavy anti-coagulation protocol). Thus an ideal membrane will allow a light anti-coagulation protocol, while being effective in capturing potentially dangerous debris.

Owing to the lack in our study of intra-procedural transcranial Doppler (TCD) monitoring, we cannot exclude the possibility of clinically silent emboli not captured by the filters. However, TCD signals are exceedingly frequent during endovascular procedures, often represented only by air bubbles; thus, to collect really reliable data the same procedure, i.e., stenting with or without filter protection, should be randomly compared in a large numbers of patients. Moreover, the clinical relevance of tiny, TCD detectable emboli, acutely neurologically silent, seems to be low or absent [5-26].

CONCLUSION

In our experience filters proved reliable and easy to use, acting as good diagnostic guide wires and not adding to the procedure a significant amount of technical complication or waste of time; direct lesions of the arterial wall have never been observed, and spasm of the distal I.C.A. also proved rapidly regressive. We collected a small amount of plaque material inside the filter in only one case, thus we have no strong evidence that they are useful to prevent a clinically significant intracranial embolization.

The major technical drawback is coagulation on the filter membrane. This condition, which determines a slowing or block of intracranial flow, can be counteracted by a more aggressive anti-coagulation protocol; however, the two complications with hemorrhagic brain infarction we observed in our present series could be related to the latter.

We observed two other major complications; thus in this series with use of filters the incidence is 7.5%, higher than usually reported in the literature and higher than the one of 1.4% reported by one of the authors in a previous series of 71 patients [4].

Consequently, it is our opinion that safety of filters deserves a more thorough evaluation in larger numbers, and also that great care must be taken in their use before such safety is proved; probably an improvement in materials and construction features of the filters is needed, to eliminate in-filter coagulation, spasm and risk of passage of debris laterally to the device.

RÉFÉRENCES


