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ORIGINAL RESEARCH

Long-Term Clinical and Imaging Follow-Up of Complex Intracranial Aneurysms Treated by Endovascular Parent Vessel Occlusion

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BACKGROUND AND PURPOSE: Flow-diverting stents are increasingly being used for the treatment of complex intracranial aneurysms, but the indications for their use in lieu of traditional endovascular PVO have yet to be precisely defined. The purpose of this study was to review the clinical and imaging outcomes of patients with intracranial aneurysms treated by PVO.

MATERIALS AND METHODS: A total of 28 patients with intracranial aneurysms, treated by PVO between July 1992 and December 2009, were reviewed. Aneurysms arising from peripheral arteries were excluded. Clinical and imaging data were retrospectively analyzed from a prospectively maintained data base.

RESULTS: There were 28 patients with 28 aneurysms treated by PVO. Aneurysms of the anterior circulation presenting with mass effect ($n = 11$) or discovered incidentally ($n = 1$), and dissecting-type VB aneurysms presenting with subarachnoid hemorrhage ($n = 6$) faired the best with high obliteration rates (83.3% and 83.6%, respectively) and no permanent major ischemic complications. In contrast, VB aneurysms presenting with mass effect ($n = 7$) demonstrated the lowest obliteration rate (57.1%), the highest rate of permanent major ischemic complications (28.6%), and a high mortality rate (28.6%).

CONCLUSIONS: PVO is a safe and effective treatment for complex intracranial aneurysms of the carotid artery and dissecting-type VB aneurysms presenting with SAH. In contrast, PVO for aneurysms of the VB circulation presenting with mass effect is less efficacious and associated with significant morbidity and mortality. It is hoped that flow diverters may represent a better treatment technique for these most difficult-to-treat lesions.

ABBREVIATIONS: BTO = balloon test occlusion; ECIC = extracranial-intracranial; PCA = posterior cerebral artery; PVO = parent vessel occlusion; VA = vertebral artery; VB = vertebrobasilar

PVO is a well-established technique for the treatment of complex intracranial aneurysms not amenable to direct surgical clipping or endovascular coiling.[1,2] The recent development of low-porosity intracranial stents, or flow diverters, such as the Pipeline embolization device (ev3, Irvine, California) and Silk stent (Balt Extrusion, Montmorency, France), offers a novel therapeutic alternative for many of these same lesions.[3-6] Although initial published results indicate a generally favorable risk-benefit profile for flow diverters, early and delayed complications are increasingly reported, including rupture of previously unruptured aneurysms, ipsilateral intraparenchymal hemorrhage, and in-stent thrombosis.[7-10] Moreover, obliteration of the aneurysm is not guaranteed, especially if there is incorporation of a side branch into the aneurysm body or neck. Currently, the indications for recommending PVO versus flow-diverting stents are not known. The purpose of this study was to characterize the long-term clinical and angiographic outcomes of patients with complex intracranial aneurysms treated by endovascular PVO, focusing on complication and aneurysm obliteration rates.

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Materials and Methods

At the Toronto Western Hospital, clinicoradiographic and treatment data on intracranial aneurysms are prospectively collected and maintained in a data base. A retrospective review was performed to identify all consecutive patients with intracranial aneurysms treated at our institution by endovascular PVO between July 1992 and December 2009. Patients with unruptured and ruptured intracranial aneurysms were included. Patients who underwent open surgical PVO or endovascular PVO supplemented by ECIC bypass were excluded. Patients harboring aneurysms arising on distal peripheral branches or associated with AVMs were also excluded. Data base records were verified and supplemented by a review of electronic charts. Clinical follow-up was obtained from chart notes dictated at our multidisciplinary clinic.

