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Citation	Acta Neurochirurgica, 153(11), 2151-2158 <a href="https://doi.org/10.1007/s00701-011-1111-5">https://doi.org/10.1007/s00701-011-1111-5</a>
Issue Date	2011-11
Doc URL	<a href="http://hdl.handle.net/2115/50371">http://hdl.handle.net/2115/50371</a>
Rights	The original publication is available at <a href="http://www.springerlink.com">www.springerlink.com</a>
Type	article (author version)
File Information	AN153-11_2151-2158.pdf



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# Endovascular treatment for aneurysms of the posterior cerebral artery:12 years experience with 21 cases.

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## **Abstract**

**Background and Purpose:** To discuss and summarize the strategies and complications of endovascular embolization for aneurysms of the posterior cerebral artery (PCA).

**Methods:** Data of patients with PCA aneurysms treated by an endovascular procedure were analyzed retrospectively (n = 21). Twenty patients with aneurysms were treated by detachable coil embolization, and 1 patient was treated with n-butyl cyanoacrylate. Of the 21 aneurysm embolization cases, 9 were treated by parent artery occlusion (PAO), and 12 were treated by selective occlusion of the aneurysm (SOA).

**Results:** All 12 aneurysms treated by SOA showed complete occlusion. Two aneurysms became recanalized 6 months after the first embolization and were then re-embolized; complete healing was observed on follow-up angiography. All patients showed acceptable outcomes without any procedural complications, except 1 patient who died 2 days after treatment. PAO resulted in 100% occlusion of all aneurysms. Cerebral infarction was noted in most patients (78%). However, the area of infarction was small. Permanent neurological deficit was observed in 2 patients (22%), but their condition was not critical.

**Conclusions:** Aneurysm embolization with SOA is well indicated for saccular aneurysms with well-defined necks, whereas PAO carries a risk of ischemic complications. Although the PCA is rich in collateral circulation, ischemic complications were noted in most patients after PAO, and it was difficult to predict occurrence of these complications. However, the area of cerebral infarction tended to be small, and the neurological deficits observed were not critical.

**Key words:** aneurysm, endovascular treatment, posterior cerebral artery, parent artery occlusion.

## **Introduction**

Aneurysms of the posterior cerebral artery (PCA) are rare, constituting approximately 0.7–2.3% of all intracranial aneurysms [7, 9, 11, 14]. Surgical approaches to the treatment of PCA aneurysms are complicated by their anatomy [12, 19–22]; however, intravascular surgery is useful for repairing aneurysms in this region. In this study, patient outcomes after endovascular surgery for PCA aneurysms were investigated retrospectively. The application and associated limitations of the technique are discussed.

## **Methods**

### *Patients*

During a 12-year period, from 1998 to 2010, we retrospectively analyzed data of all patients with PCA aneurysms who were treated with an endovascular procedure. Patients with aneurysms of the posterior communicating artery were excluded from the study. In all, 21 patients with aneurysms were treated with an endovascular procedure (7 men and 14 women; age, 33–78 years; mean age, 60.9 years). Of the 21 aneurysms, 15 were saccular and 6 were fusiform. Clinically, 15 patients were diagnosed with subarachnoid hemorrhage (SAH), 1 patient was diagnosed with mass sign, and 5 patients were asymptomatic. One patient had PCA aneurysm associated with moyamoya disease, 1 had an unruptured arteriovenous malformation, and 1 had a dural arteriovenous fistula. The neurological status of all the patients was recorded at admission by using the Hunt and Kosnik grading system (H–K grade). Follow-up data were collected using the most recent outpatient records. Clinical outcomes were assessed by 2 experienced clinicians using the Glasgow outcome scale. Conventional angiography or magnetic resonance imaging (MRI) time-of-flight angiography was performed at 6–12 months to determine whether lesions had reduced or healed. Thereafter, additional image evaluation was performed annually, if required. In particular, parent artery occlusion (PAO) cases were evaluated by computed tomography (CT), MRI, and the assessment of neurological symptoms in detail, 1 day and 7 days after the treatment. Aneurysm locations were classified as follows: P1 (3 cases), P1–2 (4 cases), P2 (5 cases), P2–3 (4 cases), P3 (2 cases), and P4 (3 cases; Table 1). All procedures were performed under general anesthesia. In all cases, 3000 IU of heparin was administered at the start of the procedure, followed by 1000 IU every hour until completion of the procedure.

