Extracranial internal carotid artery aneurysms: Results of a surgical series with long-term follow-up

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Purpose: The purpose of this study was to analyze mode of presentation, surgical treatment, and early and long-term results of a series of extracranial internal carotid artery aneurysms (EICAA).

Methods: A retrospective analysis was performed on all cases treated for EICAA in a single institution from March 1974 to March 1995. Patient follow-up was obtained by a surveillance protocol, with duplex scanning performed 3 months after surgery and yearly thereafter.

Results: Twenty-four EICAA in 20 patients were treated over a 21-year period. The cause was fibromuscular dysplasia in 12 cases (50%), nonspecific "atherosclerosis" in nine (37.5%), previous carotid artery surgery in two (8.3%), and trauma in one case (4.1%). Neurologic symptoms were present in a total of nine cases (37.5%) and were hemispheric in seven (29.1%) and nonhemispheric in two (8.3%). Operative techniques were performed with patients receiving general anesthetic and included aneurysm excision with internal carotid artery reanastomosis (8 cases [33.3%]) or reimplantation onto the external carotid artery (1 case [4.1%]); interposition graft (10 cases [41.6%], 7 veins, 3 polytetrafluoroethylene) or simple aneurysmectomy and closure of the wall defect either with (3 cases [12.5%]) or without (2 cases [8.3%]) a patch. Elective surgery was performed in 22 cases, with a 0% mortality rate and 4.5% stroke rate. Emergency operations were performed in two cases of ruptured aneurysms (one spontaneous and one iatrogenic); one patient (50%) died. Cranial nerve morbidity occurred in five cases (20.8%). Mean follow-up was 96.7 ± 88.15 months (range 4 to 240 months) and included 2 of 7 (28%) complications in saphenous vein grafts, 1 (4.1%) late transient ischemic attack, and a recurrent aneurysm after 19 years.

Conclusions: Symptoms and potential complications caused by EICAA suggest a broad surgical indication. EICAA can be treated safely because of the good early and long-term results. (J VASC SURG 1996;23:587-95.)

Extracranial internal carotid artery aneurysms (EICAA) are uncommon lesions with variable causes. Few large surgical series[1-7] have been reported in the literature since the first report of a case of aneurysm ligation by Sir Astley Cooper[8] in 1805, and several aspects of this disease still need to be completely analyzed and discussed. The exact incidence of this disease compared with the more common carotid artery occlusive disease and other peripheral aneurysms is seldom reported. A precise definition for carotid artery aneurysm is difficult to establish because of the physiological dilation of the bulb. Consequently, operative indications for borderline asymptomatic lesions are not clear. Another point that requires clarification is the cause of these aneurysms, which are not "atherosclerotic" in a significant percentage of cases. Finally, data on long-term results of surgical treatment are sparse.

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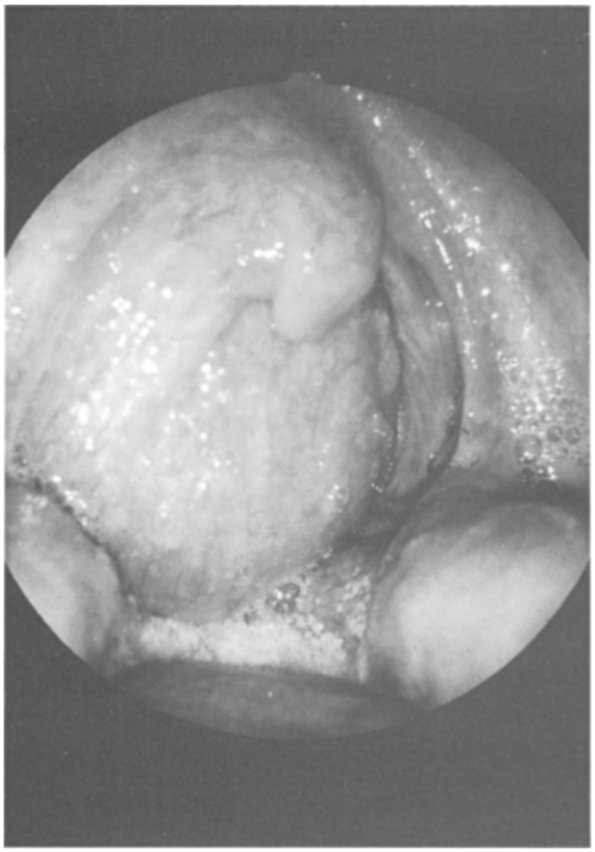

Fig. 1. Intralaryngeal aspect of EICAA protrusion.

