Retrospective multicenter study on the management of asymptomatic carotid artery stenosis with coexistent unruptured intracerebral aneurysm

Giulio Illuminati, MD MD,[a] Paolo Missori, MD,[b] Aurélien Hostalrich, MD,[c] Xavier Chaufour, MD, PhD,[c] Priscilla Nardi, MD,[a] *and* Jean-Baptiste Ricco, MD, PhD,[d] *Rome, Italy; and Toulouse and Poitiers, France*

ABSTRACT

Objective: To evaluate the results of carotid endarterectomy (CEA) in patients with a concomitant asymptomatic intracranial aneurysm discovered at preoperative diagnostic imaging.

Methods: From January 2000 to December 2020, 75 consecutive patients admitted for surgical treatment of asymptomatic more than 70% (North American Symptomatic Carotid Endarterectomy Trial) carotid artery stenosis presented at preoperative computed tomography angiography (CTA) with a concomitant, unruptured intracranial aneurysm (UIA). Aneurysm diameter was 5 mm or less in 25 patients (group A), from 6 to 9 mm in 38 patients (group B), and 10 or more mm in 12 patients (group C). Sixty UIAs (80%) were treated before performing CEA, 10 in group A (40%), 38 (100%) in group B, and 12 (100%) in group C. Twenty-five UIAs (42%) were subjected to surgical clipping and 35 (58%) to coiling. The mean time intervals were 48 days (range, 20-55 days) between clipping and CEA, and 8 days (range, 4 -13 days) between coiling and CEA. CEA was standard and performed through eversion of the internal carotid artery in 36 patients (48%) and through longitudinal arteriotomy with systematic patch closure in 39 patients (52%). The primary end points of the study were mortality and morbidity related to each of the two treatments, including any complication occurring during the time interval between the two procedures or within 30 days after the last procedure. Secondary end points were mid-term survival and freedom from ischemic or hemorrhagic stroke and carotid restenosis.

Results: One patient died during the 30 days after the clipping of a 11-mm diameter UIA of the basilar artery. No other death or complication was observed after CEA and treatment of the UIA, or during the time interval between the two procedures. During a median follow-up of 26 months (interquartile range, 18-30 months), no late stroke and no carotid restenosis were observed. At 22, 27, 29 and 31 months after CEA, four patients in group A underwent surgical clipping of an enlarging intracranial aneurysm that had not been treated initially owing to its small diameter. The cumulative survival rate at 30 months by Kaplan-Meier plots was 83 ± 5%.

Conclusions: Concomitant asymptomatic carotid artery stenosis and UIA is a rare entity. Our study suggests that in this setting, prior treatment of the UIA followed by CEA is safe. (J Vasc Surg 2022;76:1298-304.)

Keywords: Carotid artery stenosis; Intracranial aneurysms; Elective treatment; Carotid endarterectomy

The incidence of internal carotid artery (ICA) stenosis with a concomitant asymptomatic, unruptured intracranial aneurysm (UIA) ranges between 1.9% and 5.0%.[1-4] Given the rarity of this incidental finding, a definite protocol for treatment of the two conditions has not been well-established.[3,5] Different strategies have been proposed, including UIA treatment before carotid endarterectomy (CEA),[6] simultaneous treatment of the two conditions,[7] or initially not treating the UIA.[1,7-9] In general, symptomatic carotid artery stenosis associated with an UIA requires prior CEA within the 2 weeks after the index neurological event, at least in patients with an UIA of less than 10 mm.[1,3-5,8,9] When considering patients presenting asymptomatic severe ICA stenosis associated with an UIA, a different strategy could be considered by underlining the risk of aneurysm rupture after CEA in conjunction with a hyperperfusion syndrome.[2,5,10,11] We report here the experience of three European academic centers on this rare condition with recommendations for treatment.

METHODS

From January 2000 to December 2021, in three centers (Rome La Sapienza, Toulouse University Hospital, and Poitiers University Hospital), the records of 75 consecutive

From the Department of Surgical Sciences,[a] and Department of Neurosurgery,[b] University of Rome "La Sapienza," Rome; the Department of Vascular Surgery, University of Toulouse, Toulouse[c]; and the Department of Surgical Research, University of Poitiers, Poitiers.[d]

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Correspondence: Jean-Baptiste Ricco, MD, PhD, Department of Surgical Research, University of Poitiers, Poitiers, France (e-mail: jean.baptiste.ricco@univ-poitiers.fr).

