A 20-year Experience with Surgical Management of True and False Internal Carotid Artery Aneurysms

R. Pulli [a], W. Dorigo [a,*], A. Alessi Innocenti [a], G. Pratesi [a,b], A. Fargion [a], C. Pratesi [a]

[a] Department of Vascular Surgery, University of Florence, Viale Morgagni 85, 50134 Florence, Italy
[b] Department of Vascular Surgery, University of Rome Tor Vergata, Rome, Italy

WHAT THIS PAPER ADDS?
This study provides an insight of the contemporary management of internal carotid artery aneurysms and pseudo-aneurysms and of its early and long-term results. The growing role of endovascular surgery also in this field of application is underlined.

Aim of the study: The aim of this study was to retrospectively analyse early and late results of surgical management of internal carotid artery (ICA) true and false aneurysms in a single-centre experience.
Materials and methods: From January 1988 to December 2011, 50 consecutive interventions for ICA aneurismal disease were performed; interventions were performed for true ICA aneurysm in 19 cases (group 1) and for ICA post-carotid endarterectomy (CEA) pseudo-aneurysm in the remaining 31 (group 2).
Early results (<30 days) were evaluated in terms of mortality, stroke and cranial nerves' injury and compared between the two groups with χ^2 test.
Follow-up results (stroke free-survival, freedom from ICA thrombosis and reintervention) were analysed with Kaplan–Meier curves and compared with log-rank test.
Results: All the patients in group 1 had open repair of their ICA aneurysm; in group 2 open repair was performed in 30 cases, while three patients with post-CEA aneurysm without signs of infection had a covered stent placed. There were no perioperative deaths. Two major strokes occurred in group 1 and one major stroke occurred in group 2 ($p = 0.1$). The rates of postoperative cranial nerve injuries were 10.5% in group 1 and 13% in group 2 ($p = 0.8$).
Median duration of follow-up was 60 months (range 1–276). Estimated 10-year stroke-free survival rates were 64% in group 1 and 37% in group 2 ($p = 0.4$, log rank 0.5); thrombosis-free survival at 10 years was 66% in group 1 and 34% in group 2 ($p = 0.2$, log rank 1.2), while the corresponding figures in terms of reintervention-free survival were 68% and 33%, respectively ($p = 0.2$, log rank 1.8).
Conclusions: Surgical treatment of ICA aneurismal disease provided in our experience satisfactory early and long-term results, without significant differences between true and false aneurysms. In carefully selected patients with non-infected false aneurysm, the endovascular option seems to be feasible.

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Extracranial carotid aneurysms (ECAs) are uncommon[1] and are usually distinguished in true aneurysms and so-called pseudo-aneurysms.[2–6] While the former are atherosclerotic, the latter are the result of dissection, neck injury or, more frequently, post-carotid endarterectomy (CEA) with patching. Due to the substantial risk of neurological or local complications if untreated, their surgical repair has been recommended,[1,2] and open repair has been described to provide good early and long-term results,[1–4] even if most reports include different aetiologies and approaches with regard to patient anatomy, co-morbidities and physician preference. In the last few years, with the increasing use of endovascular procedures, an endovascular approach has been advocated for these patients, too;[7] however, the applicability of such techniques in this kind of lesions and its effectiveness are still controversial.

The aim of this study was to retrospectively review our experience with open and endovascular treatment of true and post-CEA ECAs in the last two decades, analysing early and long-term results.

* Corresponding author. Tel.: +39 055412029; fax: +39 0554220144.
E-mail address: dorigow@unifi.it (W. Dorigo).
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PATIENT POPULATION AND METHODS

From January 1988 to December 2011, 6830 consecutive interventions (4630 in males and 2200 in females) for extracranial carotid artery disease were performed at our academic institution.