We have reviewed our surgical series over a 21-year period with the aim of evaluating these issues.

MATERIAL AND METHODS

All patients with an aneurysm involving the extracranial internal carotid artery (ICA) seen in our Institution from March 1974 to March 1995 have been considered. Aneurysms limited to the external carotid artery were excluded. Patient data have been retrospectively collected with particular attention to epidemiologic characteristics, risk factors, clinical presentation, and treatment. All diagnostic images available were retrieved and evaluated. In particular, angiography and computed tomography (CT) scanning served to precisely define the diameter of the aneurysm to comply with the current definition of carotid artery aneurysm.[9]

Postoperative results were reviewed, and late outcome was obtained by our follow-up program, which is based on duplex examination results obtained 3 months after surgery and yearly thereafter.

Histologic specimens of all available cases were reviewed blindly to define the cause of the aneurysm, and these findings were compared with the gross structure of the aneurysm described in the surgical notes.

Statistical analysis was by chi square or independent t testing, with significance set at $p < 0.05$.

RESULTS

Incidence, epidemiologic pattern, and clinical presentation. During the study period 24 EICAA have undergone operation in 20 patients. This results in a 1.8% incidence of 1324 carotid artery interventions performed in the same period.

Most patients (13 [65%]) were women, and the mean age was 54.8 ± 16.7 years. Other epidemiologic data include white race (20 patients [100%]), history of smoking (16 patients [80%]), history of hypertension (6 patients [30%]), and history of hypercholesterolemia (2 patients [10%]). No patient in this series had diabetes mellitus.

A pulsatile neck mass was present in 19 cases (79.2%). Two of eight (25%) patients had an intralaryngeal protrusion of the aneurysm (Fig. 1). Diagnostic procedures included arteriography in 23 cases (95.8%) and CT scanning of the neck in 10 (41.7%). The diameter of the aneurysm ranged between 1.2 to 5.8 cm. Neurologic symptoms were present in a total of nine cases (37.5%) and were hemispheric in seven (29.1%) and nonhemispheric in two (8.3%) (Table I).

Cause. Twenty-one (87.5%) EICAA were primary aneurysms (Fig. 2). Twelve EICAA (50%) were caused by fibromuscular dysplasia (FMD) in this series. Three (25%) of them were either bilateral (two cases) or recurrent (one case). The recurrent aneurysm developed 19 years after the original one (Fig. 3).

There were nine (37%) degenerative "atherosclerotic" aneurysms[9]; none of them was either bilateral or multiple. One of them (11.1%) was associated with a 4.3 cm abdominal aortic aneurysm.

Demographic data were similar in the fibromuscular and atherosclerotic groups (Table I), except for age, which was significantly younger in the FMD group ($p = 0.03$). The difference between bilateral/recurrent aneurysms was not significantly different in the two groups by chi-squared analysis ($p = 0.1$). Structure of primary EICAA according to cause is summarized in Table II. Elongation of the ICA causing either kinking or tortuosity was present in 8 of 12 (67%) fibrodysplastic EICAA and in one of nine atherosclerotic EICAA (Table II). This difference was significant ($p < 0.01$).

Three aneurysms (12.5%) in this series were

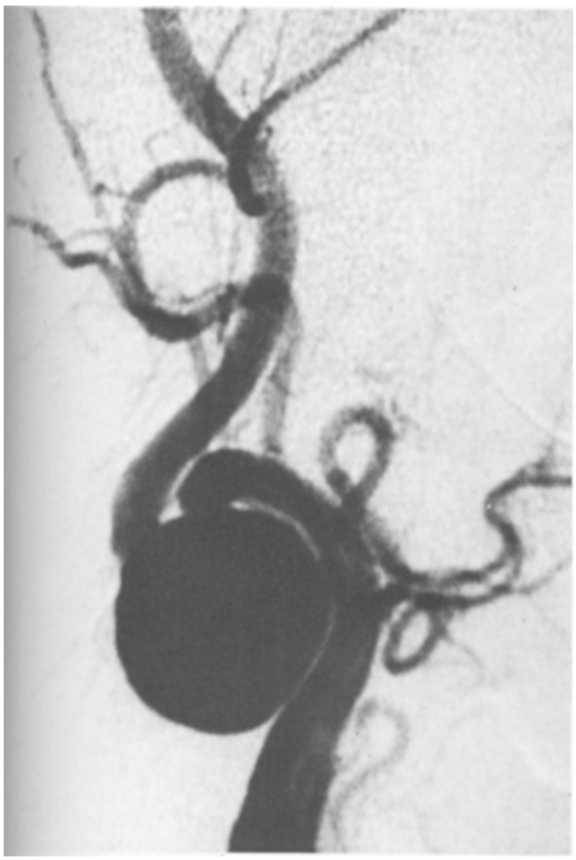

Fig. 2. Angiography of saccular EICAA involving ICA distal to bifurcation. This aneurysm, dysplastic in nature, was treated by excision and end-to-end anastomosis of two ICA stumps.

