Posterior Cerebral Artery Aneurysms: Treatment and Outcome Analysis in 121 Patients

Felix Goehre[1,2], Behnam Rezai Jahromi[1], Martin Lehecka[1], Hanna Lehto[1], Riku Kivisaari[1], Hugo Andrade-Barazarte[1], Tarik F. Ibrahim[1], Richard Gonzalo Párraga[4], Christopher Ludtka[2], Hans Jörg Meisel[2], Timo Koivisto[3], Mikael von und zu Fraunberg[3], Mika Niemelä[1], Juha E. Jääskeläinen[3], Juha A. Hernesniemi[1]

■ OBJECTIVE: **Aneurysms of the posterior cerebral artery (PCA) are uncommon. Because of their low incidence, only 5 series with more than 30 patient cases have been reported. The treatment of PCA aneurysms is challenging because of the high frequency of fusiform aneurysms and closeness to important neuroanatomic structures.**

■ METHODS: **A total of 121 patients with 135 PCA aneurysms were reviewed. The clinical and radiologic data, treatment strategies, and 1-year outcomes were analyzed. Patients with giant aneurysms, associated aneurysms, and aneurysms on arteriovenous malformation-feeding PCAs were considered as complex cases. Outcomes were categorized into 3 groups: good (modified Rankin Scale [mRS], score 0–1), moderate (mRS score, 2–4), and poor (mRS score, 5–6).**

■ RESULTS: **There were 52 ruptured (39%) and 83 unruptured (61%) PCA aneurysms in 121 patients, with the following distribution: P1 ($n = 53$), P1/2 ($n = 39$), P2 ($n = 28$), and P3 ($n = 15$). The incidence of fusiform PCA aneurysms was high (24%). Microsurgical treatment was applied to 63 aneurysms and endovascular treatment to 19 aneurysms; 55 aneurysms were treated conservatively. The following treatment results were achieved: for patients with unruptured PCA aneurysms, $n = 19$; 12 good outcomes, 63%; 6 moderate, 31%; 1 poor, 1%; for patients with ruptured PCA aneurysms, $n = 27$; 10 good, 37%; 9 moderate, 33%; 8 poor, 30%; and for patients with complex neurovascular diseases and PCA aneurysms, $n = 96$; 42 good, 43%; 40 moderate, 42%; 14 poor, 15%.**

■ CONCLUSIONS: **Aneurysms of the PCA are infrequent and often associated with other vascular diseases. Microsurgery and endovascular treatment are effective for the occlusion of PCA aneurysms. The preservation or reconstruction of the parent vessel is crucial for favorable treatment outcomes.**

INTRODUCTION

Aneurysms of the posterior cerebral artery (PCA) are uncommon, representing less than 2% of all aneurysms.[1-3] Because of this low incidence, most of the institutional series on PCA aneurysms are small[2,4-17]; only 7 reports with more than 25 patients have previously been published (Table 1).[1,18-23] The largest series was reported by Drake et al.[7] in a study of 125 cases.

PCA aneurysms are classified into 5 groups according to their segment origin (Figure 1): the P1 segment (P1 aneurysms) or precommunicating segment; the P1/P2 junction (P1/P2 aneurysms); the P2 segment (P2 aneurysms); the P3 segment (P3 aneurysms); and the P4 segment (P4 aneurysms).[24]

In general, treatment of PCA aneurysms is challenging because of their deep anatomic location, closeness to sensitive

Key words
- Aneurysm
- Outcome
- Posterior Cerebral Artery
- Treatment

Abbreviations and Acronyms
AVM: Arteriovenous malformation
DSA: Digital subtraction angiography
H&H: Hunt and Hess grade
ICH: Intracerebral hemorrhage
IVH: Intraventricular hemorrhage
mRS: modified Rankin Scale
P1: P1 segment of posterior cerebral artery
P1/2: P1/P2 junction of posterior cerebral artery
P2: P2 segment of posterior cerebral artery
P3: P3 segment of posterior cerebral artery
P4: P4 segment of posterior cerebral artery
PCA: Posterior cerebral artery
SAH: Subarachnoid hemorrhage

From the [1]Department of Neurosurgery, Helsinki University Hospital, Helsinki, Finland; [2]Department of Neurosurgery, Stroke Center, Bergmannstrost Hospital, Halle, Germany; [3]Department of Neurosurgery, NeuroCenter, Kuopio University Hospital, Kuopio, Finland; [4]Department of Neurological Surgery, Hospital Univalle, Cochabamba-Cercado, Bolivia

To whom correspondence should be addressed: Felix Goehre, M.D. [E-mail: fgoehre@gmail.com]

Citation: World Neurosurg. (2016) 92:521-532. http://dx.doi.org/10.1016/j.wneu.2016.03.063

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

Table 1. Previous Reports on Posterior Cerebral Artery Aneurysms with More Than 25 Patients

Reference	Cases	Treatment
Drake et al. 1996[1]	125	Surgical
Zhitao et al. 2010[18]	42	Surgical
Chang et al. 2010[19]	33	Surgical and endovascular
Wang et al. 2015[20]	30	Surgical
Taylor et al. 2003[21]	30	Surgical and endovascular
Sanai et al. 2008[22]	27	Surgical and endovascular
Kim et al. 2013[23]	25	Surgical and endovascular

neuroanatomic structures, and the long intravasal route to the terminal segments of the posterior circulation.[25,26] Furthermore, the specific morphologic features of PCA aneurysms complicate their treatment, especially the high frequency of fusiform shape and closeness to important midbrain perforators.[27]

Surgical approaches, such as the subtemporal, pterional, orbitozygomatic, supracerebellar transtentorial, and posterior interhemispheric approaches, have been described for microsurgical management of aneurysms on various PCA segments.[1,6,17,21,28,29] However, during the last 3 decades, endovascular techniques have evolved rapidly and PCA aneurysms close to the brainstem are also treated using endovascular techniques.[4,7,16,30]

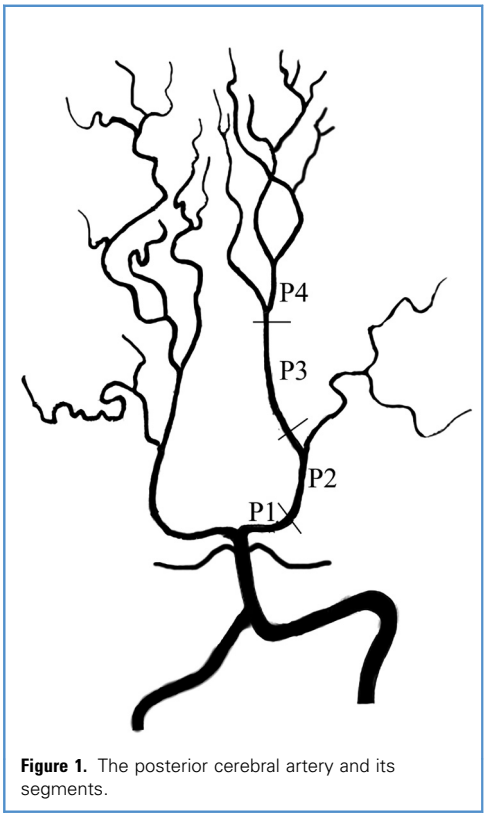

Figure 1. The posterior cerebral artery and its segments.