The decision to undertake PVO for the treatment of complex intracranial aneurysms was always made a priori by a multidisciplinary team consisting of interventional neuroradiologists and cerebrovascular neurosurgeons, and never the unintentional result of a procedural complication. The decision was informed by an assessment of the patient's collateral circulation that always consisted of a detailed anatomic review of the completeness of the circle of Willis. In addition, for unruptured aneurysms, a BTO was performed under local anesthesia. To be deemed amenable for PVO, the patient had to pass on clinical (no neurologic deficit) and angiographic (delay in cortical venous drainage less than 3 seconds between occluded and contralateral hemispheres) grounds.[11] Technetium Tc99m–labeled hexamethylpropyleneamine oxime SPECT (relative quantification) and a hypotensive challenge during BTO were not routinely performed. For patients presenting with dissecting-type VA pseudoaneurysms and SAH, a BTO was not performed. In these cases, to be deemed amena-

Table 1: PVO for anterior circulation aneurysms: demographics, follow-up, and complications

Case No.	Age (yr)/Sex	Location	Size (mm)	Presentation	FU (days)	Aneurysm Obliterated	Complications/Comments
1	85/F	Cavernous	32	H/A, visual loss	n/a	No	Balloons deflated, infarction without clinical deficit, treatment abandoned
2	56/F	Cavernous	17	Visual loss	65	Yes	None
3	62/F	Cavernous	29	H/A, CN palsy	115	Yes	Transient Horner syndrome
4	62/F	Cavernous	23	H/A, CN palsy	236	Yes	Partial deflation of proximal balloon, no clinical sequelae
5	58/F	Cavernous	27	H/A, CN palsy	n/a	No	Partial deflation of proximal balloon, cortical infarction with minor clinical deficit, early aneurysm growth provoked surgical trapping with STA-MCA bypass
6	68/F	Cavernous	35	H/A, CN palsy	867	Yes	None
7	39/M	Cavernous	30	H/A, CN palsy	4914	Yes	Growth of posterior circulation aneurysm
8	52/F	Cavernous	30	H/A, CN palsy, visual loss	6539	Yes	Growth of contralateral (mirror) cavernous aneurysm
9	60/F	Parophthalmic	14	Incidental	181	Yes	None
10	62/F	Parophthalmic	25	Visual loss	416	Yes	None
11	64/F	Parophthalmic	39	Cognitive decline, hydrocephalus	420	Yes	None
12	74/F	Parophthalmic	16	Visual loss	484	Yes	None

Note:—CN indicates cranial nerve; H/A, headache; STA-MCA = superficial temporal artery-middle cerebral artery.

ble for PVO as first-line therapy, a subjective assessment was made based on the 1) size of the contralateral VA, and 2) completeness of the circle of Willis.

PVO was conducted with the patient under general anesthesia. Activated clotting time was maintained between 250 and 300 seconds by systemic anticoagulation with heparin. PVO was performed by coil embolization or by using detachable balloons at the discretion of the angiographer; however, in general, coil embolization was preferred for dissecting-type pseudoaneurysms and detachable balloons were favored for large and giant aneurysms presenting with mass effect. The following types of coils were used: GDC coils (Boston Scientific, Natick, Massachusetts), MicroCoils (Micrus, San Jose, California), Trufill DCS coils (Cordis, Miami Lakes, Florida), MicroPlex coils (MicroVention, Aliso Viejo, California), and HydroCoils (MicroVention). In the case of dissecting-type VB and PCA pseudoaneurysms, initial coil loops were deposited within the pseudoaneurysm to serve as an anchor, and coiling continued proximally to cover the entire dissected segment. For permanent ICA or VA occlusion using detachable balloons (Goldbal; Balt Extrusion, Montmorency, France), a first balloon was detached in close proximity to the aneurysm, followed by a second, more proximally situated "safety" balloon. We routinely pretreated patients with unruptured aneurysms with 325 mg of aspirin daily for 5 days, followed by an additional 4–8 weeks.

Radiologic follow-up was initially performed by DSA with 3D-rotational angiography but was supplanted over time by MRA using the auto-triggered elliptic-centric-ordered 3D gadolinium-enhanced technique. We have previously reported on this MR imaging protocol and its utility in the surveillance of coiled aneurysms.[12–14] CTA was also utilized, as dictated by clinical circumstances. Clinical evaluations were performed at our multidisciplinary clinic, attended by interventional neuroradiologists and cerebrovascular neurosurgeons. The timing and frequency of clinicoradiographic follow-up for these complex lesions were variable and determined on a case-by-case basis.