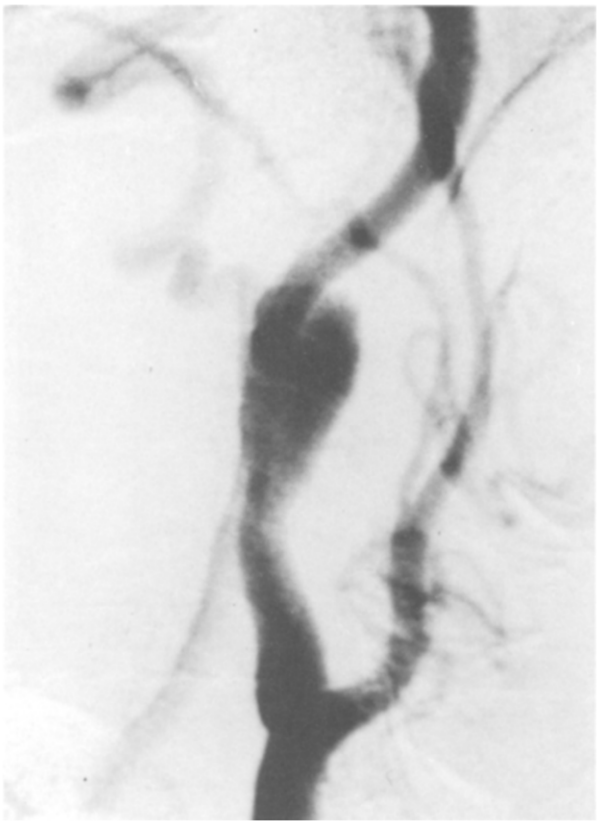

Fig. 3. Angiogram of recurrent EICAA. This aneurysm developed distally to saphenous vein interposition graft implanted 19 years before for replacing FMD EICAA and was successfully treated with PTFE interposition graft.

caused by either trauma or previous surgical procedure. One pseudoaneurysm developed in a 16-year-old girl as a result of blunt trauma in the neck. Two aneurysms occurred in another patient after he underwent two surgical procedures. This patient, a 48-year-old man with hypertension, had undergone carotid endarterectomy 2 years before in a different institution. An aneurysmal degeneration of the saphenous vein patch developed after 2 years and was treated at our institution with replacement of the involved segment of the carotid artery with an interposition saphenous vein graft. This graft subsequently underwent repeated aneurysmal dilation and was finally replaced with a polytetrafluoethylene (PTFE) graft after only 4 months.

No associated cerebral aneurysms were detected in this series.

Operative details. The standard carotid approach anterior to the sternomastoid muscle was used in all patients. No aneurysm localization was so high that it required additional maneuvers (i.e., mandibular subluxation, nasotracheal intubation) to achieve complete distal control.

On the basis of the structure of the EICAA, different surgical techniques were used. Nine cases (37.5%) were managed by removing the aneurysm and directly transposing the ICA to the proximal ICA (5 cases, Fig. 4, *A*), to the common carotid artery (3 cases, Fig. 4, *B*), or to the external carotid artery (1 case, Fig. 4, *C*). This technique was particularly feasible when a saccular aneurysm or an elongation of the ICA was present. An interposition graft (7 veins, 3 PTFE 6 mm diameter) was used in 10 cases (41.6%), usually for fusiform EICAA. Five aneurysms were treated with aneurysmectomy and closure of the wall defect either with (3 cases [12.5%]) or without (2 cases [8.3%]) a patch (Table II).