Data concerning these interventions were prospectively collected in a dedicated database whose characteristics have been already described.[8] A post-hoc analysis of this database was performed and 53 interventions for ECA were found: the interventions were performed for true ECA in 19 cases (group 1) and for carotid pseudo-aneurysm in the remaining 34 (31 post-CEA, two iatrogenic and post-traumatic in the remaining one). For homogenisation sake, we excluded patients with traumatic lesions from the study and we considered only the 31 patients with post-CEA aneurysms (group 2).

The two groups of patients were compared in terms of demographic data, common risk factors for atherosclerosis and co-morbidities. Risk factors and co-morbidities included arterial hypertension (defined as blood pressure greater than 140/85 mmHg or the need for anti-hypertensive drugs), hyperlipaemia (defined as both triglycerides and cholesterol values $>$200 mg dl), coronary artery disease (history of myocardial infarction, angina, previous coronary revascularisation), diabetes mellitus (defined as the need for specific drugs to maintain metabolic control) and peripheral arterial disease (ankle/brachial index $<$0.9).

Patients in group 1 were more frequently females and were younger than patients in group 2; moreover, there was a trend towards a higher percentage of other aneurysms in different sites among them. Patients in group 2 had more frequently the presence of common risk factors for atherosclerosis and of cardiovascular co-morbidities, even if the difference with group 1 was not statistically significant (Table 1).

Patients were considered to be asymptomatic in the absence of neurological symptoms (transient ischaemic attack (TIA) or stroke) within 6 months from the intervention.

All these patients underwent duplex scanning of extracranial vessels and computed tomography (CT) of cerebral parenchyma. Until 2002, all patients used to undergo digital subtraction angiography of the arch vessels, whereas angio-CT scan of the intra- and extracranial vessels was performed in the last decade. In patients of group 2, a careful assessment for the presence of clinical (the presence of fever, of abscesses or fistulae with purulent secretion at the site of CEA) and radiological (pericarotid liquid or abscess) signs of infection was performed. In these patients, white blood cell (WBC) count and blood cultures were performed; moreover C-reactive protein (CRP) and, in last few years, pro-calcitonine levels were determined. The presence of an aneurysm exceeding 1.5 cm in diameter was considered an indication for treatment in group 1, while in the presence of post-CEA pseudo-aneurysms the intervention was performed regardless of both the diameter of the lesion and the presence of symptoms. Due to the frequent presence of preoperative symptoms and their high embolic potential, we preferred to treat all the patients in group 1 with open surgery; in group 2, we used an endovascular approach in selected patients once infection had been excluded. The interventions, both open and endovascular, were performed in the operating room. Patients operated on with open procedure had either general anaesthesia with cerebral monitoring with somatosensory evoked potentials (SEPs) and selective shunt insertion on the basis of SEPs abnormalities[9] or Cooperative Patient General Anaesthesia (CoPaGeA)[10] with clinical neurologic monitoring during carotid clamping and selective shunt insertion. Intra-operative medical treatment consisted of intravenous sodium heparin administration (30 IU kg^{-1}) at carotid clamping. The kind of arterial reconstruction and the material for reconstruction were chosen on the basis of the length and of the morphology of the aneurismal lesion and of status of arterial wall. A sample of the arterial wall was routinely excised and sent for pathological and bacteriological examinations. At the end of the intervention completion angiography was routinely performed. Patients operated on with endovascular procedure had local anaesthesia and clinical monitoring of cerebral functions. A cerebral protection device was selectively used on the basis of the morphological aspects of the lesion, the presence of thrombus and the type of the stent used. Intra-operative medical treatment consisted of intravenous sodium heparin administration (5000 IU) at the beginning of the procedure.

Postoperative medical treatment consisted of single or double anti-platelet treatment (acetylsalycilic acid, 150 mg once a day, and/or ticlopidine, 250 mg twice a day), at surgeon's discretion. In all the patients statin therapy was continued indefinitely.