We present a consecutive series of 121 patients with 135 PCA aneurysms treated between 1980 and 2014 at the 2 given neurosurgical centers, which are responsible for a population of approximately 2.8 million people. The aim of this analysis is to establish a foundation for the further development of treatment strategies for PCA aneurysms.

METHODS

From 1980 to 2014, 121 patients with 135 PCA aneurysms were evaluated and treated at 2 neurosurgical centers (Helsinki and Kuopio), responsible for the entire population of southern and eastern Finland of 2.8 million people. Overall, slightly more than 14,500 patients with intracranial aneurysms were treated.

The hospital records and images were evaluated for all patients with PCA aneurysms. The following clinical data were collected: patient age, sex, Hunt and Hess (H&H) grade on admission, treatment modality, time to treatment, occlusion grade, complications, neurologic deficits, and cause of death. The modified Rankin Scale (mRS) was used to measure outcomes, evaluated at 12-month follow-up. For further analysis, outcomes were categorized into 3 groups: 1) good (mRS score, 0–1), 2) moderate (mRS score, 2–4), and 3) poor (mRS score, 5–6).

We performed a PubMed search for articles published from 1980 to 2015 on the treatment of PCA aneurysms with the following keyword(s): PCA, posterior cerebral artery, aneurysms. We identified 7 series with over 25 patients, presented in Table 1.

All patients underwent computed tomography angiography, digital subtraction angiography (DSA), or magnetic resonance angiography for diagnosis. The radiologic images were stored in the hospital's digital archiving system (PACS; AGFA, IMPAX, version 4.5, launched in 1998) or in the central radiographic image archive, from which the relevant images were recalled. The image analyses were conducted by 2 experienced neurosurgeons, 1 (R.K.) with double specialization in neurosurgery and neuroradiology. The following radiologic data were collected: aneurysm height, neck diameter, dome width, previous treatment (coiling or clipping), and the presence of remnant aneurysm parts after the treatment.

Using these methods, 121 consecutive patients (36 male, 85 female) with 135 PCA aneurysms were identified. Ruptured PCA aneurysms were found in 52 patients. For further analyses, the patients were divided into 9 groups: 1) unruptured saccular aneurysms ($n = 9$); 2) unruptured fusiform aneurysms ($n = 10$); 3) unruptured giant aneurysms ($n = 7$); 4) unruptured PCA aneurysms with associated aneurysms ($n = 21$); 5) ruptured saccular aneurysms ($n = 20$); 6) ruptured fusiform aneurysms ($n = 7$); 7) ruptured PCA aneurysms with associated aneurysms ($n = 18$); 8) subarachnoid hemorrhages (SAHs) from an associated aneurysm rupture ($n = 17$); and 9) SAHs from an associated arteriovenous malformation (AVM) ($n = 12$).

Microsurgical treatment was applied to 63 aneurysms and endovascular treatment to 19 aneurysms; 55 aneurysms were treated conservatively.

Ethics and Statistical Analyses

The data were collected under the approval of local university ethic committees (469/Eo/04 HUCH - KUH). Data were analyzed with a commercially available statistical software package (SPSS for Mac, version 22.0, 2012 [SPSS Inc., Chicago, Illinois, USA]).

RESULTS

Distribution of PCA Aneurysms ($n = 135$)

In this series, the most frequently affected segment for PCA aneurysms was the P1 segment ($n = 53$; 39%), followed by the P1/2 junction ($n = 39$; 29%), the P2 segment ($n = 28$; 21%), and the P3 segment ($n = 15$; 11%). P3 aneurysms presented the highest rupture rate, with 9 ruptured of the 15 total aneurysms (60%). In addition, fusiform aneurysms were often found on the P2 segment ($n = 10$). However, the rupture rate was highest in fusiform P3 segment aneurysms, with 3 of 4 being ruptured.

Shapes and Sizes of PCA Aneurysms ($n = 135$)

A saccular shape was present in 102 (76%) of the 135 PCA aneurysms; the remaining 33 (24%) aneurysms were fusiform. Most of the evaluated saccular PCA aneurysms were smaller than 7 mm ($n = 77$, 75%), even when ruptured ($n = 22$, 56%). In this series, we saw only 9 large and 7 giant aneurysms, altogether representing 12% of the studied PCA aneurysms. The characteristics of PCA aneurysms are given in Table 2.

Clinical Presentation

Fifty-two patients (43%) had SAH from a PCA aneurysm (Figure 2); 21 of them also presented with an intraventricular hemorrhage (IVH), 3 with an intracerebral hemorrhage (ICH), and 1 with an SDH. Seventeen patients (14%) were diagnosed in conjunction with a ruptured associated aneurysm. Twelve patients (10%) had an AVM-related PCA aneurysm. Oculomotor nerve affection occurred in 7 patients (6%); 3 of these patients had an unruptured PCA aneurysm, 1 patient a ruptured PCA aneurysm, and 2 patients a ruptured posterior communicating artery aneurysm. Six patients with unruptured aneurysms (5%) manifested symptoms of ischemia after embolism and 2 patients with unruptured aneurysms became symptomatic from mass effect.

Multiple PCA Aneurysms

Multiple PCA aneurysms were found in 10 patients. Nine of these patients presented the second PCA aneurysm on the ipsilateral PCA and 1 patient on the contralateral, whereas 4 of these 9 had a ruptured PCA aneurysm. Four patients had 3 PCA aneurysms each, and for 2 patients, these aneurysms were located on an AVM-feeding PCA.

Associated Aneurysms

Associated aneurysms were found in 67 patients (55%), of whom 19 presented SAH (16%) (Table 3). Two ruptured associated aneurysms were located on an AVM feeder. Forty-one patients (34%) had more than 1 associated aneurysm. The distribution of associated aneurysms along the intracranial vessels is given in Table 3.