Results

Between July 1992 and December 2009, endovascular PVO was attempted on 28 patients with 28 complex intracranial aneurysms. For the analysis, we divided our cohort into 2 groups based on aneurysm location: anterior circulation (Table 1) and posterior circulation (Table 2).

Anterior circulation aneurysms presented with symptoms of mass effect ($n = 11$) or were discovered incidentally ($n = 1$). These comprised cavernous ($n = 8$) and paraophthalmic ($n = 4$) aneurysms. The mean age of patients was 61.3 ± 11.3 years (range 39–84 years). Most of the patients were female (11 of 12 patients, 91.7%), and most of the aneurysms were large and giant in size (mean 26.4 ± 7.8 mm, range 14–39 mm). Excluding patients 1 and 5, due to treatment failure, clinical follow-up was available for a mean of 3.9 ± 6.3 years (range 65 days to 17.9 years). The last imaging follow-up demonstrated nonfilling of the aneurysm—that is, obliteration—in most of the patients (10 of 12 patients, 83.3%) (Tables 1 and 3). Universally, these patients reported some degree of symptom relief, sometimes dramatic, with no patients reporting progression of presenting symptoms. As mentioned, there were 2 early treatment failures. Patient 1 was an 85-year-old woman who presented with progressive left monocular vision loss, secondary to an enlarging left giant cavernous aneurysm. When her right-sided (contralateral) vision was affected, a decision was made to treat with endovascular PVO of her left carotid artery. Detachable balloons were positioned just proximal to the aneurysm and in the petrous segment. Early postoperative imaging before discharge, however, demonstrated deflation of both Goldbal balloons with their migration into the aneurysm sac. There was no change in the degree of aneurysm filling. The patient declined any further interventions. Patient 5 was a 58-year-old woman who presented acutely with right-sided retroorbital pain and a cavernous sinus syndrome, secondary to a giant cavernous aneurysm. Upon detachment of the first Goldbal balloon in the petrous segment, it partially deflated, requiring the deposition of an additional 2 balloons. Immediately postoperatively, the patient experienced right hemispheric TIAs, with persistent filling of the aneurysm via the ophthalmic artery. The TIAs persisted despite full systemic herparinization. Follow-up MR imaging/MRA performed 1 week later showed new infarcts in the right carotid territory and interval growth of the filling portion of the aneurysm. The

Table 2: PVO for posterior circulation aneurysms: demographics, follow-up, and complications

Case No.	Age (yr)/Sex	Location	Size (mm)	Presentation	FU (days)	Aneurysm Obliterated	Complications/Comments
13	44/F	VB	7	SAH	63	Yes	None
14	52/F	VB	13	SAH	185	Yes	None
15	63/M	VB	5	SAH	656	Yes	None
16	60/F	VB	6	SAH	729	Yes	None
17	50/F	VB	8	SAH	n/a	No	Persistent filling of aneurysm after proximal VA occlusion, large PICA incorporated into dissecting pseudoaneurysm, underwent surgical trapping and PICA-PICA bypass
18	51/F	VB	5	SAH	1379	Yes	None
19	64/F	VB	25	Brain stem compression, H/A	515	Yes	None
20	55/F	VB	26	CN palsy	794	Yes	Lateral medullary infarction with clinical deficit
21	44/M	VB	34	Brain stem compression	1839	Yes	None
22	19/M	VB	20	Brain stem compression	2123	Yes	Brain stem infarction with clinical deficit
23	58/F	VB	26	Brain stem compression	3360	No	Transient episode of "tunnel vision"
24	67/F	VB	20	Brain stem compression, cognitive decline	n/a	No	Vessel rupture, death
25	74/M	VB	28	Brain stem infarction	n/a	No	Brain stem and cerebellar infarctions, death
26	54/M	PCA	20	Incidental	213	Yes	None
27	30/M	PCA	29	Incidental	770	Yes	Transient hemisensory and motor deficits
28	32/F	PCA	26	Incidental	2439	No	Transient homonymous hemianopsia

Note:—CN indicates cranial nerve; H/A, headache.