### *Strategy of intervention*

Diagnostic angiography was performed in all patients before embolization. Selective occlusion of the aneurysm (SOA) was the preferred procedure for all the patients. In cases where it was difficult to perform SOA safely (because of thrombosed, small, fusiform, berry, or dissecting aneurysms), or the location of the aneurysm was distal to the P2a segment, embolization was performed by PAO. PAO was performed by placing coils in both the aneurysm sac and the parent artery. PAO was carried out at sites

proximal and distal to the neck of the aneurysm. The smallest possible length of the artery was occluded using detachable coils. In the case of fusiform aneurysm, embolization was performed in the aneurysmal dilatation. Coils were selected based on the size of the artery to be occluded. All PAO interventions were performed without the involvement of surgical bypass procedures.

#### *Anatomy of the posterior cerebral artery*

In this study, PCA anatomy was classified as described by Drake et al. [10]. The PCA is divided into 4 segments: P1 arises from the basilar artery to the junction with the posterior communicating artery, P2 extends to and includes the first major branch on the side of the midbrain, P3 runs within the perimesencephalic cistern up to the origin of the parieto-occipital and calcarine arteries, and P4 represents the termination of the artery.

#### **Results**

We identified 21 patients with PCA aneurysms. Of these, 12 patients were treated by SOA, and 9 patients, by PAO. The results are summarized in Table 1.

#### *Selective occlusion of the aneurysm*

All of the 12 aneurysms treated by SOA showed complete radiographic occlusion. No technique-associated complications were noted. Two cases (16.7%) became recanalized 6 months after the first embolization. Both of these cases were re-embolized and showed complete healing in the follow-up angiogram. One of the 12 SOA cases was a large aneurysm, with oculomotor palsy as a mass sign. After SOA, the patient's double vision disappeared. Seven patients presented with SAH, and 4 were asymptomatic. The outcomes of all of the patients in the 12 cases were favorable, except 1 (patient in case 8), who presented with SAH H–K grade 4 and had severe disability (SD).

#### *Parent artery occlusion*

All patients showed acceptable outcomes without any procedural complications, except 1, who died 2 days after treatment. This was caused by increased intracranial pressure. PAO resulted in 100% occlusion of all the aneurysms. Although the PCA is rich in collateral circulation, cerebral infarction was noted in most patients (78%). However, the area of infarction was small. Permanent neurological deficit occurred in 2 cases (22%), but they were not critical. The aneurysm types treated by PAO were thrombosed (n = 3), fusiform (n = 1), saccular (n = 3), dissection (n = 1), and berry (n = 1).

#### *Illustrative cases*

**Case 6:** A 66-year-old woman was diagnosed with SAH (H–K 1), with large thrombosed aneurysms.

MRI was useful in the diagnosis of SAH. Cerebral angiography revealed a large aneurysm of the left P2 segment. The patient underwent PAO of the P2 segment in areas immediately proximal and distal to the aneurysm and in the aneurysm sac; this technique was performed using a detachable coil for re-rupture prevention. Radiographically, the aneurysm was occluded, and postoperative MRI revealed cerebral infarction in the PCA territory (thalamus and occipital lobe). The patient had incomplete homonymous hemianopsia and numbness of the fingers. After 2 years, follow-up magnetic resonance angiography (MRA) showed no evidence of subsequent aneurysm (Fig. 1).

**Case 7:** A 69-year-old woman with sudden headache and vomiting was diagnosed with SAH (H-K 4). CT images indicated SAH. Cerebral angiography revealed a small aneurysm of the right P3 segment. PAO was performed using a detachable coil directly proximal and distal to the aneurysm and in the aneurysm sac. Although postoperative CT images showed cerebral infarction in the occipital lobe, the patient's vision was clinically preserved (Fig. 2).

**Case 5:** A 74-year-old woman had a chief complaint of double vision. MRI showed a large aneurysm compressing the midbrain. Neurologically, oculomotor nerve palsy was observed. Complete occlusion was noted after SOA using a detachable coil. Three months after the treatment, oculomotor palsy had disappeared. Follow-up MRA showed no evidence of aneurysm recurrence (Fig. 3).