Neurological evaluation at 30 days was independently performed in all the patients by an experienced neurologist, who assessed the presence of minor and major strokes. Minor stroke was defined as any postoperative neurological event lasting more than 24 h with recovery in the following days or weeks without or with minimal residual functional impairment. Major stroke was defined as any postoperative neurological event lasting more than 24 h with residual invalidity and/or inability. Otolaryngologist evaluation at 30 days in terms of vocal cords' motility and cranial nerves' injury was also performed in all the patients by a phoniatrist.

Perioperative ($<$30 days) results of interventions were analysed and compared in terms of stroke, death and cranial nerves' injuries with χ^2 test and Fisher's exact test, when necessary.

Table 1. Patients' demographic data, risk factors and comorbidities.

	Group 1 (19 pts.)	Group 2 (31 pts.)	*p*
Female gender	9 (47%)	4 (13%)	0.007
Median age (years)	66	72.8	0.1
Hyperlipaemia	7 (36%)	16 (51%)	0.3
Diabetes	2 (10.5%)	9 (29%)	0.1
Arterial hypertension	14 (73%)	27 (87%)	0.1
Coronary artery disease	4 (21%)	11 (35%)	0.2
Peripheral artery disease	5 (26%)	13 (42%)	0.3
Smoker or past smoker	8 (42%)	17 (55%)	0.3
Other aneurysms	5 (26%)	3 (10%)	0.1

Follow-up was performed at 1, 3, 6, 12 months, and yearly thereafter by clinical examination and duplex scan. Patients who did not accomplish follow-up examinations had telephone interviews. During the telephone interview, some points were assessed: patient's survival and cause of death, if known; neurological events and their time of appearance. Moreover, the patients were asked to report the results of their last duplex ultrasound control, wherever it was performed, to assess the status of operated internal carotid artery. Additional data regarding long-term survival and major cardiovascular events were obtained from the Regional Health Care database. Follow-up data were analysed by life-table analysis (Kaplan–Meier test) in terms of stroke, death and patency of the operated internal carotid artery and the results in the two groups were compared by means of log-rank test.

RESULTS

Clinical characteristics

Neurological symptoms were observed in nine cases in group 1 (47%) and in four cases in group 2 (13%; $p = 0.007$). Symptoms consisted of TIAs in seven patients in group 1 and in three patients in group 2, whereas three patients (two in group 1 and one in group 2) suffered from a previous major stroke. Six patients of group 2 had an urgent intervention due to the sudden enlargement of the pulsatile neck mass. Among the patients operated on for post-CEA pseudoaneurysm, the mean time from the first operation was 3 years (range 1 month–12 years; Fig. 1). All of them had had prosthetic patch closure at the time of the first operation. The mean diameter of the aneurysm was 22.6 $\pm$ 0.7 mm in group 1 and 28 $\pm$ 1 mm in group 2 ($p = 0.04$).

Operative management

All the patients in group 1 had open repair of their ICA aneurysm; in group 2 open repair was performed in 28 cases, while three patients underwent endovascular treatment. In group 1, general anaesthesia with SEP monitoring was used in 16 cases, while three patients underwent CoPaGeA with clinical monitoring.

Interventions consisted of aneurismectomy with end-to-end anastomosis in six cases, in aneurismal resection with expanded polytetrafluoroethylene (ePTFE) patching in three cases and in carotid bypass in the remaining 10 patients. All the bypasses except one were performed using ePTFE, while the remaining patient had an autologous saphenous vein bypass.

In group 2, among patients undergoing open surgical treatment, general anaesthesia was used in 26 cases and CoPaGeA in two. Interventions consisted of carotid bypass in 14 cases (nine with ePTFE and five with autologous saphenous vein), aneurismectomy with arterial suture in eight cases and in aneurismal resection with patching in six cases (ePTFE in five cases and autologous saphenous vein in the remaining one).