Table 2. Characteristics of 135 Posterior Cerebral Artery Aneurysms

Variables	Ruptured Saccular PCA Aneurysm (n = 39), n (%)	Ruptured Fusiform PCA Aneurysm (n = 13), n (%)	Unruptured Saccular PCA Aneurysm (n = 63), n (%)	Unruptured Fusiform PCA Aneurysm (n = 20), n (%)
Location				
P1As	13 (33)	3 (23)	30 (47)	7 (35)
P1/2As	15 (38)	4	15 (24)	5 (25)
P2As	5 (13)	3 (23)	13 (21)	7 (35)
P3As	6 (15)	3 (23)	5 (8)	1 (1)
Side				
Left	23 (59)	9 (69)	32 (51)	4 (20)
Right	16 (41)	4 (31)	31 (49)	16 (80)
Gender				
Male	10 (26)	6 (46)	18 (29)	5 (25)
Female	29 (74)	7 (54)	45 (71)	15 (75)
Size (saccular, diameter)				
Small (<7 mm)	22 (56)		55 (87)	
Medium (7–14 mm)	15 (38)		4 (6)	
Large (15–24 mm)	3 (8)		2 (3)	
Giant (>24 mm)	1 (3)		2 (3)	
Size (fusiform, length)				
Small (<7 mm)		2 (15)		6 (30)
Medium (7–14 mm)		7 (54)		10 (50)
Large (15–24 mm)		2 (15)		2 (10)
Giant (>24 mm)		2 (15)		2 (10)
Wall structure				
Irregular	27 (69)	10 (77)	20 (32)	7 (35)

As, aneurysms; P1,P2,P1/2 junction, P2, P3, segments of the posterior cerebral artery; PCA, posterior cerebral artery.

Patients with Unruptured Saccular PCA Aneurysms ($n = 9$)

Six patients were treated conservatively, including 1 patient with oculomotor palsy. That particular decision was made together with the patient, primarily based on the patient's older age (88 years, mRS score 1 after 1 year and persistent oculomotor palsy). One patient died after a cardiac infarction during the first year of

follow-up. The other patients had aneurysms smaller than 4 mm and had favorable outcomes (mRS score, 0). Three patients were treated microsurgically, 1 of whom had a moderate outcome caused by parent vessel occlusion and consecutive PCA infarction. All other patients, including 1 who underwent bypass bridging, had a good outcome (Table 4).

Patients with Unruptured Fusiform PCA Aneurysms ($n = 10$)

Nine of 10 patients were diagnosed incidentally and the remaining patient became symptomatic with embolic symptoms. One patient with a small aneurysm (<3 mm) was treated conservatively and had a good outcome. The remaining 9 patients were treated by microsurgical means. In 3 patients, a proximal occlusion was performed. One aneurysm was clipped after initially failed coiling and 4 aneurysms were directly clipped. One aneurysm was bridged with a bypass and trapped. One clipped aneurysm required reoperation to trap the aneurysm. Two patients developed a PCA infarction after proximal occlusion. Three patients developed permanent oculomotor nerve palsy after direct clipping. The overall outcome was good in 5 cases and moderate in 5 cases (Table 4).

Patients with Unruptured Giant PCA Aneurysm ($n = 7$)

Only 3 of the 7 patients with a giant PCA aneurysm (diameter ≥25 mm) became symptomatic via rupture (Table 2). Two patients became symptomatic by mass lesion, 1 by thromboembolism, and 1 patient's aneurysm was found incidentally via a computed tomography scan taken for other reasons. Six of these aneurysms were located distal from the P1/2 junction and 4 giant aneurysms presented a fusiform shape. The following treatment methods were applied: proximal occlusion via subtemporal approach ($n = 3$), coil embolization with parent vessel occlusion ($n = 1$), thrombectomy and PCA reconstruction ($n = 1$), complex excimer laser-assisted nonocclusive anastomosis bypass ($n = 1$), and conservative treatment after iatrogenic injury during DSA ($n = 1$). An additional ventriculoperitoneal shunt was required in 1 patient. The achieved outcomes were: 1 good, 5 moderate, and 1 fatal (Table 4).

Patients with Unruptured PCA Aneurysms with Associated Unruptured Aneurysms ($n = 21$)

Twenty-one patients were affected by multiple aneurysms associated with PCA aneurysms. The overall outcome was good in 13 patients and moderate in 7 cases. One patient died after a diagnostic DSA procedure (Table 4).

Ten patients were treated by microsurgery. In 3 of these patients, the PCA aneurysm was occluded in the same procedure with associated aneurysms, from a pterional or lateral supraorbital approach. Two-step surgeries were necessary in 5 patients. In 2 of these cases, a distal PCA aneurysm (P3 segment) was occluded from an additional posterior interhemispheric approach. In 1 patient, the PCA aneurysm was treated by flow modulation during a complex bypass procedure for the treatment of multiple posterior circulation aneurysms. Three patients were treated by endovascular techniques and all aneurysms were occluded during 1 interventional session. In 1 patient, 3 PCA aneurysms were treated by endovascular occlusion of the proximal aneurysm and the parent vessel. In 8 patients, the PCA aneurysm was treated conservatively because of small aneurysm size. In 4 patients, the aneurysm was smaller than 4 mm. Three patients presented poor clinical conditions in general.

Patients with Unruptured PCA Aneurysms with Associated Ruptured Aneurysms ($n = 17$)

Associated aneurysms were ruptured in 17 patients, exhibiting the following H&H grade distribution: I ($n = 3$), II ($n = 7$), III ($n = 1$),