Two EICAA (8.3%) underwent operation on an emergency basis. One atherosclerotic EICAA ruptured spontaneously. The patient, an 83-year-old woman, experienced loss of consciousness, followed by hemiparesis and laterocervical pain. Emergency

Table I. Differences in epidemiologic pattern in 21 primary cases of EICAA according to cause

	Atherosclerosis (n = 9)	*FMD (n = 12)*
Male sex	3 (33.3%)	3 (25%)
Age (mean ± SD)	66 ± 10.9*	51.7 ± 15.4*
Race, white	9 (100%)	12 (100%)
History of smoking	5 (55.5%)	9 (75%)
Hypertension	2 (22.2%)	2 (16.7%)
Diabetes mellitus	0	0
Hypercholesterolemia	2 (22.2%)	0
Hemispheric symptoms	3 (33.3%)	4 (33.3%)
	2 transient contralateral upper limb weakness, 1 transient dysphasia	3 transient contralateral upper limb weakness, 1 transient dysphasia
Nonhemispheric symptoms	1 (11.1%)	1 (8.3%)
	Recurrent vertigo and diplopia	Recurrent vertigo
Multiple (either bilateral or recurrent)	0†	3 (25%)†
Association with other aneurysms	1 (11.1%)	0

*$p = 0.03$.
†p = NS.

Table II. Surgical treatment in EICAA according to structure, cause, and location

	ICA + CCA	*ICA only*	*Technique*
Primary			
Fusiform	2 ATS, 2 FMD	3 ATS, 2 FMD	3 SV grafts, 1 PTFE graft, 2 aneurysmectomies + patch (1SV,1 PTFE patch), 1 aneurysmectomy, 2 transpositions
Fusiform + tortuosity	—	4 FMD	2 SV grafts, 2 transpositions
Saccular	—	3 ATS	1 SV graft, 1 aneurysmectomy + jugular vein patch, 1 transposition
Saccular + tortuosity	—	1 ATS, 4 FMD	4 Transpositions, 1 PTFE graft
Secondary			
Fusiform	1 (patch dilation)	1 (dilation of SV graft)	1 SV graft, 1 PTFE graft
Saccular	—	1 (trauma)	Aneurysmectomy

ATS, Atherosclerotic; *SV*, saphenous vein; *CCA*, common carotid artery.

angiography (Fig. 5) showed a ruptured EICAA that was successfully treated with aneurysmectomy and transposition of the distal ICA to the external carotid artery (Fig. 4, *C*). No residual neurologic deficit was evident after operation. The second EICAA was an iatrogenic lesion of a true EICAA mistaken for a neck neoplasm during an ear, nose, and throat surgical procedure. Despite prompt vascular surgical intervention, the patient had development of a postoperative massive stroke and subsequently died.

All patients underwent operation receiving general anesthetic; cerebral function during carotid artery clamping was monitored with stump pressure measurement until 1988 (14 cases, 58.3%) or somatosensory evoked potential in successive cases (10 cases, 41.7%). The use of a shunt was believed to be necessary (stump pressure <50 mm Hg; somatosensory evoked potential <50% from baseline) in four cases (16.6%).

Early results. No deaths were observed in the 22 cases treated with operation on an elective basis. One patient—who underwent direct ICA reimplantation for an atherosclerotic aneurysm—had development of a postoperative stroke with hemiplegia and aphasia after early thrombotic occlusion of the ICA. At reintervention a distal dissection of the ICA was found and treated with thrombectomy and endarterectomy. Only minimal clinical improvement occurred. The central neurologic morbidity rate was 4.5%.

One patient treated on an emergency basis had a massive postoperative stroke and subsequently died, for a mortality/morbidity rate of 50% in the emergency cases category .

Overall, transient cranial nerve deficits occurred in five cases (20.8%) and involved the facial (one case), the hypoglossal (one case), the hypoglossal and superior laryngeal (one case), and the superior laryngeal nerve alone (two cases). All these deficits caused only

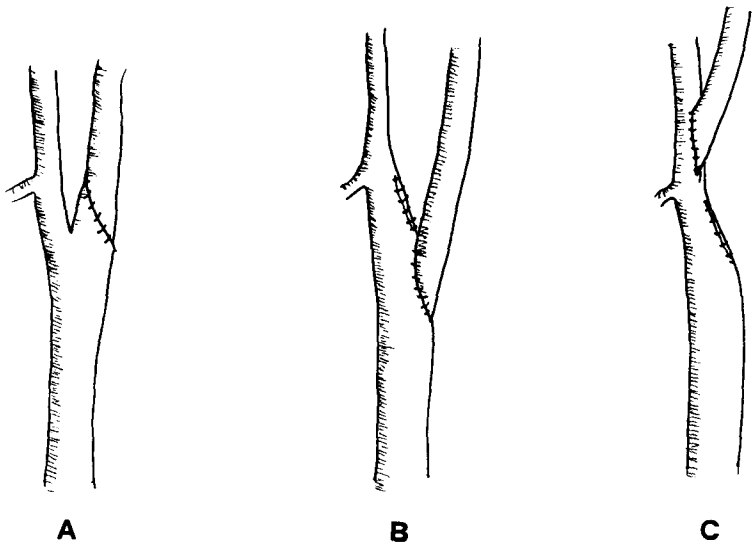

Fig. 4. Scheme of ICA transposition techniques. **A,** Internal-to-internal carotid artery. **B,** Internal-to-common carotid artery. **C,** Internal-to-external carotid artery. This latter technique was performed in case shown in Fig. 5.

minor discomfort to the patient and resolved in 1 to 6 weeks.