Table 3: PVO for complex intracranial aneurysms: summary of obliteration rates, morbidity, and mortality

	Obliteration Rate (%)	Mortality (%)	Permanent Major Deficit (%)	Permanent Minor Deficit (%)
Anterior circulation aneurysms	83.3	0.0	0.0	8.3
Posterior circulation aneurysms presenting with SAH	83.6	0.0	0.0	0.0
Posterior circulation aneurysms presenting with mass effect	57.1	28.6	28.6	0.0
PCA aneurysms discovered incidentally	67.7	0.0	0.0	0.0

patient subsequently underwent successful ECIC bypass and surgical trapping of the aneurysm. Although no patients suffered a major permanent neurologic deficit, a single patient suffered a minor permanent neurologic deficit (patient 5, hand clumsiness), and a single patient had a transient Horner syndrome (patient 3) (Tables 1 and 3). Interestingly, the 2 patients with the longest follow-up (patients 7 and 8) demonstrated interval growth of known, but untreated, complex basilar termination and mirror cavernous aneurysms, respectively, after successful PVO.

Patients with posterior circulation aneurysms were divided into 3 subgroups based on aneurysm location and mode of presentation: VB aneurysms presenting with SAH ($n = 6$); VB aneurysms presenting with mass effect ($n = 7$); and PCA aneurysms discovered incidentally ($n = 3$) (Tables 2 and 3). Considering patients with VB aneurysms presenting with SAH, the mean age was 52.7 ± 6.9 years (range 44–63 years), most were female (5 of 6 patients, 83.3%), and all demonstrated relatively small dissecting-type pseudoaneurysms of the VAs (mean 7.3 ± 3.0 mm, range 5–13 mm). Most of the PVO procedures were performed with coils (5 of 6, 83.3%). The last imaging follow-up demonstrated aneurysm obliteration in most of the patients (5 of 6 patients, 83.3%) (Tables 2 and 3). A single patient (patient 17) demonstrated immediate persistent filling of a ruptured, dissecting-type right vertebral artery pseudoaneurysm after proximal PVO. Notably, the dissected segment involved the origin of a large right PICA. The patient underwent successful surgical trapping supplemented by a PICA-PICA bypass. There was no neurologic morbidity associated with PVO in this subgroup. Excluding patient 17, clinical follow-up was available for a mean 1.7 ± 1.4 years (range 63 days to 3.8 years). There were no instances of recurrent SAH during the follow-up period.

Considering patients with VB aneurysms presenting with mass effect, the mean age was 54.2 ± 18.5 years (range 19–75 years), only slightly more than half were female (4 of 7 patients, 57.1%), and all demonstrated partially thrombosed large and giant aneurysms with brain stem compression (mean 25.8 ± 4.8 mm, range 20–34 mm). Two patients (patients 24 and 25) died as the result of PVO (2 of 7 patients, 28.6%). Patient 24 was a 67-year-old woman who presented with a 2-year history of progressive cognitive decline. An MR imaging/MRA demonstrated a large partially thrombosed aneurysm arising from the left VA, with significant brain stem compression. Upon inflation of a Goldbal detachable balloon in the proximal right VA, a rapid conformational change in the balloon was accompanied by an acute rise in blood pressure. These findings were suggestive of vessel rupture. Despite emergent salvage maneuvers, the patient was declared brain dead shortly thereafter. Postprocedural imaging demonstrated massive SAH. Patient 25 was a 74-year-old man who presented with an acute-onset right-sided facial palsy and crossed arm and leg weakness. These clinical deficits corresponded to a pontine infarct that probably resulted from com-