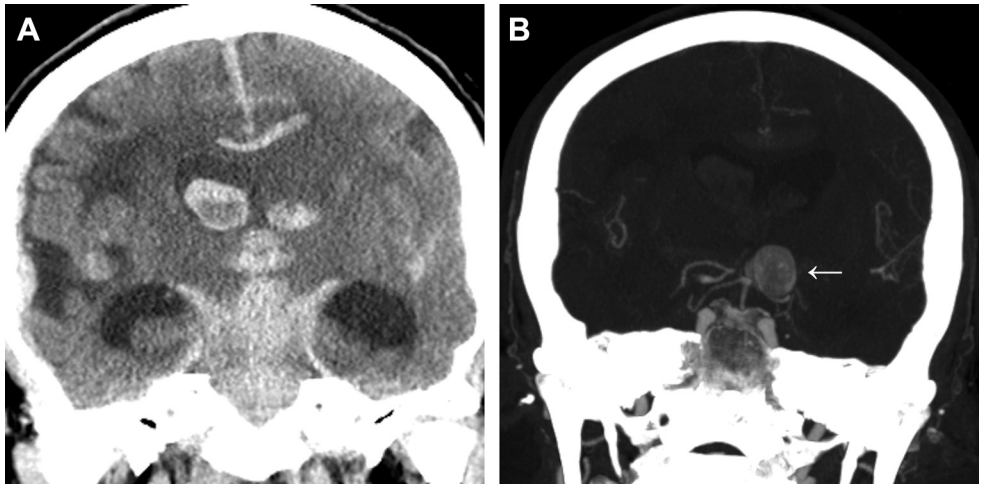

Figure 2. Coronal computed tomography (**A**, coronal) shows severe subarachnoid hemorrhage accompanied by intraventricular hemorrhage. The computed tomography angiography (**B**, coronal) shows that the bleeding was caused by the rupture of a large left-sided posterior cerebral artery P1/2 junction aneurysm (*arrow*). The outcome in the presented case was fatal.

Table 3. Distribution of 156 Associated Aneurysms

Vessel	Unruptured Associated Aneurysm (n = 137), n (%)	Ruptured Associated Aneurysm (n = 19), n (%)
Internal carotid artery	43 (28)	4 (3)
Anterior cerebral artery	12 (8)	1 (1)
Anterior communicating artery	12 (8)	2 (1)
Middle cerebral artery	33 (21)	4 (3)
Posterior cerebral artery	13 (8)	1 (1)
Basilar artery	16 (10)	4 (3)
Superior cerebellar artery	2 (1)	
Vertebral artery	6 (4)	3 (2)

IV (n = 2), and V (n = 4). The following intracranial vessels were affected by ruptured aneurysms: middle cerebral artery (n = 5), basilar artery (n = 4), internal carotid artery (n = 3), anterior communicating artery (n = 3), and vertebral artery (n = 2). In 3 patients, the PCA aneurysms were treated during the same procedure as the ruptured associated aneurysm, representing 3 endovascular approaches. A multistage procedure was applied in 4 cases using a microsurgical approach for the PCA aneurysm. A conservative treatment was chosen for 10 patients with an unruptured PCA aneurysm after the treatment of the ruptured aneurysm. One patient was in such poor condition that the cerebral aneurysms could not be treated. Treatment of a secondary hydrocephalus by an external ventricular drain was necessary in 5 cases (29%), but implantation of a permanent shunt was not needed. A favorable 1-year outcome was achieved for 7 patients, a moderate outcome for 3 patients, and a fatal outcome in 6 cases (Table 4).

Patients with Ruptured Saccular PCA Aneurysms (*n* = 20)

All patients became symptomatic by SAH and 1 suffered from an additional oculomotor palsy. Four patients were treated conservatively: 3 because of poor clinical condition on admission (H&H V) and 1 because of old age and severe cardiac dysfunction. Three patients were treated by endovascular techniques, 2 by direct coil application, and 1 with a stent/coil technique. Two of the interventions were uncomplicated and the patients recovered well with a good outcome; 1 patient developed an anterior circulation infarction during the initial treatment and recovered with an mRS score of 4 after 1 year. Thirteen patients were microsurgically treated. In all of these patients, a direct clip occlusion was possible. In 5 patients, preservation of the parent vessel was not possible. In 1 patient, the aneurysm ruptured during clipping from a pterional approach; the patient was initially vegetative and later died. One aneurysm was incompletely clip occluded and the patient died after rerupture; diagnosis was confirmed in autopsy. Six patients had a good 1-year outcome and 4 others a moderate outcome (Table 4).

Patients with Ruptured Fusiform PCA Aneurysms (*n* = 7)

All patients became symptomatic with SAH. Three were conservatively treated because of their initially poor clinical condition. Only 1 of these patients survived. Two of the 7 were treated successfully by endovascular techniques. One patient was treated by direct clip occlusion, but the aneurysm ruptured intraoperatively and the patient developed a PCA infarction after extended temporary clipping. One patient had a favorable 1-year outcome after proximal occlusion (Table 4).

Patients with Ruptured PCA Aneurysms with Associated Unruptured Aneurysms (*n* = 18)

We treated 18 patients with ruptured PCA aneurysms and associated aneurysms. Three patients were treated conservatively because of their poor clinical condition. The outcome was fatal in all of these cases. Three initially presented with H&H grade V. One suffered from a cardiac infarction after SAH. Ten patients were treated microsurgically. In 6 patients, the ruptured PCA was addressed directly and all reachable aneurysms were occluded from the same approach. In 4 patients, additional surgeries were necessary. The outcome was good in 3 cases, moderate in 6 cases, and fatal in 1 case. In 5 patients, the PCA aneurysms were treated endovascularly. Associated anterior circulation aneurysms were treated in 4 cases by clip occlusion. Two patients attained a good outcome and 3 a moderate outcome (Table 4).

Patients with an Associated AVM (*n* = 12)

There were 12 patients with associated AVMs, 8 of whom had aneurysms (n = 16) located on an AVM-feeding PCA. Treatment by direct microsurgical occlusion was considered necessary in 2 cases: 1 case for a ruptured saccular P2 aneurysm and the other for a P3 aneurysm together with a flow-related basilar bifurcation aneurysm. In both cases, these aneurysms were clip occluded from a subtemporal approach. In 6 patients, the AVM was occluded and the aneurysm was considered to be flow related, therefore warranting indirect treatment. A conservative treatment was chosen for 4 patients. One patient was in poor condition after AVM rupture, and because the remaining aneurysms were small, the patient was addressed at follow-up. Only 3 of the patients in this group had a good 1-year outcome, whereas 8 patients had a moderate outcome, and 1 patient had a poor outcome. The outcome deficit was primarily related to the AVM and its treatment; 4 AVMs were ruptured and 1 flow-related associated anterior communicating artery aneurysm caused an SAH. Only 1 flow-related PCA aneurysm was ruptured (Table 4).