Follow-up. Mean follow-up in this series was 96.7 ± 88.15 months (range 4 to 240 months) and was obtained by duplex scanning. Before duplex scanning was available, continuous-wave Doppler scanning was used; however, all patients had at least one duplex scan obtained during their follow-up. Four patients (20%) were eventually lost to follow-up after 48, 72 (two cases), and 120 months. As reported earlier, one saphenous vein graft underwent aneurysmal dilation and needed replacement 4 months after implantation. Another saphenous vein graft developed a significant stenosis after 5 years and was successfully treated with patch angioplasty. Thus the complication rate in saphenous vein grafts was two of seven (28%). One patient had a transient ischemic attack (TIA) ipsilateral to the operative site 2 years after surgery with no lesions detectable at CT scanning nor subsequent sequelae. As reported earlier, one patient had recurrence of a primary FMD aneurysm distal to the site of previous ICA reimplantation 19 years after surgery (Fig. 3). This was successfully treated through an interposition PTFE graft. PTFE grafts had a mean follow-up of 20 ± 12 months with no complications.

DISCUSSION

The incidence of EICAA in the literature is reported in the range of 0.1% to 2% of all carotid artery procedures.[1,7,10,11] In other articles, EICAA were reported to be 0.4% to 1% of all arterial aneurysms[1,12] or 4% of peripheral aneurysms.[13] These figures are in part similar to those found in this series (1.8%) of carotid artery procedures.

Although these data have been available for a long time, no clear definition of EICAA is present in the literature. Although the problem does not exist for distal ICA aneurysms, a dilation of the carotid bulb may simply represent a physiological enlargement of the bifurcation. The accepted definition for "aneurysm" is "an artery having at least a 50% increase in diameter compared to the expected normal diameter of the artery,"[9] but dilation of the carotid bulb may lead to difficulty in interpretation, especially if one tries to define precisely the end of the bulb and the beginning of the ICA. For that reason, deJong et al.[5] proposed to define EICAA as a bulb dilation greater than 200% of the diameter of the ICA or 150% of the diameter of the common carotid artery. These are the criteria we have used in this series; we believe that, in the absence of further data on the natural history of these lesions, they should be used for surgical indication. Four patients had aneurysms of the carotid bulb, whereas 17 of 21 (81%) primary aneurysms were limited to the ICA in our series (Table II).

The reported mean values for carotid bulb diameter are 0.99 ± 0.10 cm for men and 0.92 ± 0.10 for women,[14] and the physician should compare mea-

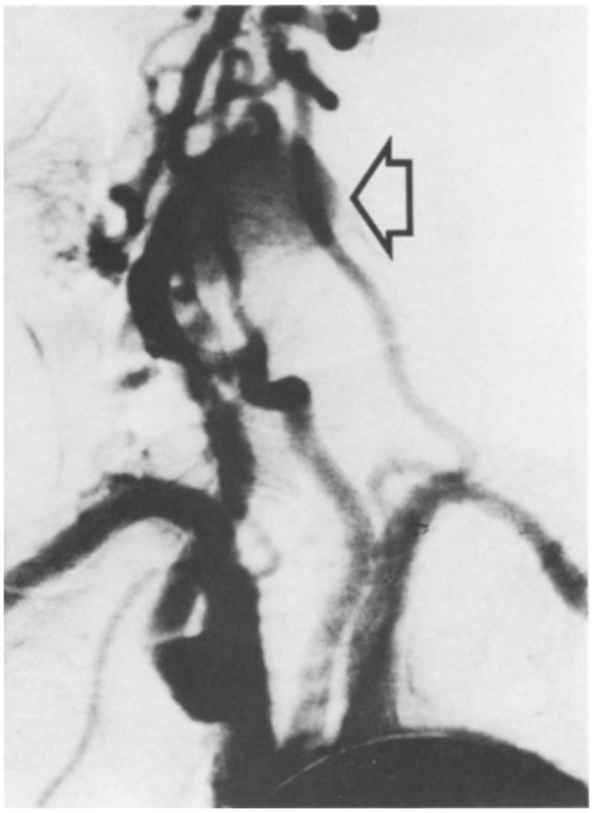

Fig. 5. Angiogram of ruptured EICAA *(arrow)*. This was "atherosclerotic" aneurysm that was found to be ruptured in posterior wall. Successful treatment, performed 4 hours after appearance of symptoms, was performed as described in Fig. 4, *C*.

