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Endovascular Treatment of Anterior Choroidal Artery Aneurysms

Michel Piotin, Charbel Mounayer, Laurent Spelle, Marc T. Williams, and Jacques Moret

BACKGROUND AND PURPOSE: Ischemic stroke is the most common complication after surgical clipping of anterior choroidal artery (AChA) aneurysms, and the reported morbidity-mortality rates vary from 5% to 50%. We report the findings in a series of 18 consecutive patients who underwent endovascular treatment (EVT) for an AChA berry aneurysm.

METHODS: In this retrospective study, the aneurysms were defined according to their size and position, the presence of a concomitant arteriovenous malformation (AVM), the mode of presentation. The patients were clinically assessed before and after the EVT, as well as at each angiographic follow-up.

RESULTS: All were small-sized aneurysms, with greater diameters ranging from 2 to 8 mm (mean, 4 mm), arising from the supraclinoid internal carotid artery close to the origin of the AChA. Two were AVM-associated aneurysms. Fourteen patients (14/18 [78%]) presented with subarachnoid hemorrhage. All aneurysms were selectively embolized with coils. There was one (1/18 [5.5%]) treatment-related death due to aneurysm perforation. Another patient (1/18 [5.5%]) developed a transient contralateral hemiparesis. Fourteen patients (14/18 [78%]) were followed up clinically and angiographically for 3–32 months (mean, 14 months). None of them (re)hemorrhaged during this period.

CONCLUSION: The EVT of AChA berry aneurysms is effective to protect from rebleeding. Our complication rate compares favorably with those of the surgical series.

Anterior choroidal artery (AChA) aneurysms account for 4% of all intracranial aneurysms. Ischemic stroke has been reported as the most common complication after surgical clipping of aneurysms of the internal carotid artery (ICA)/AChA complex. Patients with aneurysms located on the AChA itself have even a higher risk for postoperative stroke. Few surgical papers have addressed the high morbidity-mortality rates of surgical clipping of aneurysms arising from this specific anatomic location. In the literature, the reported morbidity-mortality rates of surgical procedures vary from 5% to 50% (1–6). With the goal of assessing the efficacy of, as well as the complication rates after, endovascular treatment (EVT) of these aneurysms, we retrospectively studied a series of 18 consecutive patients for whom an AChA aneurysm was selectively embolized.

Methods

Patient Population

Between February 1995 and November 2001, 23 patients harboring AChA aneurysms were admitted to our institution for endovascular treatment. For each patient, we recorded demographic data, clinical presentation, location of the aneurysm, presence of other arterial aneurysms, presence or absence of coexisting intracranial arteriovenous malformation (AVM), therapeutic interventions and immediate anatomic results, and clinical and angiographic follow-up information (Table 1). All data were entered into a computer data base (Open Access III, version 3.0.2; Software Products International, San Diego, CA). Radiography files containing relevant CT and/or MR and angiographic studies were available for all patients.

Classification of Anterior Choroidal Artery Aneurysms

In this retrospective study, the analysis of aneurysm characteristics were performed by a senior neurointerventionist (C.M.). The AChA aneurysms were lesions arising from the supraclinoid ICA in the vicinity of the origin of the AChA or arising from the proximal segment of the AChA itself. Aneurysms were classified according to their greater sac diameter. The size of the aneurysm neck was measured and related to the largest sac diameter. When the neck-to-sac ratio was greater than 0.85, the aneurysm was judged to be wide-necked.

Endovascular Treatment

All aneurysm embolizations were carried out with detachable coils by using a femoral or a bifemoral 6F access sheath(s);