Microsurgical Treatment of 63 PCA Aneurysms (2 Patients After Failed Endovascular Treatment)

Sixty-three aneurysms were treated by microsurgical means, 2 of which had previously been incompletely occluded by coiling. The aneurysms were located on the P1 segment (n = 18), P1/2 junction (n = 22), P2 segment (n = 15), and P3 segment (n = 8). Nineteen aneurysms were fusiform. Thirty-three aneurysms were treated from a subtemporal approach, 12 from a pterional approach, 10

Table 4. Treatment of 121 Patients with 135 Posterior Cerebral Artery Aneurysms Treated Between 1980 and 2012

	Number of Patients with Unruptured Saccular PCA Aneurysms (n = 9)	Number of Patients with Unruptured Fusiform PCA Aneurysms (n = 10)	Number of Patients with Unruptured Giant PCA Aneurysms (n = 7)	Number of Patients with Unruptured PCA Aneurysms with Associated Unruptured Aneurysms (n = 21)	Number of Patients with Unruptured PCA Aneurysm and Ruptured Associated Aneurysm (n = 17)	Number of Patients with Ruptured Saccular PCA Aneurysms (n = 20)	Number of Patients with Ruptured Fusiform PCA Aneurysms (n = 7)	Number of Patients with Ruptured PCA Aneurysms with Associated Unruptured Aneurysms (n = 18)	Number of Patients with an Associated AVM (n = 12)
Treatment									
Microsurgical treatment	3	9	5	10	4	13	2	10	2
Endovascular treatment	0	0	1	3	3	3	2	5	0
Conservative	6	1	1	8	10	4	3	3	10
One-year outcome									
mRS 0–1 (good)	7	5	1	13	7	8	2	5	3
mRS 2–4 (moderate)	1	5	5	7	4	6	3	9	8
mRS 5–6 (poor)	1	0	1	1	6	6	2	4	1

AVM, arteriovenous malformation; mRS, modified Ranking Scale; PCA, posterior cerebral artery.

from a lateral supraorbital approach, and 3 from a posterior interhemispheric approach (Figure 3). Modified temporal approaches were used for 2 bypass procedures. The following procedures were applied: neck clipping ($n = 45$), failed clipping ($n = 1$), neck clipping after failed endovascular occlusion ($n = 2$), aneurysmorrhaphy ($n = 1$), trapping ($n = 2$, 1 after failed clipping), proximal occlusion ($n = 7$), parent vessel occlusion after bypass ($n = 4$, 2 excimer laser-assisted nonocclusive anastomosis procedures), and wrapping ($n = 1$). During 23 of these procedures, associated aneurysms were also treated via the same approach. Six patients had an incomplete aneurysm occlusion after neck clipping, 1 of whom was treated by aneurysm trapping. Nine patients developed oculomotor nerve palsy and 1 patient became affected by trochlear nerve palsy after microsurgical treatment.

Endovascular Treatment of 19 PCA Aneurysms

The first endovascular treatment of a PCA aneurysm in our institutions was in 1998. The aneurysms were located on the P1 segment ($n = 8$), P1/2 junction ($n = 4$), P2 segment ($n = 3$), and the P3 segment ($n = 4$). Four of these aneurysms were fusiform. The following techniques were applied: coiling ($n = 15$), stent-assisted coiling ($n = 1$), and sole stenting ($n = 2$). In 1 patient, the PCA was occluded for treatment of a total of 3 PCA aneurysms.

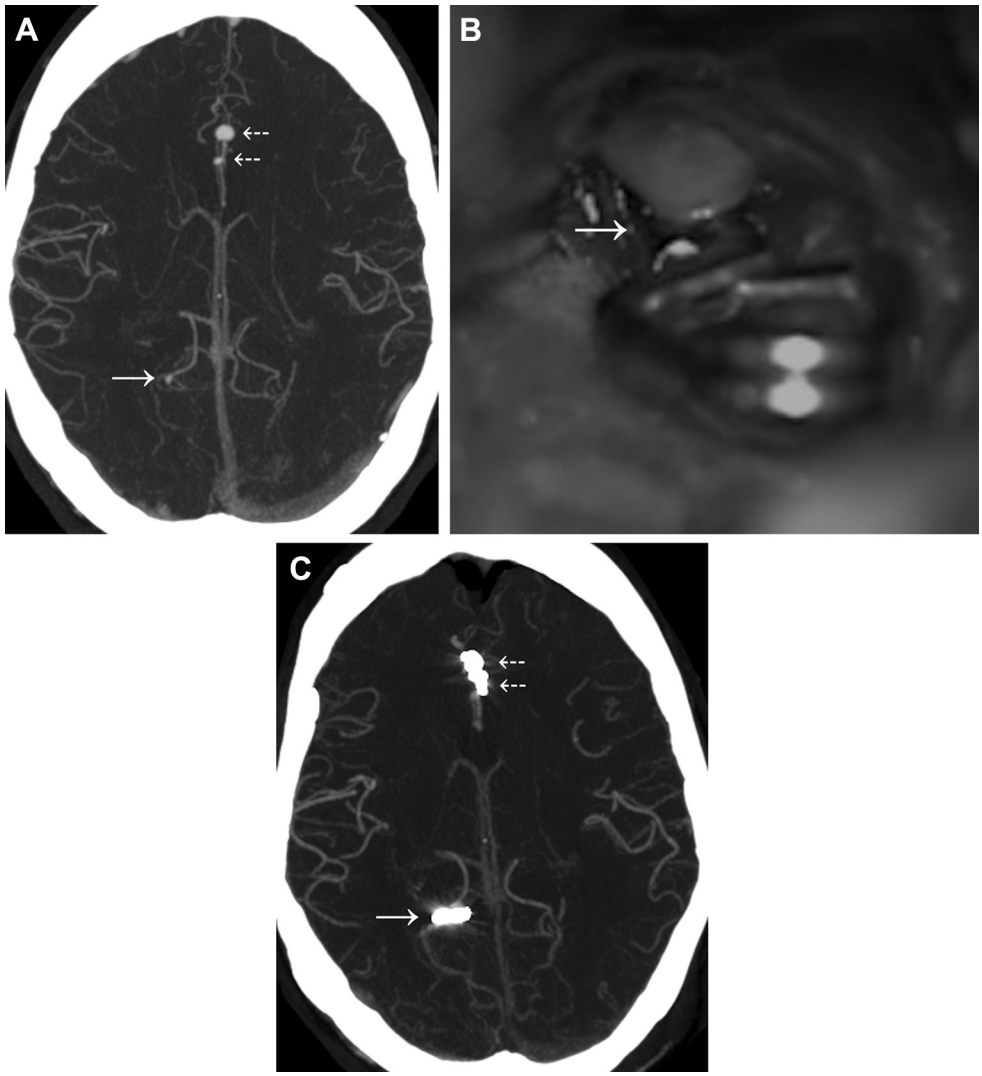

Figure 3. Preoperative computed tomography angiography (**A**, axial) shows a nonruptured right-sided posterior cerebral artery P3 segment aneurysm (*rightward arrow*). The aneurysm was exposed from a right posterior interhemispheric approach and occluded with a Yasargil miniclip (*arrow*), preserving the parent vessel under direct visual control under high magnification (**B**). The postoperative computed tomography angiography (**C**, axial) shows patency of posterior cerebral artery branches distal from the clip occluded aneurysm (*rightward arrow*). Associated anterior cerebral artery aneurysms are apparent in the angiographic images (*leftward interrupted arrows*).