Among the three patients in group 2 treated with an endovascular procedure, none had clinical or radiological signs of infection. All the patients had normal WBC count and blood cultures were negative for infection; moreover, CRP and pro-calcitonine levels were normal. In these patients (post-CEA pseudo-aneurysms occurring after 3 months, 4 and 6 years from the primary intervention, respectively) we used a stent graft (Viabahn, Gore, Flagstaff, AZ, USA). In two cases, a cerebral protection device (Epi-filter EZ 6.5, Boston Scientific, Natick, MA, USA) was used.

Postoperative medical treatment consisted of single anti-platelet therapy in 17 patients in group 1 and in 28 patients in group 2; the remaining patients had double anti-platelet treatment.

Perioperative results (Table 2)

There were no perioperative deaths. Three major strokes occurred, two in group 1 and one in group 2. One patient in group 1, operated on with ePTFE carotid bypass under general anaesthesia without SEP modifications, developed 6 h after the end of the intervention a contralateral hemiplegia with duplex ultrasound finding of acute thrombosis of the bypass. Successful surgical thrombectomy was immediately performed, however without neurological improvement. The other patient with true aneurysm, operated on with autologous vein carotid bypass under general anaesthesia with shunt insertion after 12 min of carotid clamping due to critical SEP reduction, showed contralateral hemiplegia at awakening with normal patency of the bypass and of internal carotid artery. The third stroke occurred in

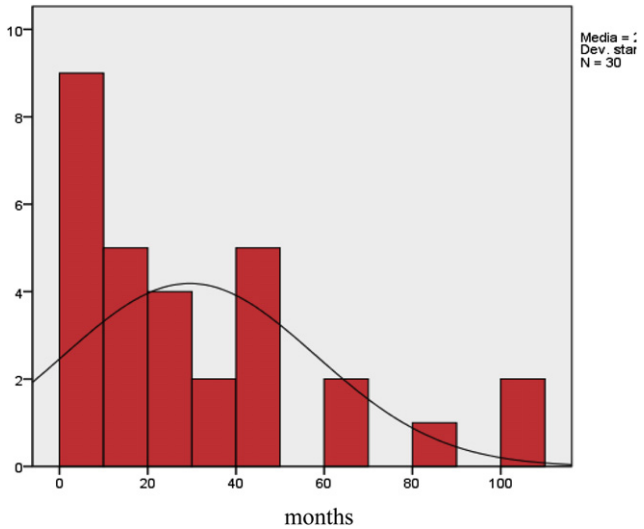


Figure 1. Time distribution of post-CEA pseudoaneurysms with respect to primary intervention.

Table 2. Perioperative (<30 days) results.

	Group 1 (19 pts.)	Group 2 (31 pts.)	*p*
- TIA	–	–	–
- Minor stroke	–	–	–
- Major stroke	2	1	0.1
- Death	–	–	–
Thrombosis	2[a]	1	0.8
Cranial nerve injuries	2	4[b]	0.8

[a] In one case symptomatic.
[b] In two cases persistent.

a patient of group 2, who had undergone an ePTFE bypass under general anaesthesia without SEPs modifications, and developed aphasia and contralateral upper limb plegia with normal patency of the bypass and of internal carotid artery.

One patient in group 1 developed an asymptomatic occlusion of an ePTFE bypass detected at pre-discharging duplex scanning, which was medically managed; another patient in group 2, operated on with a vein bypass for infected post-CEA pseudo-aneurysm, suffered from an asymptomatic thrombosis detected at 30-day control, associated with pericarotid abscess. Despite specific antibiotic treatment for 3 months, the abscess persisted and required bypass removal and common carotid artery ligature.

Overall 30-day stroke and death rate was 6% (three cases, two in group 1 and one in group 2; $p = 0.1$).

In six cases (two in group 1 and four in group 2) postoperative cranial nerve injuries were recorded. The two patients in group 1 had a transient deficit of the hypoglossal nerve, with complete recovery at 30-day control. In group 2 all the four patients had ipsilateral vocal cord paralysis, transient in two cases and permanent in the remaining two. Cumulative cranial nerve injury rate was 12% (10.5% in group 1 and 13% in group 2; $p = 0.8$).