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patients admitted for treatment of severe (North American Symptomatic Carotid Endarterectomy Trial >70%), asymptomatic stenosis of the ICA with an asymptomatic noncavernous, UIA incidentally discovered at preoperative computed tomography angiography (CTA) were reviewed in a multidisciplinary conference, taking into account its size and morphology in view of defining a strategy (careful watching or intervention), followed by CEA.

Patient demographic and clinical data were collected in the three centers using an electronic medical record system with coding appropriate for carotid stenosis, CEA, and UIA. All data were entered in an institutional database, including age, gender, carotid stenosis, (side, degree, symptoms, treatment, complications), and UIA (location, side, treatment, perioperative mortality and morbidity, and follow-up with UIA growth or rupture). UIA diameter and ICA stenosis were retrospectively assessed by radiologists from retrieved images (magnetic resonance imaging and CTA) or by angiologists from written reports (duplex ultrasound examination). These 75 patients represent 1% of the 7503 carotid interventions carried out during the same period in the three centers. Thirty-four patients (45%) were male and 41 were female (55%) with a mean age of 63 years (range, 52-77 years). Risk factors and characteristics of the UIA are summarized in Table I. Among the risk factors, chronic renal insufficiency was defined as a serum creatinine level of more than 150 mg/dL. Coronary artery disease was defined as any history of myocardial ischemia, including medical treatment, previous coronary artery bypass grafting, or percutaneous intervention. Otherwise, no patient presented either a family history of intracranial aneurysm or a dominant polycystic kidney disease, or any other condition known to be associated with an increased incidence of intracranial aneurysm.

For the purposes of the study, patients were divided into three groups (Fig 1). Patients in group A (n = 25) presented an UIA of 5 mm or smaller diameter, patients in group B (n = 38) presented an UIA of 6 to 9 mm diameter, and patients in group C (n = 12) presented an aneurysm 10 or more mm in diameter.

The location of the UIA is shown (Fig. 2), with details regarding lateralization of the UIA relative to carotid stenosis presented in Table II. Thirty-two UIAs were located in the middle cerebral artery, 22 in the posterior communicating artery, 7 in the anterior communicating artery, 6 in the anterior cerebral artery, 3 in the superior cerebellar artery, 3 in the basilar artery, and 2 in the anterior choroid artery.

The decision on which lesion required prior treatment was made after a multidisciplinary meeting bringing together vascular surgeons, neurosurgeons, and interventional neuroradiologists in each institution, who considered aneurysm morphology including daughter sac, wall irregularity, diameter and size ratio between aneurysm diameter and that of the affected artery. Their consensus was reported systematically in the patient's records and in the informed consent form signed by the patient. After this consensus meeting, a decision was made concerning the sequencing of the

ARTICLE HIGHLIGHTS

- **Type of Article:** Multicentric retrospective cohort
- **Key Findings:** Over a 20-year course, 75 patients with asymptomatic carotid stenosis and unruptured intracranial aneurysm (UIA) were included in the study. Sixty UIAs (80%) were treated before performing carotid endarterectomy, 25 (42%) underwent surgical clipping and 35 (58%) coiling. One patient died during the 30 days after the clipping of an aneurysm of the basilar artery. No other death or complication was observed subsequent to carotid endarterectomy and treatment UIA, or during the time interval between the two procedures. During follow-up, four patients with an UIA of 5 mm or smaller diameter underwent surgical clipping of an enlarging intracranial aneurysm that had not been treated initially owing to its small diameter.
- **Take Home Message:** Concomitant asymptomatic carotid artery stenosis and UIA is a rare entity. Our study suggests that prior treatment of the UIA followed by carotid endarterectomy is safe.

Table I. Demography, risk factors, cerebral aneurysm, and carotid stenosis in 75 patients

	Number	(Percent)
Age, years (range)	63	52-77
Male sex	34	(45)
Hypertension	57	(76)
Current smokers	32	(43)
Diabetes	20	(27)
Coronary artery disease	16	(21)
Chronic renal insufficiency	4	(5)
ASA classification		
Class 2	44	(59)
Class 3	31	(41)
Unruptured intracerebral aneurysm		
Group A: diameter ≤5 mm	25	(33)
Group B: diameter 6-9 mm	38	(51)
Group C: diameter ≥10 mm	12	(16)
Carotid artery stenosis		
Asymptomatic	75	(100)
Degree of stenosis (NASCET criteria)	≥70%	(100)

NASCET, North American Symptomatic Carotid Endarterectomy Trial.