In another patient, the PCA was occluded intentionally; this patient developed a PCA infarction that was subsequently asymptomatic (Figure 4). An incomplete aneurysm occlusion was observed in 5 patients, 2 of whom were subsequently surgically treated and 3 of whom were followed up. Two patients developed permanent oculomotor nerve palsy secondary to the aneurysm treatment.

Conservative or Indirect Treatment of 55 PCA aneurysms

Consideration for conservative treatment was based on the patient's condition (ie, H&H grade V, $n = 10$), aneurysm size (<4 mm, $n = 29$), and the presence of AVMs. Associated aneurysms were treated by microsurgical occlusion in 2 cases; all others underwent follow-up ($n = 14$). One fatality occurred shortly after diagnostic DSA of a patient with a large fusiform P2 segment aneurysm, precluding any active treatment. One patient received conservative treatment because of advanced age.

Complications and Poor Outcome Analyses

Twenty-one patients with PCA aneurysms (52% ruptured vs. 48% unruptured, but 33% SAH form associated aneurysm rupture) died during the first year and 2 remained vegetative. There were 13 patients with H&H grade V and poor clinical conditions on arrival; they were primarily treated conservatively for palliative reasons. Five patients had an SAH (H&H III–IV): 1 patient died by cardiac infarction, 1 bled in an infarct area after proximal PCA occlusion,

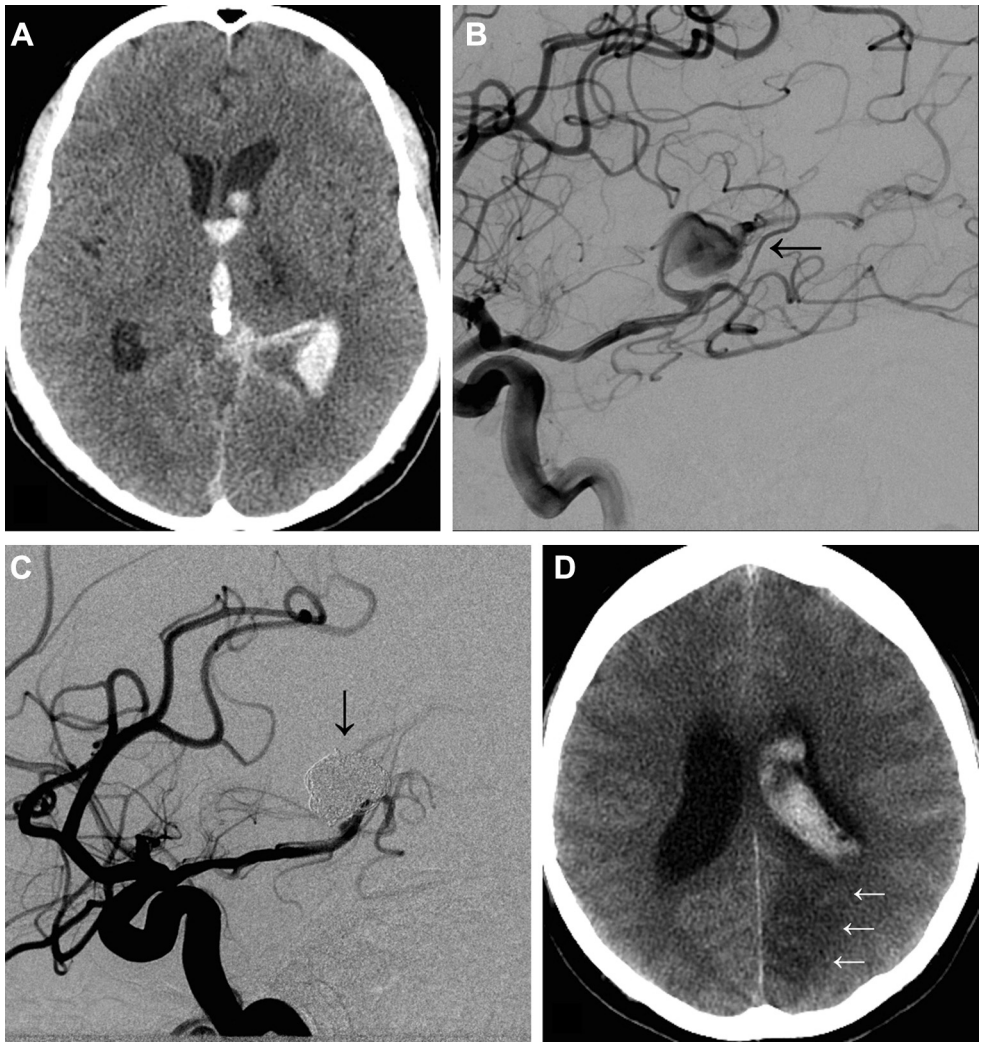

Figure 4. Computed tomography (**A**, axial) show a subarachnoid hemorrhage with intraventricular bleeding. The lateral view of digital subtraction angiography (**B**, left internal carotid injection) shows a fetal-type posterior cerebral artery carrying a posterior cerebral artery P3 segment aneurysm. The aneurysm was treated by coil occlusion of the aneurysm together with the parent artery (**C**). The postoperative computed tomography (**D**, axial) shows a right-side posterior cerebral artery infarction (*arrows*).