## **Discussion**

### *Selective occlusion of the aneurysm*

Since the international subarachnoid aneurysm trial (ISAT) [16], endovascular coiling is increasingly used as the primary treatment option for indicated ruptured or unruptured aneurysms. In the present study, 2 cases became recanalized and were retreated without complication. SOA for the treatment of PCA aneurysms resulted in favorable outcomes. Generally, surgical approaches to the treatment of PCA aneurysms are anatomically difficult, and the perforating branches of the P1 segment are vulnerable to injury. Some studies do suggest the use of surgical techniques and a skull-base approach, but direct surgery continues to remain a challenge [12, 19–22]. These observations suggest that SOA should be the first choice for the treatment of saccular aneurysms of the PCA. Clinical studies have yielded favorable findings for endovascular treatment, which preserves the parent artery in this lesion type [6, 18, 23]. However, symptomatic aneurysms of the PCA, especially those of the P2 or distal segments, tend to be larger and thrombosed [11]. SOA in these areas, compared to SOA for aneurysms located in the P1 segment, is less safe. This discrepancy is attributable to embryological differences. The P1 segment of the PCA and the basilar artery have identical origins, whereas the P2 and distal segments belong to the so-called “true PCA.” However, treatment in cases of giant aneurysms in the P1 segment continues to be challenging.

### *Parent artery occlusion*

Biondi et al. reported that endovascular occlusion of the parent artery appears to be a relatively safe and effective technique in the treatment of peripheral giant and large aneurysms [2]. Hallacq et al. discussed the safety of PAO as a treatment for aneurysms in the P2 segment [13]. In the literature, 6 series (38 cases) report the use of endovascular PAO for PCA aneurysms by using bare metal coils [1, 2, 6, 13, 23, 25] (Table 2).

**Radiological results:** In our series, PAO resulted in 100% occlusion of all the aneurysms. As in previous studies, no patient presented with recanalization after PAO using detachable coils. These results suggest that PAO is an effective method to obliterate PCA aneurysms.

**Clinical results:** Seven of the 9 patients who underwent PAO (78%) had cerebral infarction after the treatment. This included 5 patients with infarction in the occipital lobe, and 2 patients with infarction in the occipital lobe and thalamus. Although the PCA is rich in collateral circulation, cerebral infarctions were noted in most patients, and it was difficult to predict their occurrence. However, the area of cerebral infarction after PAO tended to be small. Some studies have indicated the relative safety and a low probability of permanent neurological deficit after PAO [1, 8, 13, 17], but few radiological studies have elucidated the area of cerebral infarction after PAO [18]. We believe that our strategy of occluding the smallest possible length of the artery, by placing coils directly proximal and distal to the aneurysm and within the aneurysm sac, is not different to previously reported strategies. PAO has the potential to induce cerebral infarction, detected by MRI, both with and without neurological deficit. In our study, most cerebral infarction occurred in the occipital lobe. However, 1 patient (case 6) had thalamic infarction due to PAO at P2a, resulting in occlusion of the thalamogeniculate artery arising from P2 segment. Previously, 3 cases of thalamic infarction after PAO have been reported (1 case in a P2 aneurysm, 2 cases in P2–3 aneurysms). Most neurological deficits consisted of homonymous hemianopsia. In this study, 1 patient had homonymous hemianopsia, and 1 patient had homonymous hemianopsia and numbness as the permanent deficits (22% of cases). These findings suggest that PAO is a comparatively safe procedure for the treatment of aneurysms located in or distal to the P2 segment, and that SOA cannot be performed safely at a site distal to the P2 segment. The use of PAO should be limited to cases that require interventional treatment. In cases of flow-related aneurysms in unruptured brain arteriovenous malformation (BAVM) or dural arteriovenous fistula (DAVF), and small dissecting aneurysms that have the potential for spontaneous healing, it is necessary to obtain sufficient data about the risks and benefits of the method before its use.

#### *Aneurysm sac embolization in parent artery occlusion*

In the current study, coils were placed in both the parent artery and the aneurysm sac during PAO in all cases. Five previous studies report performing PAO in this way. Only in 1 study, conducted by Biondi et al., occlusion of the parent artery without aneurysm sac occlusion was performed for the treatment of large and giant aneurysms. When considering the angiographic results, there are no differences between

PAO carried out with and without aneurysm sac embolization (all cases presented no recanalization). There were no statistically significant differences in permanent deficit between the 2 methods (Fisher's exact test,  $p = 0.372$ ). Boindi et al. suggested that elective PAO at the level of the aneurysm without the insertion of coils into the aneurysm could increase shrinkage and resorption of giant aneurysms. Therefore, the treatment of giant or large aneurysms with PAO without aneurysm sac embolization may be beneficial; however this issue remains controversial.