Histopathological examinations in patients of group 1 showed that the aneurysms were atherosclerotic in 13 cases, while six patients had fibrodysplastic lesions.

Bacteriological examination on the specimens obtained from the patients of group 2 operated on with open surgery showed the presence of infection in five cases; St. Aureus was the involved bacteria in all the cases. None of the these patients had showed any sign of early infection at the time of the primary intervention.

Follow-up results

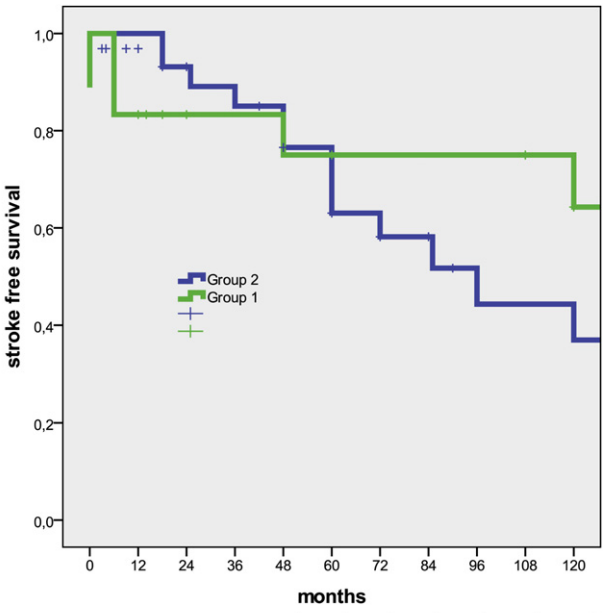
Median duration of follow-up was 60 months (range 1–276); 48 patients (96%) had at least one postoperative follow-up examination. During follow-up 19 deaths occurred, six in group 1 and 13 in group 2. The causes of death were cancer in four cases, ipsilateral stroke in one case, acute myocardial infarction in two cases, cerebral haemorrhage in one case and chronic renal failure in one case. One patient died of a ruptured abdominal aortic aneurysm, while another one committed suicide. Another patient, who had suffered a perioperative stroke, died at the second postoperative month due to respiratory complications. In seven patients, the cause of death was unknown.

The fatal stroke occurred in a patient of group 1 after 4 years from the intervention in the presence of a patent internal carotid artery.

Estimated 10-year survival rates were 66.2% in group 1 (SE 0.12) and 36.4% in group 2 (SE 0.11; $p = 0.3$, log rank 0.8).

One non-fatal contralateral stroke occurred in a patient in group 1 as a complication of a contralateral CEA performed 6 months after the primary intervention.

Estimated 10-year stroke-free survival rates were 64.3% in group 1 (SE (standard error) 0.12) and 36.9% in group 2 (SE 0.11; $p = 0.4$, log rank 0.5; Fig. 2).



Months	0	30	60	90	120	S.E.
Group 1 (n. at risk)	16	10	8	8	6	0.12
Group 2 (n. at risk)	29	22	17	7	5	0.11

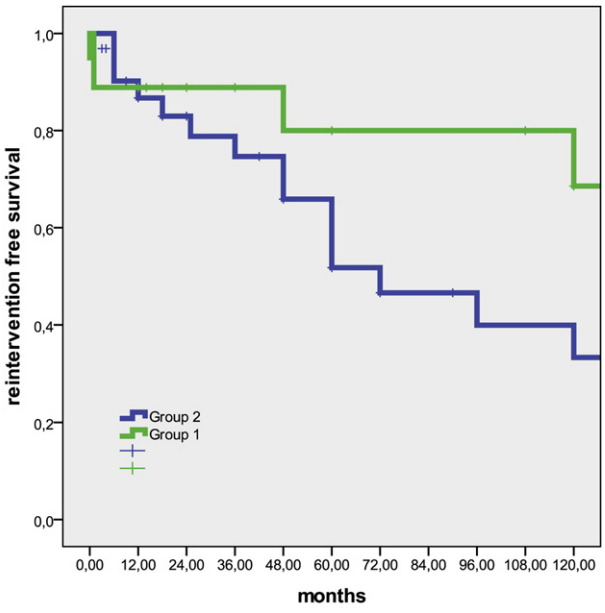
Figure 2. Kaplan–Meyer curves for stroke free survival in both groups with number of patients at risk.