Table 5. PCA Infarction After Parent Vessel Occlusion

Patient	Treatment	Occluded Segment	Aneurysm Shape	Infarction
1	Microsurgical trapping	P2	Fusiform	Infarction
2	Microsurgical trapping	P2	Saccular	—
3	Microsurgical proximal occlusion	P3	Fusiform	—
4	Microsurgical proximal occlusion	P2	Fusiform	Infarction
5	Microsurgical proximal occlusion	P2	Fusiform	—
6	Microsurgical proximal occlusion	P3	Fusiform	Infarction
7	Microsurgical proximal occlusion	P2	Fusiform	Infarction
8	Microsurgical proximal occlusion	P1/2	Fusiform	—
9	Microsurgical proximal occlusion	P2	Fusiform	Infarction
10	Endovascular aneurysm and parent vessel occlusion	P3	Fusiform	—
11	Endovascular aneurysm and parent vessel occlusion	P3	Fusiform	Infarction

and 3 died after severe vasospasm and multiple infarctions. Three of the patients without an SAH died: 2 after DSA complications and 1 of a cardiac infarction. Of the 2 vegetative patients, 1 suffered an intraoperative aneurysm rupture and the other an associated AVM rupture. In 11 patients, the PCA parent vessel was occluded and 6 of these show a symptomatic PCA infarction (Table 5). Six patients had an incomplete aneurysm occlusion after neck clipping, 1 of whom underwent a second surgery for aneurysm trapping. An incomplete aneurysm occlusion was observed in 5 patients after coil embolization, 2 of whom underwent subsequent neck clipping. Nine patients developed oculomotor nerve palsy after microsurgical treatment, 1 developed trochlear nerve palsy after microsurgical treatment, and 2 developed oculomotor nerve palsy after endovascular treatment.

DISCUSSION

The presented series contains 121 patients and is therefore one of the largest studies of PCA aneurysms. Treatment strategies and associated outcomes were retrospectively analyzed. All patients were treated at 1 of 2 neurosurgical centers, with a defined catchment time range of 1980–2014.

Because of the low incidence of PCA aneurysms, which account for less than 2% of all aneurysms, previous experience is mainly based on single case descriptions or small case series of fewer than 25 patients, which are often reported together with other posterior circulation aneurysms. The exception to this trend is the highly selected series by Drake et al.[1]

Table 6. Characteristics of 12 Patients with Ruptured Posterior Cerebral Artery Aneurysms Treated Between 1954 and 2012

Variables	Number of Patients
Patients	12
Posterior cerebral artery aneurysms	12
Gender	
Male	5
Female	7
Age at diagnosis (years), mean (range)	43 (23–63)
Aneurysm size	
Small (<7 mm)	8
Medium (7–14 mm)	3
Large (15–24 mm)	1
Hunt and Hess Grade	
Unruptured	4
Grade 1	0
Grade 2	2
Grade 3	3
Grade 4	0
Grade 5	3
Treatment	
Conservative	12
Outcome (latest follow-up)	
mRS 0–1 (good)	1
mRS 2–4 (moderate)	5
mRS 5–6 (poor)	6

mRS, modified Ranking Scale.

Historical Aspects

Our historical series of 12 patients (1954–1980) shows a high rate (50%) of unfavorable outcomes after conservative treatment but only 4 patients had an unruptured PCA aneurysm, 1 of whom had an SAH after 42 years of conservative treatment (Table 6). A case report on a patient with a fatal outcome after a giant distal PCA aneurysm rupture was given by Bertrams in 1968[31] and perfectly reflects our impression of the historic patient records. At that time, an active and mostly microsurgical treatment was becoming increasingly important.[32] During the last 3 decades, endovascular techniques have evolved rapidly and PCA aneurysms close to the brainstem are now treated using endovascular techniques at many institutions.[4,7,16]

Clinical Presentation

The overall H&H grade was no higher for proximal PCA aneurysms than for distal PCA aneurysms; however, 5 of the 9 ruptured P3 aneurysms led to an IVH or ICH, highlighting the danger of

distal aneurysms. When compared with the second largest series on PCA aneurysms, our series showed a less frequent occurrence of cranial nerve disturbances (6% vs. 21%), which is caused by our lower rate of large and giant aneurysms (12% vs. 21%).[1]

Special Features of PCA Aneurysms

PCA aneurysms are often associated with multiple aneurysms, AVMs, a complex angioarchitecture, and a high percentage of fusiform aneurysms.[1,27] We consider 30% of our cases to be complex. We found more than 60% of patients with associated vascular lesions and 21% of the aneurysms were fusiform. Most aneurysms were located at the proximal PCA segments (P1 and P1/2 junction) and 35% of these were ruptured. Of the ruptured PCA aneurysms, 46% were smaller than 7 mm and only 12% were large or giant. The P2 segment was most often affected by fusiform aneurysms. These proximal segments are surgically accessible by standard frontotemporal or subtemporal approaches and endovascular techniques are also applicable.[1,4,7,16,17,19,28] In particular, PCA aneurysms distal to the circle of Willis have nearly identical rupture rates (38% vs. 37%). However, their deep anatomic location close to sensitive neuroanatomic structures and a long intravasal route to the terminal segments of the posterior circulation make treatment more challenging. The incidence of ICH after PCA aneurysm rupture is low, presumably because of the relatively low flow rates compared with other intracranial vessels[33] and the outflow in the basal cisterns. However, the rate of IVH after P3 aneurysm rupture was high because of closeness to the lateral ventricles.

Treatment Strategies for Saccular PCA Aneurysms

Proximal saccular PCA aneurysms are accessible by microsurgical and endovascular means. In the presented series, treatment was carried out mostly by microsurgical means, which allows for direct visual control of the important perforating branches. The most common approaches for these aneurysms are the frontotemporal and subtemporal approaches.[1,6,28,29,32] However, the most frequent complications were not related to the microsurgical approach itself but more to the chosen strategy. Endovascular treatment was used for 17 patients with 20 PCA aneurysms, but the primary aneurysm occlusion rate was only 70%, compared with 90% in the microsurgical group. A complete closure of the aneurysm by endovascular techniques often requires a parent vessel occlusion.[4,7,34] We found both endovascular and open microsurgical techniques suitable for the occlusion of aneurysms distal from the P2 segment.

Treatment Strategies for Fusiform PCA Aneurysms

A fusiform shape is common in aneurysms affecting the PCA. Perforating branches can originate from the affected segment and the aneurysm shape complicates a direct surgical clip occlusion. Clip occlusion was possible in 7 of 29 of these aneurysms (29%); however, 2 of these were only partially occluded. Proximal occlusion or wrapping of aneurysms must be performed with great caution to prevent devastating infarction. We can recommend this treatment strategy only as an ultima ratio option. In our opinion, reconstructive procedures should be discussed for these cases and a balloon occlusion test can prove helpful, despite the risks.[19,35-39]

Revascularization Procedures for PCA Aneurysms

Based on our own results, we have to be critical regarding proximal PCA occlusions. The appearance of PCA infarction after PCA occlusion is a compromising outcome factor. Therefore, we changed our own treatment strategy for PCA aneurysms. We found a protective superficial temporal artery-PCA (P2/3) bypass from a subtemporal approach to be useful in 2 patients, where a direct microsurgical or endovascular aneurysm treatment was not possible, to prevent territorial stroke in the distal PCA supply area.