pression of pontine perforators by a giant partially thrombosed aneurysm of the VB system. There was associated severe compression/deformation of the pons. A planned PVO of the dominant right VA was performed with coils. On final angiographic runs, however, there remained significant, persistent filling of the aneurysm via the left VA. Unfortunately, the patient awoke with new cerebellar and brain stem findings, including pronounced dysphagia. He died 4 days later from aspiration pneumonia. Excluding patients 24 and 25, clinical follow-up was available for a mean 4.7 ± 3.1 years (range 1.4–9.2 years). The last imaging follow-up demonstrated nonfilling of the aneurysm in most of the surviving patients (4 of 5 patients, 80%). Two of these patients, however, suffered PVO-related brain stem infarcts with associated major permanent neurologic deficits (2 of 5 patients, 40%). The single patient with persistent, albeit significantly reduced, filling of a giant, partially thrombosed basilar termination aneurysm demonstrated marked improvement in her presenting clinical symptoms (dysarthria, dysphagia, ataxia, and inappropriate affect)—a clinical improvement sustained over 9.2 years.

Considering patients with PCA aneurysms discovered incidentally, the mean age was 38.3 ± 13.3 years (range 30–54 years), and only 1 of 3 patients (33.3%) was female. The mean size of aneurysms was 25.0 ± 4.6 mm (range 20–29 mm). Clinical follow-up was available for a mean 3.1 ± 3.2 years (range 213 days to 6.7 years). The last imaging follow-up demonstrated nonfilling of the aneurysm in 2 of 3 patients (66.7%) (Tables 2 and 3). A single patient with persistent filling of a giant partially thrombosed PCA aneurysm underwent a complex treatment course, characterized by surgical clipping under hypothermic circulatory arrest, progressive growth of the dysplastic parent PCA vessel, PVO with coils complicated by multiple, transient episodes of homonymous hemianopsia, and, ultimately, repeat surgical clip reconstruction. The aneurysm, albeit significantly reduced in size, continued to fill but remained stable over a follow-up period of 6.7 years. None of the 3 patients in this subgroup suffered permanent neurologic deficits.

Discussion

For a majority of aneurysms, endovascular coiling or surgical clipping represent definitive management options for the exclusion of intracranial aneurysms from the circulation. However, complex aneurysms are not easily amenable to either treatment technique and pose significant challenges for interventional neuroradiologists and cerebrovascular neurosurgeons. Examples of such difficult-to-treat aneurysms include large and giant aneurysms presenting with mass effect, dissecting-type pseudoaneurysms, fusiform aneurysms, and blister aneurysms. An alternative treatment strategy for these lesions is PVO, popularized by Drake and colleagues at the University of Western Ontario.[1,2] Although the technique can be extremely effective in obliterating aneurysms not amenable to a direct endovascular or surgical approach, the risk of immediate or delayed cerebral ischemia remains an ever-present specter over this destructive procedure. Recently, low-porosity stents, or flow diverters, have become available for clinical use and represent a novel reconstructive therapeutic strategy for these most complex vascular lesions.[3-6] Although initial reports documented high obliteration and low complication rates, aneurysm obliteration is not always achieved, and unanticipated, delayed complications are increasingly reported, such as rupture of previously unruptured aneurysms, remote intraparenchymal hemorrhage, perforator ischemia, and in-stent thrombosis.[7-10,15] It is in this context that we sought to retrospectively review our experience with endovascular PVO for the management of complex intracranial aneurysms that may also be candidates for treatment with next-generation flow-diverting stents.