One asymptomatic thrombosis occurred at 2 months in a patient in group 2 and it was medically managed; thrombosis-free survival at 10 years was 66% in group 1 and 34% in group 2 (SE 0.11 in both groups; $p = 0.2$, log rank 1.2).

There was recurrence of post-CEA pseudo-aneurysm in two patients operated on for non-infected lesions; in one case, previously treated with ePTFE patch, a vein bypass was performed at 12 months, while, in the other patient, who had had a ePTFE patch too, endovascular treatment with a bare metal stent (Wallstent, Boston Scientific, Natick, MA, USA) was performed. Reintervention-free survival rates at 10 years were 68.6% and 33%, respectively (SE 0.10 in both groups; $p = 0.2$, log rank 1.8; Fig. 3). No dilatations of saphenous vein bypasses or patches were noticed during follow-up.

DISCUSSION

ECAs represent a rare disease; however, under the definition of carotid aneurysm different situations are comprised with completely different aetiologies and solutions. In fact, the true carotid aneurysm is mainly due to an atherosclerotic localisation at that level, while carotid pseudo-aneurysms either are the result of a neck injury or can represent the complication of a vascular or a non-vascular surgery of the neck. Less frequently a true aneurysm can be caused by an arterial fibrodysplasia; under this term are included a heterogeneous group of non-atheromatous vessel wall derangements, which are suggested among the aetiological mechanisms of spontaneous carotid dissection. Aneurysms can develop after a previous spontaneous dissection of the internal carotid artery, and the rise in their frequency has



Months	0	30	60	90	120	S.E.
Group 1 (n. at risk)	17	11	8	8	6	0.10
Group 2 (n. at risk)	29	19	13	8	5	0.10

Figure 3. Kaplan—Meyer curves for reintervention free survival in both groups with number of patients at risk.

been attributed to the systematic surveillance of the patients (angiography or CT angiography) with an episode of dissection.[11] Also in our series, even if the percentage of atherosclerotic aneurysms was higher, we recorded six cases of fibrodysplastic lesions, even in the absence of a history of previous recognised dissections.

Looking at the distribution of these aetiologies in main surgical series,[1–8] pseudo-aneurysms are the most frequent, and this was also in our experience where the main cause of pseudo-aneurysm was the complication of a previous CEA. The aetiology of post-CEA pseudo-aneurysm formation includes suture failure, degeneration of arterial wall or patch material and infection. Infection as a result of post-CEA pseudo-aneurysm is uncommon as the incidence of post-CEA infection is as low as 0.025—0.625%, mostly caused by staphylococci.[12] Also in our series the percentage of infections was low and St. Aureus was involved in all the cases.

While true aneurysms are often responsible for cerebral embolism causing neurological impairments, pseudo-aneurysms more frequently cause dysfunction of postganglionic sympathetic nerve fibres and cranial nerves due to compression or distension. Moreover, in our series we had a higher frequency of preoperative neurological symptoms in patients operated on for true aneurysms.