surements from angiograms and duplex findings with these figures. However, these measurements are the results of a single study, and it is unclear whether differences in the population that was studied would lead to a wider range of measures. Although this problem may be relevant for fusiform aneurysms, saccular aneurysms are more easily defined. Other than size, additional issues should be considered when the indication for surgery is unclear. The most frequent complication in the development of EICAA is not rupture but rather cerebral embolization, so the physician should consider particularly the presence of either endoluminal thrombus or luminal defects, as well as absolute diameter. Moreover, the presence of an associated kinking or atherosclerotic plaque may prompt a more aggressive approach in borderline EICAA.

As one can see from Table III, the reports in the literature refer to a variety of causes for EICAA. The incidence of atherosclerosis as an etiologic factor is extremely variable and accounts for 9.5% to 83% of cases.* Some authors suggest that the incidence of FMD is greater in European series compared to North American series.[6] Although this seems to be the case in our series, overall data are not conclusive in this regard, because other European series report different results[15] (Table III). Moreover, the terminology used, with the exception of the common atherosclerotic cause, is sometimes confusing.

We believe that histologic findings are essential to precisely define the pathologic characteristics of EICAA in absence of a clear cause (i.e., pseudoaneurysm). The atherosclerotic changes that are often grossly evident are not necessarily the primary cause of the aneurysm, because they may be a secondary process in a dysplastic artery. Stewart et al.[17] reported an incidence of 20% atherosclerotic changes in 88 carotid arteries with occlusive disease caused by FMD.

The wide differences in etiologic incidence may be the result of the referral patterns of the different institutions. In the series by Berguer,[6] most cases were either traumatic or subsequent to drug injection (71.4%), a figure different from those reported by other centers, including our own. In this series, most aneurysms (87.5%) were primarily degenerative forms, either atherosclerotic or dysplastic. This is probably due to the particular referral pattern of our institution, which includes many uncommon elective cases seen in other parts of the country.

Important differences are evident if one compares the epidemiologic pattern of atherosclerotic EICAA with other peripheral aneurysms. The male/female ratio ranges from 2:1 in large series and collected experiences[4,11] to 1:2 in our own; these figures are different from the 5:1 and 30:1 ratios reported for aortic aneurysms and peripheral aneurysms, respectively.[4] Geographic distribution cannot be related to these differences because similar results have been reported in aortic and peripheral aneurysms in both American[4] and European series.[18,19]

Similar to the series by Zwolak et al.,[4] only 11% association of atherosclerotic EICAA with other aneurysms was seen in this series. Although it is difficult to speculate about the pathogenesis of EICAA on the basis of these data, clearly the pathogenesis is different from that of other atherosclerotic aneurysms. On the other side, the epidemiologic pattern of FMD aneurysms is consistent with previous data.[17] Other causes for EICAA have been sporadically reported.[20]

Surgical treatment is believed to be necessary for all EICAA by most authors[1,4,5] because of the high rate of neurologic symptoms—33% to 45% in col-

*References 1, 2, 4, 6, 7, 11, 15, 16.

Table III. Causes of EICAA in the literature

Author	*Geography*	*No. of cases*	*Atherosclerosis*	*FMD*	*Trauma*	*Pseudoaneurysm*	*After surgery*	*Mycotic*	*Congenital*	*Dissecting*
Kaupp[2]	NA	13	61.5%	38.5%	—	—	—	—	—	
Rhodes[3]	NA	23	69.5%	—	17.4%	13%	—	—	—	—
McCollum[1]	NA	37	44%	—	5.4%	51.3%	—	—	—	—
Busuttil[16]	NA	19	26%	—	32%	—	26%	5%	—	—
Pratscke[15]*	E	28	46.5%	—	32.1%	—	3.6%	7.1%	7.1%	—
Zwolak[4]	NA	52	46%	—	—	—	—	—	—	—
Painter[11]	NA	6	83%	17%	—	—	—	—	—	—
deJong[5]	E	18	66.7%	—	—	11.1%	—	5.5%	16.7%	—
Berguer[6]	NA	21	9.5%	—	47.6%	—	9.5%	23.8%	—	9.5%
Moreau[7]	E	38	31.5%	21.1%	15.8%	—	—	2.6%	13.1%	15.8%
Present series	E	24	37.5%	50%	4.1%	—	8.3%	—	—	—

*This series includes 5 external carotid artery aneurysms.
NA, North America; *E*, Europe.