Large and Giant ICA Aneurysms Presenting with Mass Effect

The management of large and giant ICA aneurysms presenting with mass effect remains controversial. For example, in the largest published cohort of carotid cavernous aneurysms, Stiebel-Kalish et al[16] reported that endovascular PVO did not alter the patient's final diplopia compared with observation alone, but only that it reduced the incidence and severity of facial pain. Consistent with this report, our policy has been to offer treatment for carotid cavernous aneurysms only for debilitating pain, visual loss from aneurysmal compression, and progressive symptoms. In our study, 12 patients underwent PVO with detachable balloons for the treatment of large and giant ICA aneurysms (8 cavernous, 4 paraophthalmic) presenting with mass effect ($n = 11$) or discovered incidentally ($n = 1$). Obliteration of the aneurysm was achieved in 10 of 12 patients (83.3%). All successfully treated patients reported some degree of clinical improvement, sometimes dramatic, with near complete resolution of presenting symptoms. Complications were uncommon and minor, and were most often related to technical difficulties during deployment of the detachable balloons. These clinical and radiographic results are in agreement with those from other high-volume centers,[16-18] as well as the classic report by Drake, Peerless, and Ferguson,[1] describing their early experience with Hunterian proximal arterial occlusion for the management of giant aneurysms of the carotid circulation. A recent systematic review of endovascular PVO for carotid cavernous aneurysms confirmed low complication and high aneurysm obliteration rates.[19] These results represent important benchmarks for efficacy and safety in the management of these complex lesions. It is interesting to note that 2 patients with the longest follow-up (>10 years) in our series demonstrated interval growth of known intracranial aneurysms after PVO. PVO is probably a risk factor for the growth of untreated and de novo intracranial aneurysms over time.[17,20] This clinical nuance should be considered when contemplating PVO, especially in younger patients.

Dissecting-Type Vertebral Artery Pseudoaneurysms Presenting with SAH

Dissecting-type vertebral artery pseudoaneurysms are rare causes of SAH, with a particularly sinister natural history.[21] For example, Mizutani et al[22] reported a rebleeding rate of 69% in unsecured lesions, with more than half of the rebleeds occurring in the first 24 hours after the initial SAH. Due to an underlying fragility of the vessel wall and requirement to treat the entire diseased vessel segment (and not just the aneurysmal pouch), direct surgical approaches have been largely abandoned in favor of endovascular PVO by most of the cerebrovascular community, except in cases of unusual complex-

ity.[23] In our study, 6 patients underwent PVO with coils for the treatment of ruptured dissecting-type vertebral artery pseudoaneurysms. Complete aneurysm obliteration was achieved in 5 of 6 patients (83.3%), with no new neurologic deficits. No patients had recurrent SAH. These results are in keeping with the published results of others.[24-29] A single patient in our series demonstrated immediate persistent filling of the ruptured pseudoaneurysm after proximal PVO. Importantly, the dissected vessel segment involved the origin of the PICA. The patient underwent successful surgical trapping supplemented by a PICA-PICA bypass. When the origin of the PICA is involved with the dissected vessel segment, or there is involvement of a dominant vertebral artery in an isolated vertebrobasilar circulation, more sophisticated therapeutic options must be considered first-line; for example, surgical trapping supplemented by vascular bypass, or more recently, endovascular stent placement using stent monotherapy, a stent-in-stent technique, or flow diverters.[21,24,30-34]

Large and Giant VB Aneurysms Presenting with Mass Effect

Unruptured large and giant VB aneurysms have a grave natural history, justifying more aggressive therapeutic approaches. In the International Study of Unruptured Aneurysms, the cumulative 5-year rupture rate for giant posterior circulation aneurysms was 50%.[35] Those presenting with mass effect portend a particularly poor prognosis.[36] Even in the most experienced cerebrovascular centers, treatments (surgical, endovascular, and combined) are associated with significant morbidity and mortality.[2,37-42] In a unique clinical series in its scope, Steinberg, Drake, and Peerless[2] reported on 201 patients with unclippable VB aneurysms treated by deliberate PVO, nearly half of which presented with mass effect and brain stem compression. It is this subgroup that demonstrated the worst clinical outcomes, with only 60.5% of patients reported as having excellent to good outcomes and an associated 33% mortality rate. Recently, Darsaut et al[37] performed a retrospective review of 184 very large and giant intracranial aneurysms treated by a multitechnique team at Stanford University Medical Center between 1984 and 2008—a series that included 44 posterior circulation aneurysms. Overall, they reported good clinical outcomes in 69% of patients and a total aneurysm-related mortality rate of 16.9%. Consistent with previous reports, they concluded that patients with poor baseline functional status, giant aneurysm size, and posterior circulation location were all predictors of poor clinical outcome. Similarly, in the largest reported series of giant intracranial aneurysms treated using endovascular techniques, including PVO, morbidity and mortality rates were high (26% and 29%, respectively).[38] In our study, 7 patients with large and giant VB aneurysms presenting with mass effect were treated by endovascular PVO. We report comparable morbidity and mortality rates (28.6% and 28.6%, respectively) for this most sinister subgroup of aneurysms. Note is made that we routinely used intravenous decadron pre-, intra-, and postoperatively in this group of patients in an attempt to mitigate cerebral swelling related to progressive thrombosis of the aneurysm after PVO. Complete aneurysm obliteration was only achieved in 57.1% of treated patients. The largest clinical reports on the use of flow diverters in the treatment of complex intracranial aneurysms have disproportionately included anterior circulation aneurysms.[3-6] It is hoped that this newer technology can improve treatment outcomes for VB aneurysms presenting with mass effect, as current management paradigms leave considerable room for improvement.