#### *The choice between open surgery and endovascular treatment*

Aneurysm of the proximal segment of the PCA can be treated by a surgical approach normally used for basilar top aneurysms. However, aneurysms in very high or low positions make open surgery difficult. In this segment, the perforating artery supplying the brain stem must be protected. Pterional, subtemporal, combined pterional and subtemporal, and posterior interhemispheric surgical approaches have been reported for aneurysms located in or distal to the P2 segment. However, the use of these approaches is still controversial owing to their associated anatomical challenges. For these reasons, endovascular treatment remains a useful intervention for aneurysm of the PCA. Previous studies have indicated the safety of PAO in this segment [1, 8, 13, 17]. However, some patients develop visual field loss after PAO. In other groups, open bypass was performed to prevent visual field loss. During endovascular treatment, open bypass presents a dual risk. The necessary anticoagulation therapy has hemorrhagic complications, and endovascular treatment itself is associated with embolic complications. When considering these points, open surgery does appear to be beneficial, because treatment is completed by 1 surgery.

#### *Open Bypass*

Microsurgical cerebral revascularization techniques have been used in some studies to prevent post-PAO ischemic complications [3, 4, 15, 24]. Chang et al. discussed the risk of bypass and stated that in cases of aneurysms of the distal PCA, clinicians should limit the number of bypasses by improving patient selection (via balloon test occlusion [BTO], stent-coiling, and assessing the patient's neurological status) [5]. BTO in the PCA is technically challenging, but it is feasible. Bypassed flow can only contribute to the prevention of visual field loss. Because it is unclear whether bypass flow contributes to the perforating artery, the problem of thalamic infarction associated with PAO in the P2 segment remains unresolved. Moreover, the bypass procedure itself may interfere with the collateral anastomosis. On the other hand, visual field loss is severely debilitating and may limit many normal activities in daily life, such as driving. Thus, sufficient investigation into the risks and benefits of the method is important in determining a therapeutic policy. For the use of bypass to be feasible, the rate of complications and the incidence of visual field loss must be lower than that in interventions without bypass. Use of bypass must be limited to patients with good neurological status. Usually, anticoagulation therapy is necessary during endovascular procedures, to prevent embolic complications. Under anticoagulation therapy, the risk of perioperative

bleeding (epidural hematoma, subdural hematoma, and bleeding from the anastomosis) associated with the open bypass procedure may be high. For these reasons, open bypass to prevent ischemic complications caused by PAO does not provide any benefit. In the future, stent coiling to preserve the parent artery will play an important role in this anatomical region, including the treatment of giant aneurysms in the P1 segment. However, the results of long-term follow-up after this procedure remain unclear.

### **Conclusions**

Intra-aneurysmal embolization is a good treatment for saccular aneurysms with well-defined necks. PAO should be performed in cases of SAH where SOA cannot be performed safely. Although the PCA is rich in collateral circulation, cerebral infarctions were noted in most patients after PAO, and it was difficult to predict the occurrence of these infarctions. However, the area of cerebral infarction tended to be small, and the neurological deficits observed were not critical. These findings suggest that PAO is a rather safe procedure for aneurysms of the P2 segment, and that SOA cannot be safely performed at a site distal to the P2 segment. Bypass techniques are used to prevent post-PAO ischemic complications. The rate of complications and the incidence of visual field loss must be lower than those in interventions without bypass. The use of bypass must be limited to patients with good neurological status. To determine the therapeutic policy for the use of bypass, it is necessary to obtain sufficient data about the risks and benefits of the method.

### **Conflict of interest**

The authors declare that they have no conflict of interest

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## **Figure Legends**

### **Fig. 1**

A magnetic resonance (MR) image showed a subarachnoid hemorrhage. Cerebral angiography revealed a large aneurysm of the left P2 segment. The patient underwent parent artery occlusion of the P2 segment directly proximal to the aneurysm; the technique was performed using a detachable coil. Radiographically, the aneurysm was occluded, and leptomeningeal collateral circulation was observed. Postoperative MR image showed cerebral infarction in the thalamus and occipital lobe

### **Fig. 2**

Postoperative computed tomography image showed a large cerebral infarction in the occipital lobe. Cerebral angiography revealed a small aneurysm of the right P3 segment. Parent artery occlusion using a detachable coil was performed immediately proximal to the aneurysm. Radiographically, the aneurysm was occluded, and leptomeningeal collateral circulation was observed

### **Fig. 3**

Angiogram showing a large saccular aneurysm in the P1–2 segment. Selective occlusion of aneurysm Complete occlusion was noted after SOA using a detachable coil. Three months after the treatment, oculomotor palsy had disappeared.