Table IV. Results of surgical treatment of EICAA in the literature

Author	*No. of cases observed*	*No. of cases treated*	*Mortality rate*	*TIA or stroke*	*Cranial nerve lesions*
Kaupp[2]	13	11	9%	0%	0%
Rhodes[3]	23	21	0%	9.5%	4.7%
Carrascal[12]	3	3	0%	0%	0%
McCollum[1]	37	28	7%	7%	0%
Busuttil[16]	19	13	0%	15.4%	NS
Welling[13]	41	22	NS	NS	NS
Zwolak[4]	24	18	0%	22%	22%
Painter[11]	6	6	0%	16%	0%
deJong[5]	18	12	0%	16.7%	8.3%
Moreau[7]	38	38	2.6%	5.3%	21%
Present series	24	24	4.2%	4.2%	20.8%

TIA, Transient ischemic attack.

lected experiences.[5,11] Nonoperative treatment is associated with a risk of stroke as high as 50%.[4] On the contrary, rupture is a very rare complication of EICAA. Only few anecdotal cases are present in the literature.[13,15,16] One case was encountered in our series (4.2%).[21] Although this complication is potentially lethal, containment of arterial bleeding by surrounding tissues in the neck may allow sufficient time for a successful treatment.

Immediate results often show significant complication rates. Many large series[4,5,11,16] report a combined mortality/morbidity incidence greater than 15% (Table IV). Our experience suggests a lower complication rate is to be expected when elective repair is undertaken. One stroke occurred (4.2%), and all cranial nerve lesions were transient (less than 6 weeks) and only caused discomfort to the patient. This support an aggressive approach to the treatment of these aneurysms. It should be kept in mind that atherosclerotic EICAA might have a greater risk of early complications than dysplastic ones, as suggested by some authors and by the stroke case in this series.[4] Cranial nerve lesions are frequent both in the literature and in this series. The high incidence of these lesions is probably associated with the difficult excision of the EICAA from the surrounding structures, because cranial nerve lesions occur in 2.5% of procedures for occlusive carotid artery disease in our institution.

Although some authors[7] report a high incidence of distal extension of EICAA, this was not the case in our series, where no adjunctive maneuvers were necessary to achieve good distal control. This again may reflect the particular referral pattern of our institution. In fact trauma is the most common cause of distal aneurysms[4,16]; in the series by Moreau et al.,[7] approximately one third of distal EICAA was due to either trauma or infection, two uncommon causes in our study population.

Many EICAA can be treated with a direct end-to-end anastomosis of the ICA either to the proximal ICA or to the common carotid artery, because of the saccular structure of the aneurysms or the associate elongation of the distal ICA in dysplastic cases. The external carotid artery can also be used as a proximal transposition site.

Autogenous saphenous vein has been advocated as the graft of choice when a primary anastomosis cannot be performed;[3,4,7] however, our experience with this conduit has not been completely satisfactory. Two of seven (28%) saphenous vein grafts implanted needed revision in this series, either for aneurysmal dilation or for the development of a hemodynamically significant stenosis. Although the dilation of the vein might have been due to weakness of the vein wall of that particular patient, who had already had a vein patch dilation, a 28% late complication rate is still too high for an "ideal" conduit. Presently we advocate primary ICA anastomosis to the common carotid artery or the proximal ICA whenever possible; alternatively the external carotid artery may be used with satisfaction, as suggested by others.[3,16,22] Our experience with PTFE grafts was limited to three cases. No complications occurred, but this experience is too limited to significantly compare this material with the saphenous vein.

In conclusion EICAA are rare and have variable causes. Safety and long-term reliability of appropriate surgical treatment warrant broad surgical indication, because of the risk of embolization and rupture of these aneurysms.

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DISCUSSION

Dr. James A. DeWeese (Rochester, N.Y.). Dr. Faggioli and associates have had the unique opportunity of treating 24 aneurysms of the extracranial ICA. Twelve of the aneurysms were caused by fibromuscular hyperplasia, which is extremely rare in our experience. Another 11 were arteriosclerotic. The authors also saw three traumatic and postoperative aneurysms, which have been the more frequent cause of aneurysms that we have seen. Do the authors have any explanation for the frequency of fibromuscular hyperplasia as the cause of the aneurysm? Did these patients

have similar lesions elsewhere? And, in addition, did the patients with arteriosclerotic aneurysms have aneurysms elsewhere?