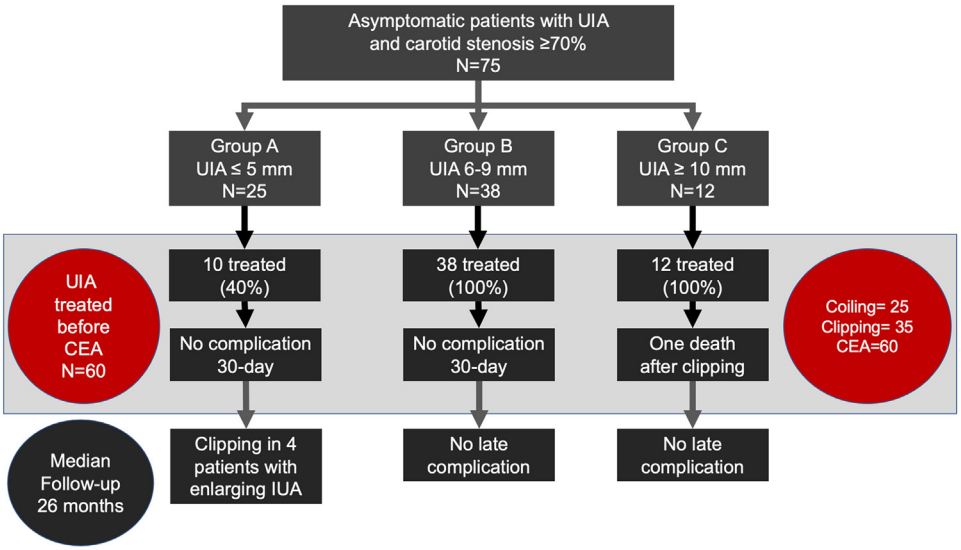

Fig 1. Flowchart of the study with the three groups of patients according to intracranial aneurysm diameter, intervention, and comparison of outcomes. *CEA*, Carotid endarterectomy; *UIA*, unruptured intracranial aneurysm.

procedures. According to these decisions, the UIA was treated before carotid stenosis by means of surgical clipping or coiling according to the morphology and location of the UIA. Whenever an endovascular approach was considered as an option with surgical clipping, the latter was preferred so as to avoid access through the stenotic ICA. The decisions resulting from the consensus meetings were convergent in the three centers. All patients were examined by a certified neurologist after UIA treatment and after CEA following the usual protocol of the three university hospitals. Postoperative myocardial infarction was defined as the association of clinical and electrical signs of myocardial ischemia together with an elevated serum troponin level. Serum troponin levels were systematically tested daily in the first 72 postoperative hours after CEA and UIA treatment in all patients.

Although oral antiplatelet treatment with either aspirin (75/150 mg/day) or clopidogrel (75 mg) was not discontinued in case of coiling, it was interrupted 7 to 10 days before surgical clipping and resumed 48 hours after surgery. All patients received statins (atorvastatin 20 mg/day), starting 1 week before the first procedure. All but 15 patients receiving atorvastatin 20 mg/day had a low-density lipoprotein (LDL) cholesterol down to 1 to 2 mmol/L. In the 15 patients whose LDL level was greater than 2 mmol/L, ezetimibe 10 mg/day had been combined with atorvastatin, resulting in an LDL level lower than 2 mmol/L. The time interval between UIA clipping and CEA was 48 days (range, 20-55 days) and 8 days (range, 4-13 days) between UIA coiling and CEA.

Before CEA, coronary angiography followed by percutaneous coronary intervention was performed in 11 patients (15%) who subsequently underwent CEA under dual antiplatelet treatment.[12,13]

UIA clipping or coiling was performed under general anesthesia. CEA was performed under general anesthesia with transcutaneous cerebral oximetry monitoring (INVOS, Medtronic, Dublin, Ireland). No shunt was used in this series. CEA was performed by eversion technique in 38 patients (51%) and through longitudinal arteriotomy with polyester patch closure in 37 (49%). After CEA, all patients were followed by duplex ultrasound examination on a yearly basis. According to neurointervention and neurosurgical protocols, magnetic resonance imaging of the brain was performed every 2 years throughout the study. The median follow-up was 26 months (interquartile range, 18-30 months). No patient was lost to follow-up.

Ethical issues. All patients signed an informed consent for both procedures. Owing to the retrospective nature of the study, institutional ethical committee approval was waived in all participating centers. According to EU regulations in line with the General Data Protection Regulation, data protection, anonymization, and security were applied in compliance with the cybersecurity framework.