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Patient characteristics

N/Age/Gender (years)	Aneurysm Location	Hemorrhage	Source of Bleeding	AVM	Hunt & Hess Grade	Device/Embolization Technique	Treatment-Related Complications	Follow- Up (months)
1/41/M	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Remod	Death	NA
2/32/M	ICA/AChA	SAH	MCA	None	1	Coils/Remod	None	None
3/40/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Remod	None	24
4/40/M	ICA/AChA	SAH	ICA/AChA	None	4	Coils/Std	None	32
5/30/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Std	Transient hemiparesis	4
7/30/F	ICA/AChA	SAH	ICA/AChA	None	2	Coils/Std	None	None
6/52/M	ICA/AChA	SAH	ICA/AChA	None	3	Coils/Std	None	30
7/49/F	ICA/AChA	SAH	ICA/AChA	None	2	Coils/Std	None	28
8/53/M	ICA/AChA	None	NA	Yes	0	Coils/Std	None	None
9/50/F	ICA/AChA	None	NA	None	0	Coils/Remod	None	3
10/48/M	ICA/AChA	SAH	ICA/AChA	None	2	Coils/Std	None	18
11/34/M	ICA/AChA	None	NA	Yes	0	Coils/Std	None	18
12/43/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Std	None	12
13/55/F	ICA/AChA	SAH	ICA/AChA	None	0	Coils/Stent	None	None
14/26/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Std	None	None
15/25/M	Distal AChA	None	NA	Yes	0	NBCA	None	6
16/25/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Remod	None	3
17/24/F	ICA/AChA	SAH	ICA/AChA	None	1	Coils/Std	None	None
18/46/M	ICA/AChA	None	NA	None	0	Coils/Std	None	24

Note.—ICA signifies internal carotid artery; AChA, anterior choroidal artery complex; SAH, subarachnoid hemorrhage; ntraventricular hemorrhage; MCA, middle cerebral artery; NA, not applicable; Remod, remodeling technique; Std, standard coiling technique.

bifemoral access was used when the balloon-remodeling technique was performed. All procedures were performed under general anesthesia and full anticoagulation (5000 IU of heparin, given as an intravenous bolus injection, followed by continuous infusion of 2500–3000 IU/h). The goal of anticoagulation therapy was to keep the activated clotting time (ACT) at two to three times above the normal value during catheterization and coil deposition. In addition, in all cases with no history of subarachnoid hemorrhage (SAH) within the last 4 weeks, 250 mg of aspirin were given intravenously as a single dose at the beginning of the procedure. Heparin was continued for 48 hours after embolization, keeping the ACT at two to three times above the normal value. Angiography by using a fast subtracted angiorotation technique was performed until November 1998; afterward, rotational angiography followed by three-dimensional (3D) reconstruction of the native projections (Integris; Philips, Best, the Netherlands) was obtained just before starting the embolization. On the basis of the images generated by the rotation acquisitions (either by the fast subtracted angiorotation or by the 3D images), one to two working projections were defined, giving to the operator the best achievable view of the aneurysm neck. At the end of the procedure, posttherapeutic angiograms in frontal, lateral, and working projections were performed to rule out any parent artery or branch occlusion. Arterial access sheaths were removed immediately after the procedure and the puncture site(s) was (were) either manually compressed (10/18 [55%] patients) or closed (8/18 [45%] patients) with a collagen-plug hemostatic closure device (Angio Seal; Daig/St. Jude Medical, St. Paul, MN). Aneurysm occlusion at the end of the procedure and at follow-up angiography was considered total when the sac and the neck were densely packed with no more aneurysm sac filling by contrast material. Subtotal occlusion was defined as the sac being occluded but suspicion of a neck remnant or an obvious tiny neck remnant remained. Incomplete occlusion was defined as loose coil packing with persistent opacification of a sac remnant.

Follow-up

The patients were clinically assessed before and after the endovascular procedure, as well as at each angiographic fol-

low-up by a member of the neurointervention team (M.P., C.M., L.S., J.M.), meaning that the clinical and the angiographic assessments were not specifically performed by the physician who was initially in charge of the EVT. We used the Glasgow outcome scale (GOS) to classify the patient's recovery (GOS 5, death; GOS 1, resumption of normal activities). Our angiographic follow-up protocol for the AChA aneurysms was the same as we normally do for other aneurysms: a first follow-up between 3 and 6 months; a second follow-up between 12 and 18 months; and a third follow-up between 24 and 36 months after the EVT. Angiographic follow-up consisted of frontal, lateral, and working projections, which were determined according to the patient's bony landmarks as defined during the endovascular procedure. Apart from angiographies, no other imaging studies were conducted for follow-up.

Results

Patient Demographics

Of the 18 patients, eight (44%) were men and 10 (56%) were women, with an average age of 40 years (range, 24–55 years) at presentation. Initial clinical presentation for the purpose of calculation of follow-up was considered to have occurred at the time of the of the endovascular treatment.