Open surgical repair with revascularisation after aneurismal resection is the preferred treatment method in most published reports; arterial continuity can be restored by aneurismectomy with end-to-end anastomosis, aneurismal resection with prosthetic or autologous patching and carotid bypass; and carotid artery ligation is reserved as the last resort or in instances of aneurysm rupture,[1] and this was also in our experience. In the last few years, the use of an endovascular approach has been suggested also for patients with ECA, and a significant improvement in perioperative results with this technique has been reported.[7] As generally observed, true aneurysms have often a significant amount of thrombus inside and frequently cause preoperative neurological symptoms; for this reason in our experience we preferred to treat them with open surgical repair; moreover, we were cautious with the endovascular approach also in patients with post-CEA pseudo-aneurysms, and we limited it to non-infected lesions, which still represent a challenge for the surgeon, even if skilled, with significantly higher reported mortality and morbidity rates than those obtained in primary interventions. We used a covered stent in all our cases; in three of them a Viabahn (Gore, Flagstaff, AZ, USA) stent was deployed. In addition to its ease of deployment and proven track record in peripheral vessels, it has a lower profile and greater flexibility than other covered stent grafts.[13,14] Furthermore, a self-expanding stent is more suitable for the use in a very delicate artery, such as internal carotid vessel. Our results with this technique were encouraging: we achieved a very good result in all the cases and all the patients were discharged stroke-free; however, the limited number of patients treated does not allow drawing firm conclusions on this issue.

Our cumulative 30-day results compare well with the results reported in the literature;[1] there was a trend towards poorer perioperative results in patients operated on for true aneurysms, whose explanation is rather difficult. One can suppose that the increased frequency of preoperative symptoms among these patients could contribute to the increased perioperative risk; moreover, the presence of parietal thrombosis on aneurismal surface can be the cause for intra-operative cerebral embolism, as it probably occurred in one of the two postoperative strokes in group 1. All the postoperative strokes occurred in patients operated on with carotid bypass; this is in our opinion an interesting finding. Usually, we observe that patients needing a carotid bypass have a largely diseased arterial wall, with long and complex lesions, and this can represent an adjunctive risk factor for perioperative complications.

Also the rate of postoperative cranial nerve injuries was in range with those reported in the literature,[15] and in most cases they were transient. The rate was similar in the two groups; however, two patients in group 2 had a permanent injury, suggesting that both the presence of the haematoma and the redo intervention can contribute to the development of more severe nerve lesions. In these cases, endovascular treatment could offer a possible advantage over open repair; however, the presence of neck haematoma could lead to the development of cranial nerve injuries regardless of the effectiveness of endovascular procedure, and, in some situations, the drainage of haematoma is mandatory.

During follow-up, whose mean duration was 5 years, a value similar to the main published series, we did not find significant differences between the two groups in terms of survival and stroke-free survival rates; however, there was a trend towards poorer results in patients operated on for

pseudo-aneurysm (possible type 1 statistical error). One can suppose that at a longer follow-up time with a larger number of patients and events this difference could become significant, and this finding is probably related to older age and to the higher percentage of co-morbidities and risk factors at the time of the intervention among patients of group 2, even if not statistically significant.

Data concerning reinterventions during follow-up confirmed this trend; no reintervention was needed among patients with true aneurysm, while in two cases a recurrent pseudo-aneurysm was detected. This is in our opinion an interesting data; in fact, there are few reports in the literature detailing late reinterventions,[16,17] and the possibility of development of recurrence of pseudo-aneurysms, even in the absence of clinical and laboratory signs of infection, should be taken into account, suggesting the need for prolonged and careful surveillance in those patients.

This study has several limitations: it is a retrospective monocentric study, with a limited number of patients and events, and a still limited number of endovascular procedures. In spite of these biases, however, in our opinion it provides an insight of the contemporary management of this uncommon disease and of its early and long-term results.

CONCLUSIONS

Surgical treatment of ICA aneurismal disease provided in our experience satisfactory early and long-term results, even if poorer than those obtained in patients operated on for CEA for obstructive disease in contemporary series. In selected patients with non-infected false aneurysms, the endovascular option is feasible and seems to provide good and durable results.

CONFLICT OF INTEREST/FUNDING

None.

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