Statistical analyses. The primary end points of the study were 30-day mortality and complications related to each procedure (stroke, UIA rupture, or myocardial infarction) or occurring during the time interval between procedures. Secondary end points were survival reported by Kaplan-Meier survival estimates with 95% confidence interval, freedom from ischemic or hemorrhagic stroke, and freedom from carotid restenosis. Descriptive statistics were reported as mean and standard deviation for continuous variables or median and interquartile range

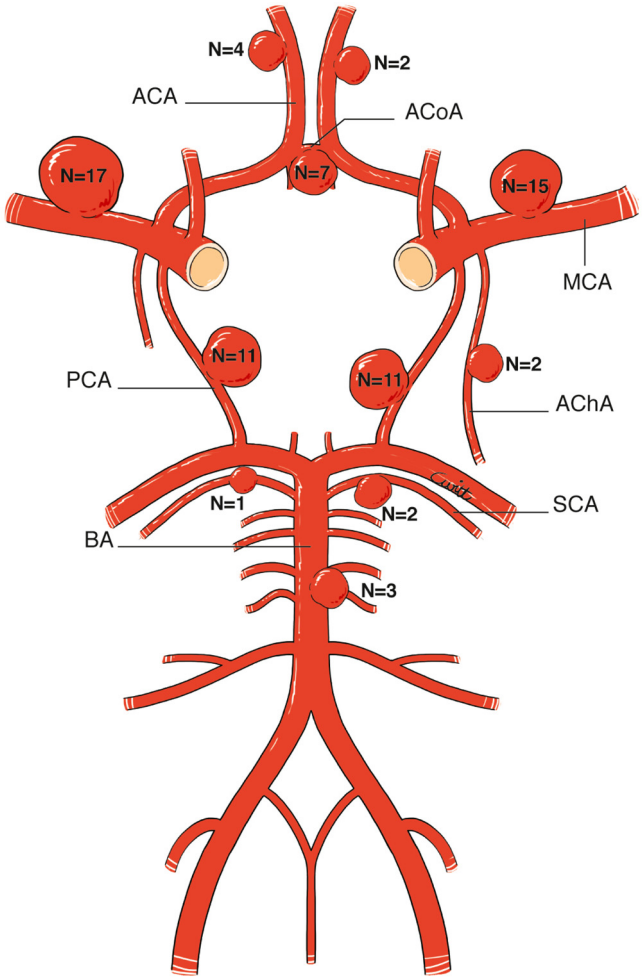

Fig 2. Location and number of asymptomatic cerebral aneurysms in the circle of Willis and its branches. The diameter of each aneurysm is proportional to the number of aneurysms present on the cerebral artery. *ACA*, Anterior cerebral artery; *AChA*, anterior choroidal artery; *ACoA*, anterior communicating artery; *BA*, basilar artery; *MCA*, middle cerebral artery; *PCA*, posterior cerebral artery; *SCA*:, superior cerebellar artery.

where appropriate and as percentages for categorical variables.

RESULTS

Procedures. All in all, 60 patients (80%)—10 in group A (40%) and all 50 (100%) patients in group B and in group C—underwent treatment of the UIA before CEA: 25 UIA (42%) underwent surgical clipping and 35 (58%) coiling. Treatment methods according to the location of the UIA are detailed in Table III: 19 UIAs of the middle cerebral artery (59%) were treated by surgical clipping. Among the 22 UIAs of the posterior communicating artery, 9 received coiling by contralateral carotid access (41%) and the other 13 underwent surgical clipping (59%). All seven UIAs of the anterior communicating artery and two aneurysms of the anterior choroid artery were treated by coiling with contralateral carotid access (100%). Surgical clipping was also used in all six UIAs of the anterior cerebral artery (100%), in two UIAs of the basilar artery (67%), and in two UIAs of the superior cerebellar artery (67%).

Primary end points. All in all, the 30-day postoperative/postprocedure mortality after treatment of IUA was 1.7%, one patient (95% confidence interval, 0.9%-9.7%). No other complications, including stroke occurred in the study. This patient died of myocardial infarction 4 days after surgical clipping of an 11-mm diameter UIA of the basilar artery. No death, myocardial infarction, or stroke was observed in the time interval between UIA treatment and CEA or after CEA. Three peripheral facial nerve palsies (4%) after CEA regressed in 2 weeks. The cumulative mortality and morbidity of the two procedures, including facial nerve palsies, was 6.6% (95% confidence interval, 2.2%-16.4%).