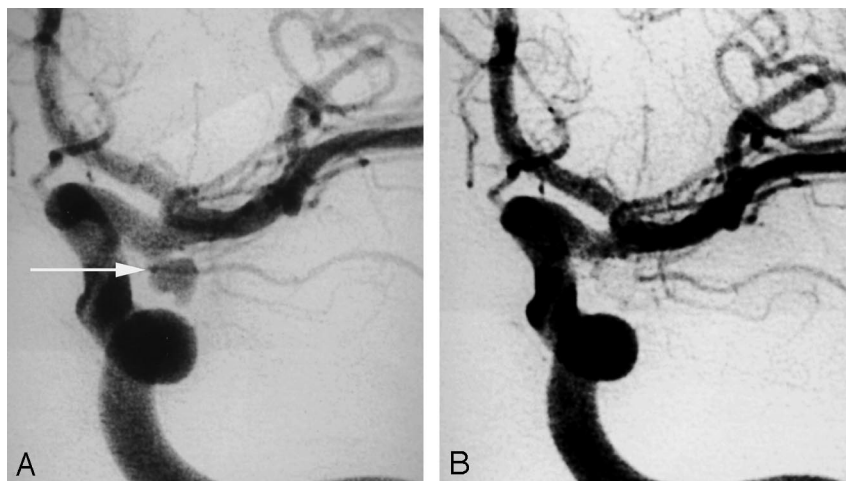
Aneurysm Characteristics

All aneurysms were small saccular aneurysms, with greater diameters ranging from 2 to 8 mm (mean, 4 mm), arising from the supraclinoid ICA close to the origin of the AChA. Seven of the aneurysms (39%) were small-necked aneurysms, and the remaining 11 (61%) were considered to be wide necked. The AChA aneurysms arose from the wall of the ICA closely to the AChA origin; the AChA arose from the neck of the aneurysm in seven patients (39%) fol-

FIG 1. 52-year-old man presenting with SAH.

A, Left ICA angiogram (working projection) showing the AChA (arrow) arising from the aneurysm neck.

B, Left ICA control angiogram (working projection) showing the total circulatory exclusion of the aneurysm with preserved patency of the AChA.



lowed by aneurysms arising laterally to the origin of the AChA (39%) and four (22%) aneurysms arising between the origin of the AChA and the carotid bifurcation.

AVM-Associated Aneurysms

Two (11%) of the 18 AChA aneurysms were AVM-associated aneurysms, located close to the origin of the AChA.

Hemorrhagic Presentation

Fourteen patients (78%) presented at our institution with an SAH. In 13 of those patients (93%), the AChA aneurysm was the source of a SAH, and in one case the SAH was due to the rupture of a middle cerebral artery bifurcation aneurysm. The source of hemorrhage in those who had bled was deduced from angiographic findings correlated with CT data. In four (22%) of 18 patients, the AChA aneurysm discovery was fortuitous. One of the 14 (7%) patients with SAH at presentation had to be shunted immediately after the EVT because of hydrocephalus. This patient (patient 4) presented with a Fisher 4 hemorrhage.

Endovascular Treatment

Thirteen of 14 (93%) of the treated aneurysms in the acute phase were AChA aneurysms; in one case, the AChA aneurysms did not bleed but were associated with other ruptured aneurysms located on the middle cerebral artery bifurcation. The treatment of the fortuitously discovered AChA aneurysm was carried out 5 months after the selective embolization of the aneurysm responsible for the bleeding (patient 2). Of the 18 AChA aneurysms treated by endovascular means, four (22%) were unruptured; two (50%) of the four unruptured aneurysms were associated with an AVM. Seventeen (94.5%) of 18 aneurysms were embolized with Guglielmi detachable coils (GDC; Target/Boston, Fremont, CA), whereas the remaining one (5.5%) was occluded with Trufill-DCS coils (Cordis, Miami Lakes, FL). Of the 18 treated aneurysms,

17 (94.5%) were completely excluded from arterial circulation; the remaining one (5.5%) was considered incompletely treated at the end of the endovascular procedure. Small-necked saccular aneurysms were treated in a standard fashion (Fig 1). The remodeling technique by using a temporarily inflated balloon across the neck was used in five (28%) cases to occlude the sac while protecting the AChA arising from the neck from inadvertent occlusion by coils. In three cases (17%), the remodeling technique was done with a Solstice balloon-microcatheter (Medtronic, Minneapolis, MN), whereas in the two remaining cases (11%) an Equinox balloon-microcatheter (Micro Therapeutics, Irvine, CA) was used. In one case (5.5%) a stent (Medtronic/AVE; Minneapolis, MN) was deployed across the neck (neck-to-sac ratio >1), allowing safe coil deposition into the sac.