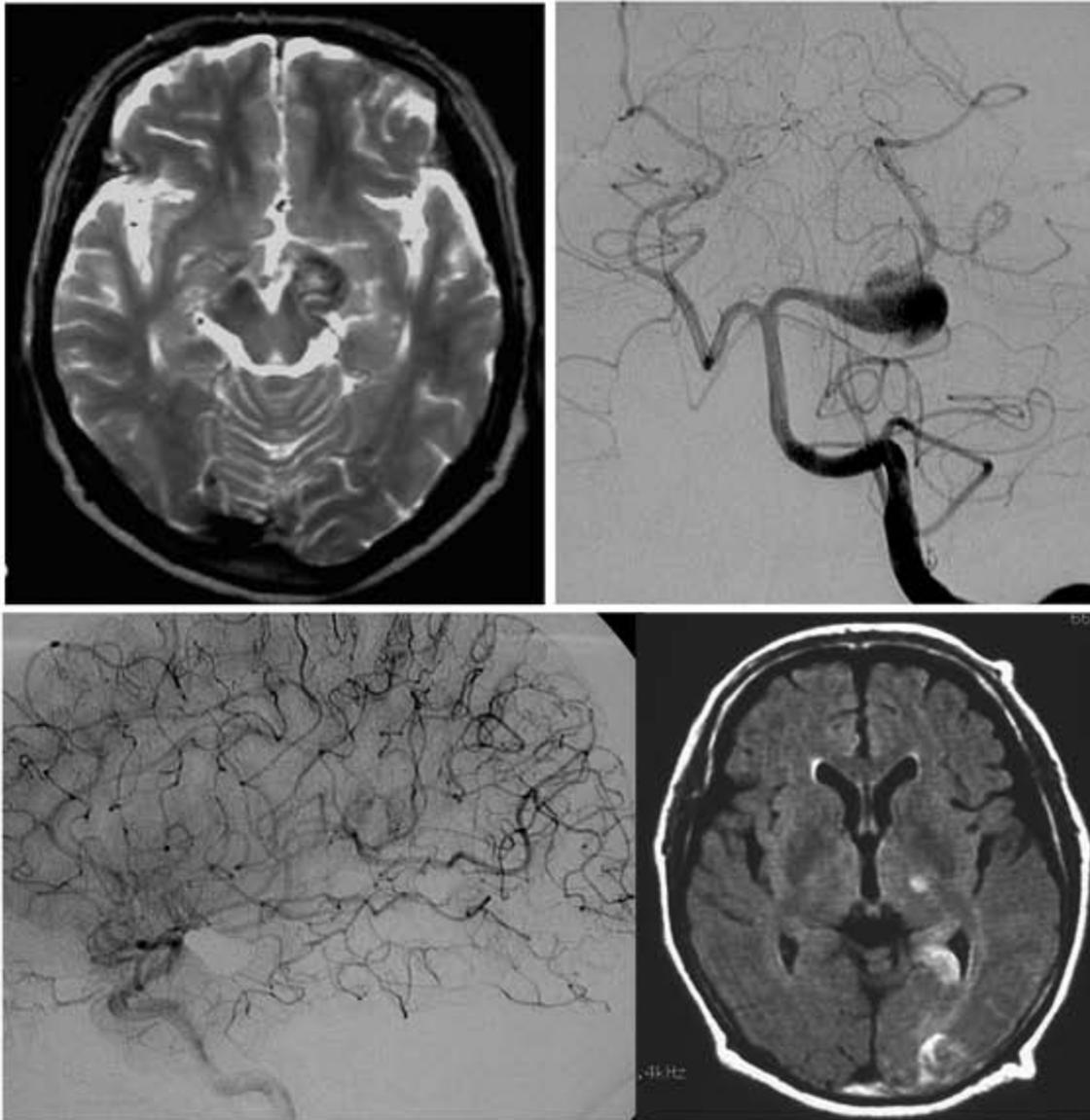


Fig 1

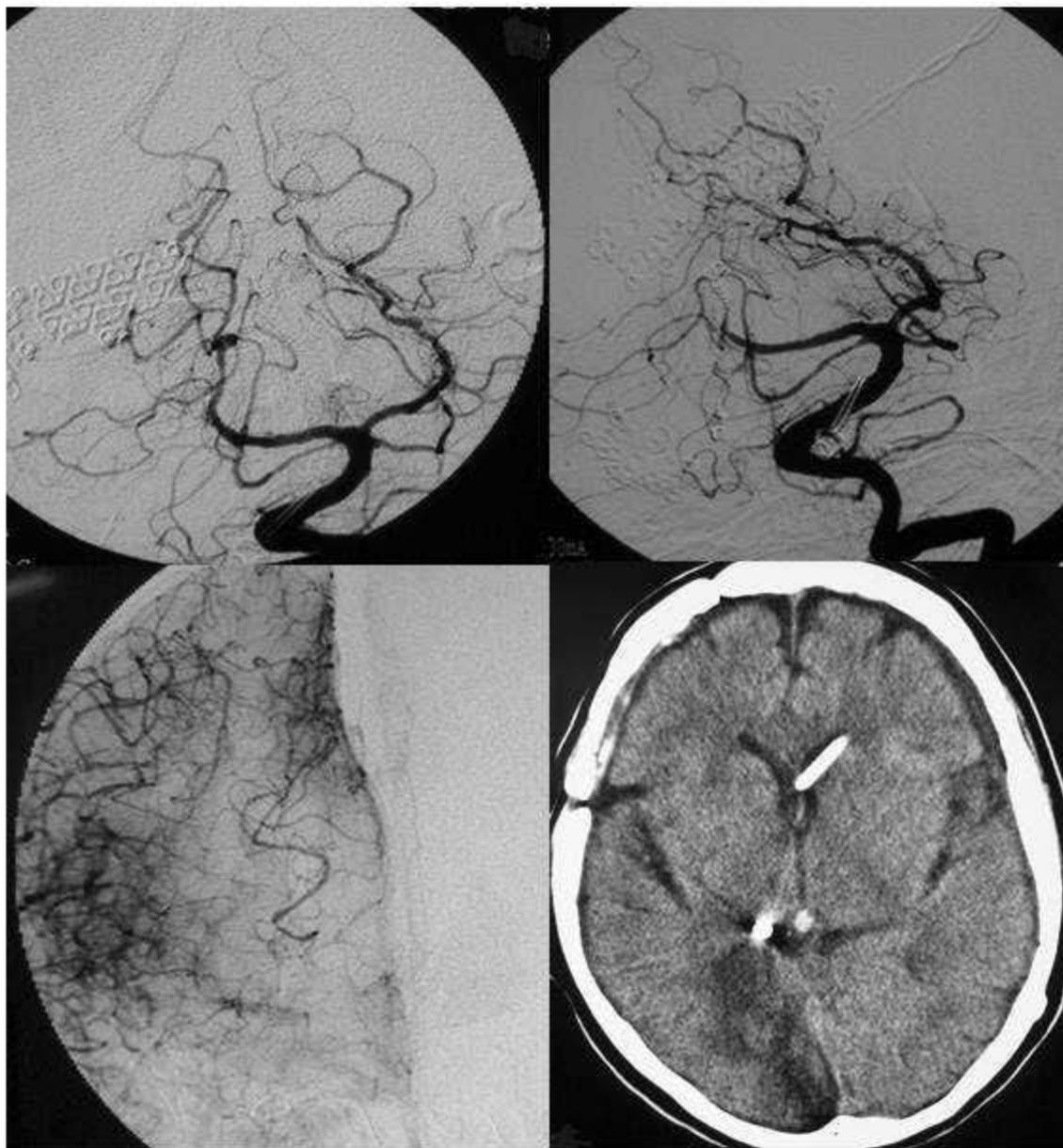


Fig 2

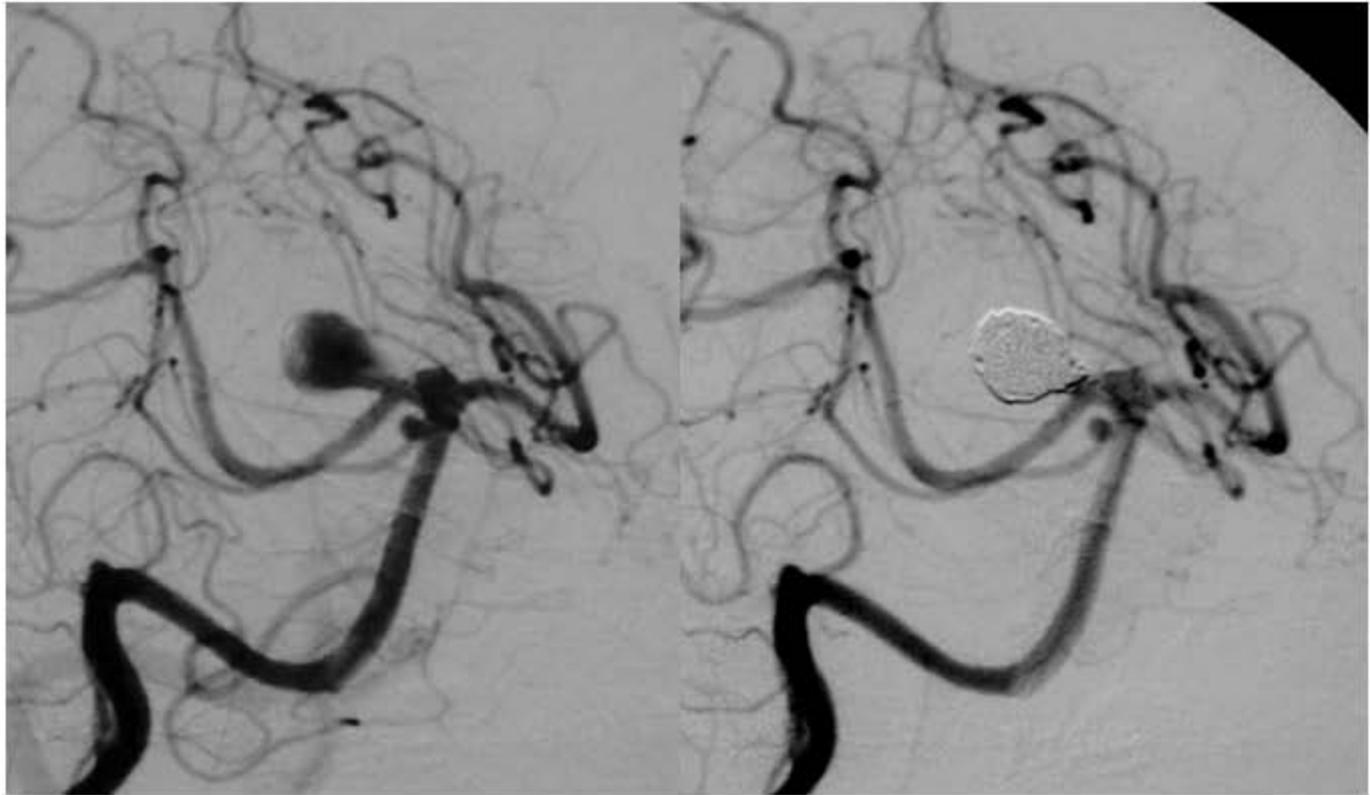


Fig 3

No/Age/Sex	clinical presentation	Site	Aneurysm type	Size	Intervention	Outcome	infarction	Associated disease
1/64/M	incidental	P4	fusiform	large	PAO	no deficit		
2/56/F	incidental	P1-2	saccular	small	IAE	no deficit		
3/58/F	SAH HK IV	P4	saccular	small	PAO	GOS4 hemianopsia	occipital (small)	
4/57/F	incidental	P1-2	saccular	small	IAE	no deficit		