Secondary end points. During follow-up, 11 patients (15%) died of causes unrelated to the procedures: 8 of cancer, 1 of end-stage renal failure, 1 of multiorgan system failure after major trauma, and 1 of complications related to coronavirus disease 2019 infection. All in all, the 30-month survival rate by Kaplan-Meier plots was 83 ± 5% (Fig 3). No late ischemic or hemorrhagic stroke was observed during follow-up and duplex ultrasound examination of the arteries to the head did not reveal any carotid restenosis.

During follow-up, four patients in group A underwent uneventful delayed surgical clipping of an enlarging intracranial aneurysm at 22, 27, 29 and 31 months after CEA. Owing to their small diameter (≤5 mm) and absence of morphological abnormality (daughter sac, irregular wall), these aneurysms had not been treated initially. One patient (1.3%) underwent eversion CEA for progressing more than 70% asymptomatic contralateral ICA stenosis.

DISCUSSION

This retrospective study suggests that patients with more than 70% asymptomatic carotid stenosis and concomitant UIA can be treated safely by coiling or clipping of UIA followed by CEA. These results were achieved in three European centers following a multidisciplinary consensus on a case-by-case basis bringing together vascular surgeons, neurosurgeons, and interventional neuroradiologists. This observant attitude is explained by the absence of recommendations and guidelines concerning the treatment of patients with severe carotid stenosis and concomitant intracranial aneurysm.

In patients with asymptomatic severe ICA stenosis, a major argument favoring prior treatment of UIA is that increased cerebral perfusion following CEA in patients with altered regulatory mechanisms of intracranial

Table II. Location of unruptured intracranial aneurysms (UIAs) with concomitant asymptomatic carotid stenosis in 75 patients

Artery	Total (n = 75)	Ipsilateral (n = 33)	Contralateral (n = 32)	Central (n = 10)
Middle cerebral artery	32	17	15	–
Posterior communicating artery	22	11	11	–
Anterior communicating artery	7	–	–	7
Anterior cerebral artery	6	4	2	–
Basilar artery	3	–	–	3
Superior cerebellar artery	3	1	2	–
Anterior choroid artery	2	0	2	–

Table III. Techniques used for treatment of unruptured intracranial aneurysms (*UIAs*) according to their location

		Ipsilateral (n = 27)		Contralateral (n = 24)		Central (n = 9)	
Location of aneurysms (n = 75)	Treated (n = 60)	Clipping	Coiling	Clipping	Coiling	Clipping	Coiling
Middle cerebral artery (n = 32)	19	11	0	8	0	–	–
Posterior communicating artery (n = 22)	22	11	0	2	9	–	–
Anterior communicating artery (n = 7)	7	–	–	–	–	0	7
Anterior cerebral artery (n = 6)	6	4	0	2	0	–	–
Basilar artery (n = 3)	2	–	–	–	–	2	0
Superior cerebellar artery (n = 3)	2	1	0	1	0	–	–
Anterior choroid artery (n = 2)	2	0	0	0	2	–	–

Among 75 patients with an UIA, 60 patients (80%) were operated and underwent treatment of the aneurysm before carotid endarterectomy. All 27 ipsilateral and 13 contralateral UIAs (n = 40) underwent clipping, and 9 patients with a central UIA underwent coiling (n = 7) or clipping (n = 2).

perfusion pressure often induced by internal carotid stenosis,[14-17] may contribute to aneurysmal rupture.[4,8,9] A risk that may persist for several days or even a month after CEA[1,2] with intraparenchymal hemorrhage and edema.[5,10,11,18-20] Several cases of previously asymptomatic UIA rupture after CEA have been reported,[9,21,22] including aneurysms of small diameter (4 mm) with a narrow aneurysmal neck.[14]

Conversely, cerebral hypoperfusion and ischemic stroke owing to carotid stenosis may occur during intracranial aneurysm repair or be triggered by intraoperative hypotension deliberately induced to facilitate UIA repair,[8,23] but these were not observed in this series.