Treatment-Related Complications

There was one (1/18 [5.5%]) death in this series. This patient had a small ruptured AChA aneurysm, which was inadvertently perforated with the microcatheter during coiling, causing rebleeding, and a massive SAH. The aneurysm finally was incompletely coiled, but the patient died three days after admission. Another patient (5.5%) developed a transient contralateral hemiparesis, but recovered within 24 hours after the embolization of a proximal saccular ruptured AChA aneurysm. There were no groin hematomas or false aneurysms in this series despite the postprocedural anticoagulation regimen.

Follow-up

Of the 18 treated AChA aneurysms, 14 (78%) were followed up, ranging from 3 to 32 months (mean, 14 months). None of the followed patients (re)hemorrhaged during this period. One of the embolized aneurysm showed a small recanalization. In another patient, we found a small enlargement of the neck remnant (<1 mm) at the first follow-up angiography, at 3 months; this remnant remained stable at the second follow-up angiography, at 18 months. All pa-

tients with a nonruptured aneurysm (4/18 [22%]) had a GOS of 1 at discharge. In the patients with ruptured aneurysms (14/18 [78%]), four patients (22%) were lost to follow-up; nine (50%) had a GOS of 1 at their last follow-up; one (5.5%) died as the result of the EVT (GOS 5).

Discussion

Surgical Treatment of AChA Aneurysms

The surgical approach is similar to that for other supraclinoid carotid artery lesions, but surgery may involve a higher risk of debilitating ischemic complications because of the critical territory supplied by the AChA. Ischemic stroke secondary to the occlusion of the AChA itself or its branches has been reported as a common complication after surgical clipping of AChA aneurysms (1–7). Inadvertent clipping of the parent artery, thromboembolism during manipulation of the AChA (temporary clipping), distortion of the clip due to postoperative edema of neighboring structures, and vasospasm are the commonly accepted factors involved in postsurgical ischemic strokes. The clinical features of AChA stroke syndrome typically consist of contralateral hemiparesis, hemisensory loss, and homonymous hemianopsia. Friedman et al, however, reported that, in their patients with postoperative AChA stroke, the clinical syndrome was typified by a contralateral hemiplegia, which in some cases was attenuated over the face, arm, and leg, whereas hemisensory symptoms and visual field defects were much less consistent (5). Because the AChA has a very functional territory, the surgical literature distinguishes the aneurysms that arise from the AChA or adjacent to its origin from other intracranial aneurysms. This intimate relationship to the AChA has a major surgical significance; inadvertent damages to the AChA during dissection or clipping may have deleterious clinical consequences. In 1968, Drake et al described their surgical technique to clip AChA aneurysms by a subfrontal approach (1). They reported a series of seven aneurysms in seven patients. Five (71%) were ruptured at the time of presentation, and two (29%) were unruptured but were associated with an adjacent posterior communicating ruptured aneurysm. Six (86%) were treated in the acute period after SAH, whereas the remaining patient did not undergo surgery because of his poor neurologic status (Hunt and Hess grade V) and died 9 days after admission secondary to a second rupture. In this small series, the authors reported two (33%) cases of aneurysmal rupture during surgery; in one of these cases (50%), the aneurysm burst as the dura was being opened, while in the second case (50%) the aneurysm was torn during its surgical dissection. Both patients died a few hours after surgery. In the four patients (66%) who survived surgical clipping, an ischemic stroke in the AChA territory related to surgery was reported in one (17%). Later, in 1978, Yasargil et al (2) reported a series of 16 AChA aneurysms surgically treated; 13 (81%) were ruptured