5/74/F	CN III palsy	P1-2	saccular	large	IAE	no deficit		
6/66/F	SAH HK I	P2	thrombosed	large	PAO	GOS4 hemianopsia+numbness	thalamus+occipital (small)	
7/69/F	SAH HK IV	P3	berry	small	PAO	GOS3	occipital (large)	
8/65/F	SAH HK IV	P1	saccular	large	IAE	GOS2		
9/57/M	SAH HK II	P2	saccular	large	PAO	GOS5	occipital (small)	
10/58/F	SAH HK II	P1	saccular	small	IAE	GOS5		
11/54/F	SAH HK I	P3	thrombosed	large	PAO	GOS5	occipital (small)	
12/68/F	SAH HK IV	P1	saccular	small	IAE	GOS5		
13/64/F	SAH HK III	P2	saccular	small	IAE	GOS4		MoyaMoya
14/66/F	incidental	P2	saccular	small	IAE	no deficit		
15/77/F	SAH HK II	P2-3	saccular	small	IAE	GOS5		
16/33/M	incidental	P1-2	saccular	small	IAE	no deficit		BAVM
17/61M	SAH HK I	P2	dissection	small	PAO	GOS4	occipital (small)	
18/49/M	SAH HK I	P2-3	saccular	small	IAE	GOS5 , retreatment		
19/78/F	SAH HK III	P2-3	saccular	small	IAE	GOS4		
20/62/M	SAH+ICH HK III	P4	saccular	small	PAO	GOS3		dAVF
21/43/M	SAH HK IV	P2-3	thrombosed	giant	PAO	GOS1	occipital (small)	
Table1 Summary of 21 patients								