Concerning the timing and sequence of treatment, most of the currently available reports concern patients with prevalence of symptomatic ICA stenoses, which require expedited treatment.[8,9,21] Although ruptures of small aneurysms occurring several months after ICA revascularization, have been reported,[9,14,21] the priority of carotid repair in cases of symptomatic ICA stenosis with concomitant UIAs seems well-established.[9] All in all, prior CEA seems to be safe if the diameter of the UIA is 5 mm or less.[5,7,22,24] For a UIA with a diameter between 6 and 10 mm, recommendations have varied[25,26] and because the ICA does not seem to increase middle-term risk of rupture for aneurysms of greater than 10 mm,[27] the priority of ICA revascularization for symptomatic stenoses seems to be justified. However, the setting of asymptomatic severe ICA stenosis associated with an UIA is markedly different. Because carotid revascularization is not urgent, the initial treatment of the UIA depends on its diameter, morphology, and potential increase of wall shear stress after CEA.[1,28,29] Furthermore, the need for antiplatelet therapy or, eventually, dual antiplatelet therapy in cases of coronary artery disease requiring percutaneous coronary intervention may increase the risk of an aneurysm rupture. Based on these assumptions, we decided to initially treat all UIAs more than 5 mm in diameter with concomitant severe asymptomatic ICA stenosis. For UIAs with a diameter or 5 mm or less, the decision was made on a case-by-case basis. In this group, 15 UIAs without morphological abnormality were left untreated with careful monitoring as recommended,[30] and 10 bilobed IUAs or with a daughter sac and irregular wall were treated. Of note, among these 15 initially untreated UIAs, 4 evolved beyond 10 mm in diameter, 1 with a daughter sac, and all four required clipping at 22, 27, 29, and 31 months after CEA. A more conservative strategy has been proposed by Tallarita et al[31] for treatment of these associated conditions. They found no early UIA rupture (within 30 days) after carotid artery

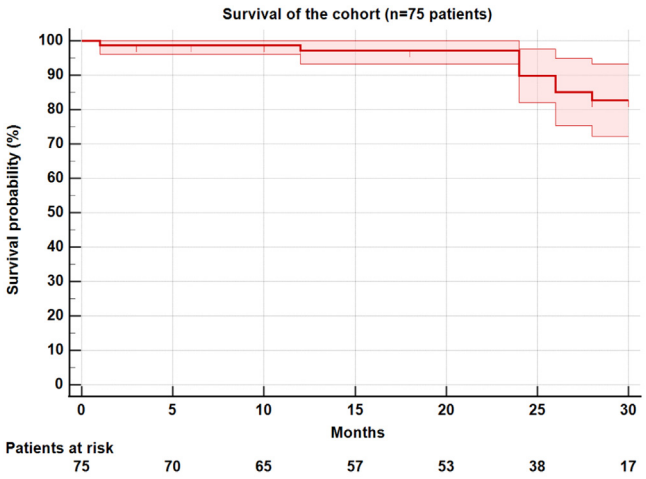

Fig 3. Kaplan-Meier survival plots of the 75 patients included in the cohort with the number of patients at risk for each time interval. Median follow-up of the study was 26 months (interquartile range, 18-30 months). Cumulative survival at 30 months was 83 ± 5%.

revascularization alone, but 3.8% of UIAs ruptured after 30 days and five delayed UIA interventions were performed after 30 days. They recommended proceeding with carotid revascularization and, if indicated, treating UIA after the patient has recovered from the carotid procedure. The results of our multicenter retrospective study suggest that patients with greater than 70% asymptomatic carotid stenosis and concomitant UIA of more than 5 mm diameter or 5 mm diameter or smaller with morphological abnormalities can be treated safely by coiling or clipping of the UIA followed by CEA and underline the importance of monitoring patients with a UIA of less than 5 mm not treated initially.

Limitations. The limitations of this study are intrinsic to its retrospective nature and to the limited number of patients. Furthermore, because this was not a population study, we cannot assess the true incidence of coexistence between UIA and severe asymptomatic carotid stenosis. In addition, as in other studies reporting concomitant carotid stenosis and UIA, the low prevalence of these tandem lesions restricted the power of our statistical analysis. However, none of these consecutive patients were lost to follow-up and over a median follow-up of 26 months, no late stroke and no carotid restenosis were observed.

CONCLUSIONS

In asymptomatic patients presenting a cerebral aneurysm with concomitant more than 70% asymptomatic carotid artery stenosis, prior treatment of the UIA followed by CEA seems in most cases to be a safe strategy.

AUTHOR CONTRIBUTIONS

Conception and design: GI, JBR
Analysis and interpretation: GI, PM, AH, XC, PN, JBR
Data collection: GI, PM, AH, XC, PN, JBR
Writing the article: GI, AH, JBR
Critical revision of the article: GI, PM, AH, XC, PN, JBR
Final approval of the article: GI, PM, AH, XC, PN, JBR
Statistical analysis: GI, JBR
Obtained funding: Not applicable
Overall responsibility: JBR

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