and three (19%) were associated with another ruptured aneurysm. Three (19%) patients had postoperative transient palsy of the ipsilateral third cranial nerve but spontaneously recovered within a few days. One patient (6%) had a postoperative lethargic course without focal neurologic deficit but recovered within 4 weeks. Three patients (19%) developed a postsurgical worsening of a preexisting hemiparesis contralateral to the aneurysm; in one of these 16 patients (6.25%), an AChA occlusion was angiographically demonstrated. Two patients (12.5%) died secondary to postsurgical sepsis. In 2001 Friedman et al (5) reported the largest AChA aneurysm series ever in the surgical literature; they treated 51 ICA/AChA aneurysms in 50 patients. Aneurysms associated to AVMs were excluded from this study. Of 33 (33/50 [66%]) patients who presented with SAH, an AChA aneurysm was identified as the bleeding source in (24/33 [73%]) cases. In nine patients (27%) the SAH was caused by the rupture of another aneurysm. The AChA aneurysm was unruptured but identified as the cause of the neurologic symptoms in three (3/50 [6%]) patients; two of these three patients (66%) had ipsilateral third nerve palsy, and one (33%) had a transient ischemia in the AChA territory. Eight (8/51 [16%]) aneurysms were incidentally discovered on angiograms performed for unrelated symptoms. Six (6/51 [12%]) aneurysms were found incidentally at surgery performed for another aneurysm. Of eight patients (8/50 [16%]) who had postoperative clinical and CT evidence of an AChA territory infarction, three (37.5%) died and five (62.5%) had major neurologic deficits. Of five patients (5/50 [10%]) who presented minor surgical morbidity, there was a small frontal hematoma, a remote cerebellar hemorrhage, a surgical wound infection, a partial third cranial nerve palsy, and a subdural hygroma requiring placement of a peritoneal shunt. Surgically, the AChA patency is often difficult to confirm by microscopy because of the small size of the artery and site of origin, usually behind the ICA as viewed surgically; however, microvascular Doppler sonography (MVDS) can detect flow instantaneously even in such a small vessel. Shibata et al (4) reported that the occurrence of an AChA syndrome can be minimized by the use of MVDS; they reported three postsurgical AChA ischemic strokes in 19 (16%) patients treated for AChA aneurysm before the introduction of MVDS, but only one of 19 patients (5%) was treated with the aid of this device. More recently Suzuki et al (6) reported the use of motor-evoked potentials (MEPs) to detect blood flow impairment into the AChA. The authors relied on the intraoperative MEPs elicited by electrical stimulation of the hand motor cortex. Their study consisted in the monitoring of 108 patients with ICA aneurysms who underwent surgery; the authors found that MEPs had a sensitivity of 100% with a specificity of 85% (positive predictive value = 0.25; negative predictive value = 1).

Endovascular Treatment of AChA Aneurysms

At the beginning of the EVT era, only aneurysms that were not amenable to surgical clipping were treated by endovascular means (8, 9). In 1998, Cognard et al (10) published the results of EVT as the primary treatment for berry intracranial aneurysms even if those were amenable to a surgical approach. They performed EVT in 86% of the cases (203 of 236 aneurysms) and reported a low morbidity rate because of the technique. The thromboembolic complication rate was 4% and mortality rate due to preoperative rupture of the aneurysm was 1%. To our knowledge, there is no specific report in the literature focusing on the EVT of AChA aneurysms. In our series, one patient (1/18 [5.5%]) died as the result of aneurysm perforation during coiling. Another patient (5.5%) had a contralateral hemiparesis after the procedure without angiographic evidence of AChA occlusion on the control angiogram after aneurysm coiling. The overall complication rate of AChA aneurysm embolization was 11% (2/18). According to Friedman et al, most postoperative strokes occur in a delayed fashion, 6–36 hours after surgery (5). In our series, the hemiparesis complicating one of the 18 procedures occurred immediately after the embolization. Perhaps the surgical approach with dissection and brain tissue retraction may have played a role in the appearance of secondary edema responsible for the delayed occurrence of neurologic deficit.

In our series only, only eight patients (8/17 [47%]) had a long-term follow-up (equal or superior to 12 months). This lack of long-term follow-up in most of our patients significantly limits one's ability to draw conclusions about the long-term efficacy of EVT.

Conclusion

The EVT of AChA aneurysms is effective to protect the patients from rebleeding. The complication rate of EVT compare favorably with those of the surgical series; however, our retrospective study has inherent limitations and is probably biased by the fact that we are a tertiary center for which most of the patients are referred from other centers for endovascular treatment and not for surgery.

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