Unruptured Giant PCA Aneurysms

PCA aneurysms are rare, comprising less than 1% of all intracranial aneurysms. Their management is often challenging, especially when large or giant in size.[43] Given the difficulties in surgical access to the region and the eloquence of proximal perforators to the brain stem and thalamus, endovascular PVO represents a mainstay of modern therapy.[41,44] For example, a recent report by Chang et al[45] from the Barrow Neurologic Institute concluded that bypass techniques for the treatment of distal PCA aneurysms were associated with a higher-than-expected rate of complications and should be reserved for exceptional circumstances. In the same study, primary coiling of these aneurysms was associated with high rates of recurrence, probably reflecting the underlying dissecting nature of many of these lesions and inadequate treatment of this aneurysm-targeted strategy.[23,45,46] Typically, PVO is well tolerated, given the rich anastomotic collateralization from the distal ACA and MCA territories.[47] In our study, only 3 patients with giant PCA aneurysms were treated by endovascular PVO. Consistent with the results of others,[47-49] the procedure proved to be generally safe and effective at treating these challenging aneurysms. We failed to completely obliterate a giant PCA aneurysm in a single patient who underwent a complex treatment course involving multiple endovascular and surgical procedures, with significant reduction in the size of the aneurysm and radiographic stability over a prolonged follow-up period of 6.7 years. No patients with large or giant PCA aneurysms presented with SAH in our series. Nonetheless, it can be anticipated that the risk of cerebral ischemia after PVO is greater in patients with SAH when vasospasm can jeopardize collateral flow, ie, perfusion of distal PCA branches via MCA collaterals. For example, Kashiwazaki et al[50] recently reported their results for the endovascular management of 21 ruptured and unruptured PCA aneurysms with selective aneurysm coiling ($n = 12$) and PVO ($n = 9$). Cerebral infarction was observed only in cases of PVO for acutely ruptured PCA aneurysms.

Study Limitations

Our study has some important limitations. First, it represents a retrospective review of a prospectively maintained data base, with all the inherent biases of this type of study design. Second, clinical outcomes were assessed by the treating neurointerventionalists and cerebrovascular neurosurgeons in the clinic using a nonstandardized approach, rather than by an independent third-party neurologist or neuro-ophthalmologist. Third, aneurysms treated by using surgical modalities or reconstructive endovascular techniques were not included in this study population. This study therefore only represents a snapshot of the complications and outcomes of endovascular PVO for the treatment of complex aneurysms. Whether patients would have better outcomes using a different management approach was not addressed in the current study. Finally, the 19-year study period has witnessed significant improve-

ment in endovascular and microsurgical techniques. Whether novel treatments, such as flow diverters, for these complex lesions would result in better outcomes is currently not known.

Conclusions

PVO is a safe and effective treatment for complex intracranial aneurysms of the carotid artery, dissecting-type VB aneurysms presenting with SAH, and large and giant PCA aneurysms. In contrast, PVO for aneurysms of the VB circulation presenting with mass effect is less efficacious and associated with significant morbidity and mortality. It is hoped that flow diverters may represent a better treatment technique for these most difficult-to-treat lesions.

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