Author	Age/Sex	clinical presentation	Site	Aneurysm type	Size	Intervention	Outcome	Associated disease
Roouj et.al	43/F	incidental	P2	Fusiform	small	PAO with aneurysm	GOS5	
Roouj et.al	64/M	SAH	P2	Fusiform,Dissection	large	PAO with aneurysm	GOS5	
Roouj et.al	32/M	SAH	P2-3	Dissection	large	PAO with aneurysm	GOS1	
Roouj et.al	27/M	SAH	P4	Mycotic	small	PAO with aneurysm	GOS1	AIDS,endocarditis
Roouj et.al	64/F	SAH	P4	Saccular	small	PAO with aneurysm	GOS4	
Xianli et al	37/F	SAH	P2	Dissection	small	PAO with aneurysm	GOS5	
Xianli et al	49M	headache+hemiparesis	P2	Dissection	large	PAO with aneurysm	GOS4	
Xianli et al	43M	headache+heminumbness	P2	Dissection	large	PAO with aneurysm	GOS5	
Xianli et al	48M	SAH+CNIII	P2	Dissection	large	PAO with aneurysm	GOS5	
Xianli et al	58F	ICH	P2	Dissection	small	PAO with aneurysm	GOS4	
Xianli et al	31M	SAH	P2	Dissection	large	PAO with aneurysm	GOS5	
Xianli et al	4/M	headache	P2	Dissection	large	PAO with aneurysm	GOS5	
Xianli et al	5/M	SAH	P2	Dissection	large	PAO with aneurysm	GOS5	
Ciceri et al	21/F	?	P1	serpentine	giant	PAO with aneurysm	GOS5	
Ciceri et al	12/M	SAH	P2	serpentine	giant	PAO with aneurysm	GOS5	
Ciceri et al	52/F	SAH	P2-3	Berry	small	PAO with aneurysm	GOS5	hemianopsia
Ciceri et al	58/M	memory loss	P2-3	serpentine	giant	PAO with aneurysm	GOS5	hemianopsia+hemiparesis
Ciceri et al	52/F	SAH	P2-3	Berry	small	PAO with aneurysm	GOS5	
Ciceri et al	56/F	?	P3	serpentine	giant	PAO with aneurysm	GOS5	
Ciceri et al	57/M	?	P3	serpentine	giant	PAO with aneurysm	GOS5	
Hallacq et al	20/M	headache	P2	Fusiform	large	PAO with aneurysm	GOS5	
Hallacq et al	60M	headache	P2	Fusiform	large	PAO with aneurysm	GOS5	
Hallacq et al	49F	Gerstman syndrome	P2	Fusiform thrombosed	giant	PAO with aneurysm	GOS5	
Hallacq et al	49F	headache	P2	Saccular	large	PAO with aneurysm	GOS5	
Hallacq et al	47M	SAH	P2	Fusiform	giant	PAO with aneurysm	GOS5	
Hallacq et al	26M	headache	P2	serpentine thrombosed	giant	PAO with aneurysm	GOS5	
Arat et al	52/M	SAH	P2	Saccular	large	PAO with aneurysm	GOS4	hemianopsia+occipital infarction
Arat et al	17/M	headache+hemiparesis	P2	Fusiform	?	PAO with aneurysm	GOS5	
Arat et al	23/M	headache	P2	Saccular	giant	PAO with aneurysm	GOS5	
Arat et al	51/M	headache	P2-3	Fusiform	?	PAO with aneurysm	GOS5	posterior thalamus infarction temporary hemiparesis hyperaesthesia
Arat et al	48/M	SAH	P2	Saccular	large	PAO with aneurysm	GOS5	posterior thalamus infarction
Arat et al	26/F	headache	P2	Fusiform	?	PAO with aneurysm	GOS5	
Arat et al	23/M	headache	P3	Fusiform	?	PAO with aneurysm	GOS5	
Arat et al	61/M	SAH	P2	Saccular	large	PAO with aneurysm	GOS5	
Biondi et al	28/F	headache	P2-3	forcal dilatation,thrombosed	giant	PAO without aneurysm	GOS5	
Biondi et al	34/M	headache	P2-3	focal dilatation,thrombosed	giant	PAO without aneurysm	GOS5	
Biondi et al	25/F	ICH,hemiplegia	P2	focal dilatation	giant	PAO without aneurysm	GOS3	partial hemianopsia
Biondi et al	50M	headache,visual trouble	P2	focal dilatation	large	PAO without aneurysm	GOS4	
Table2 The summary of the patients treated by PAO using bare metal coils in literature review								