Eight of the aneurysms were associated with hemispheric neurologic symptoms, and one aneurysm ruptured. These findings emphasize the importance of timely operations for these lesions. All of the aneurysms were excised, and the artery was reconstructed with end-to-end transpositions, patch grafting, and interposition grafting. PTFE grafts were used in three, and veins were used in seven of the interpositions. What size PTFE graft did you use? Did they remain open? How long have they been in and have they been monitored? What has happened to them?

Dr. GianLuca Faggioli. We do not have an explanation for the high incidence of dysplastic lesions encountered in our series. It seems from other series that the cause of the aneurysm was not always diagnosed by histologic study. In some of the older series, some of these forms may have been missed, especially considering that many of these aneurysms have gross morphologic atherosclerotic changes. Therefore they may have been mistaken for atherosclerotic aneurysms and may have been revealed to be a fibromuscular dysplastic form only at histologic study.

We do have an explanation regarding the low frequency of trauma and postsurgical incidence. And this is due to the particular referral pattern of our institution, which collects elective cases from two thirds of Italy; therefore we see a lot of elective cases. However, we don't see very many emergency cases, so this may explain our referral pattern.

Concerning the localization of fibromuscular lesion in these patients, we didn't see any other kind of lesion that can be called *FMD*. We examined renal arteries, all the data we had did not suggest any lesion in the renal artery.

With regard to the atherosclerotic aneurysm, we had one case in which there was a multiple aneurysm, which means there was localization of a different aneurysm and, in particular, an aortic aneurysm.

Your third question concerned the size of the PTFE and the status of the PTFE grafts, and the size of the PTFE graft was, in the three cases, a 6 mm graft. All the cases at follow-up were patent.

With regard to the length of follow-up in PTFE grafts, this was significantly shorter compared with our saphenous vein graft. Because of this fact and because of the fact that we had only three cases, we didn't make any attempt to compare PTFE with vein. We only wanted to emphasize that our saphenous veins led to high complication rates in the long run.

Dr. Thomas F. Panetta (Brooklyn, N.Y.). Would you expand on the complications related to the saphenous vein grafts? Were these due to the specific use of the saphenous vein or is this related to the fact that the saphenous vein grafts were used for the larger, more complicated aneurysms that are associated with a higher complication rate?

Dr. Faggioli. One of the patients, who underwent two operations, had aneurysmal dilation of the vein that was probably due to a specific disease in the vein. This patient had already undergone carotid endarterectomy for carotid artery stenosis; the saphenous vein patch placed primarily dilated as well, so that was believed to be a problem in the vein of the patient. This occurred only 4 months after the previous surgery. We don't know whether the vein was diseased in the other case.

Dr. Frank T. Padberg, Jr. (East Orange, N.J.). You seem to have operated on these without regard to size criteria, and that's what I wanted to inquire about. With regard to the two categories of FMD and those from atherosclerosis, would there be any difference between the two categories? Should some of these be followed up? Would you recommend a size criteria that would trigger operation?

Dr. Faggioli. The problem of the size is one of the main problems in the literature on this issue. Obviously the size of the aneurysm was difficult to assess retrospectively, especially in those cases in which we did not have a CT scan available. However, by reviewing all the images we had, we could state that all our cases fell into a category of aneurysm that is considered in the literature to be a true aneurysmal form. There is only one study in the literature that states the average size of carotid bifurcation, and it is a European study. We have to rely on that study to make a distinction between true aneurysmal forms and simple dilations. According to that study you can consider a true aneurysm when the carotid bulb is twice as big as the diameter of the ICA or 150% of the diameter of the common carotid artery. By revising all our cases, we can say that all the cases treated fell in that category. We don't know what to do when the bulb is smaller.

There is evidence in the literature that atherosclerotic forms tend to have more symptoms and tend to have worse follow-up compared with dysplastic forms, but, again, the problem is to identify the dysplastic form before surgery.

Dr. John J. Ricotta. How many of these were true ICA aneurysms?

Dr. Faggioli. Only four cases involved the bulb; the other cases were limited to the ICA, so they were easy to detect and to categorize.

Dr. Eli Sorbit (Boston, Massachusetts). How many of these patients had radiation to their neck? Which part of the saphenous vein was used, the one at the ankle or the proximal?

Dr. Faggioli. No patient had radiation of the neck. All patients had the saphenous vein taken from the groin; in the patient who had dilation of the vein twice, both saphenous veins were taken from the groin.