Treatment Strategies for PCA Aneurysms in Association with Multiple Aneurysms

The treatment of patients with multiple aneurysms is demanding. However, the general rules of aneurysm treatment are also applicable to PCA aneurysms: 1) the first intervention is focused on the ruptured or likely-to-rupture aneurysm to prevent rebleeding; 2) the most technically secure method is used; 3) if possible, all reachable aneurysms are occluded during the same procedure; 4) other treatable aneurysms are approached after the acute phase; 5) aneurysms without indication for active treatment are observed. Using this methodology, we were able to manage 67 patients with multiple aneurysms. Frontotemporal craniotomies allow access to most ipsilateral anterior circulation aneurysms.[29] However, endovascular techniques also allow the occlusion of several aneurysms during a single intervention.[40]

Treatment Strategies for PCA Aneurysms in Association with AVM

Only 3 aneurysms of AVM-feeding vessels were addressed by direct treatment. The low rupture rate during follow-up supports this decision history, which is comparable with previous reports on flow-related aneurysms and AVM treatment.[41,42] Regardless, the decision-making process for each of these individual cases is complex.

Complications

The most serious and outcome-affecting complications are rerupture and the occurrence of ischemic infarctions after parent vessel occlusion. The observed frequency of ischemic lesions after parent vessel occlusion appears to be higher than in other microsurgical and endovascular series.[1,7] Contrary to the general opinion that parent artery occlusion is a safe treatment option for distal PCA aneurysms, their preservation is critical for a favorable outcome. The rate of intraoperative third nerve injuries shows that solely visual control is insufficient and intraoperative monitoring of the third and fourth nerves is therefore highly recommended.[43]

Limitations

The major limitation of this study is the retrospective nature of the data analysis. There was no randomization and the treatment decision was based on an individual case discussion regarding the PCA aneurysm morphology and patient condition. Because of multiple associated vascular lesions, the patient group became heterogeneous. Regardless, the presented series represents the largest collection of institutional experience with this rare disease.

CONCLUSIONS

Aneurysms of the PCA are infrequent and often associated with other vascular diseases. As a result, individual treatment strategies are required. Both microsurgery and endovascular treatment are effective options for the occlusion of PCA aneurysms. Despite commonly adequate vessel collateralization of the distal PCA territory, preservation or reconstruction of the parent vessel is crucial for favorable treatment outcomes. The rate of intraoperative third nerve injuries shows that solely visual control is insufficient and intraoperative monitoring is recommended.

REFERENCES

1. Drake CG, Peerless SJ, Hernesniemi JA. *Surgery of Vertebrobasilar Aneurysms. London, Ontario Experience on 1767 Patients*. Vienna: Springer-Verlag; 1996.
2. Honda M, Tsutsumi K, Yokoyama H, Yonekura M, Nagata I. Aneurysms of the posterior cerebral artery: retrospective review of surgical treatment. *Neurol Med Chir (Tokyo)*. 2004;44:164-168.
3. Locksley H. Report on the cooperative study of intracranial aneurysms and subarachnoid hemorrhage. Section 5, part 1. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. Based on 6368 cases in the operative study. *J Neurosurg*. 1966;25:219-239.
4. Ciceri EF, Klucznik RP, Grossman RG, Rose JE, Mawad ME. Aneurysms of the posterior cerebral artery: classification and endovascular treatment. *AJNR Am J Neuroradiol*. 2001;22:27-34.
5. Coert BA, Chang SD, Do HM, Marks MP, Steinberg GK. Surgical and endovascular management of symptomatic posterior circulation fusiform aneurysms. *J Neurosurg*. 2007;106:855-865.
6. Gerber C, Neil-Dwyer G, Evans BT. An alternative approach to aneurysms of the posterior cerebral artery. *J Neurosurg*. 1993;32:928-931.
7. Hallacq P, Piotin M, Morert J. Endovascular occlusion of the posterior cerebral artery for the treatment of P2 segment aneurysm: retrospective review of a 10-years series. *AJNR Am J Neuroradiol*. 2002;23:1128-1136.
8. Hamada J, Morioka M, Yano S, Todaka T, Kai Y, Kuratsu J. Clinical features of aneurysms of the posterior cerebral artery: a 15-year experience with 21 cases. *Neurosurgery*. 2005;56:662-670.
9. Kashiwazaki D, Ushikoshi S, Asano T, Osanai T, Kuroda S, Houkin K. Endovascular treatment for aneurysms of the posterior cerebral artery: 12 years' experience with 21 cases. *Acta Neurochir (Wien)*. 2011;153:2151-2158.
10. Li Y, Lv X, Jiang C, Liu A, Wu Z. Endovascular treatment of posterior cerebral artery aneurysms. *Neuroradiol J*. 2008;21:128-136.
11. Luo Q, Wang H, Xu K, Yu J. Endovascular treatments for distal posterior cerebral artery aneurysms. *Turk Neurosurg*. 2012;22:141-147.
12. Park W, Kwon do H, Ahn JS, Lee SH, Park JC, Kwun BD. Treatment strategies for dissecting aneurysms of the posterior cerebral artery. *Acta Neurochir (Wien)*. 2015;157:1633-1643.
13. Roh HG, Kim SS, Han H, Kang HS, Moon WJ, Byun HS. Endovascular treatment of posterior cerebral artery aneurysms using detachable coils. *Neuroradiology*. 2008;50:237-242.
14. Sakata S, Fujii K, Matsushima T, Fujiwara S, Fukui M, Matsubara T, et al. Aneurysm of the posterior cerebral artery: report of eleven cases—surgical approaches and procedures. *Neurosurgery*. 1993;32:163-167.
15. Seoane E, Tedeschi H, De Oliveira E, Siqueira M, Calderon G, Rhoton A Jr. Management strategies for posterior cerebral artery aneurysms: a proposed new surgical classification. *Acta Neurochir (Wien)*. 1997;139:325-331.
16. van Rooij WJ, Sluzewski M, Beute GN. Endovascular treatment of posterior cerebral artery aneurysms. *AJNR Am J Neuroradiol*. 2006;27:300-305.
17. Yonekawa Y, Roth P, Fandino J, Landlot H. Aneurysms of the posterior cerebral artery and approach selection in their microsurgical treatment: emphasis on their approaches: SAHEA and SCTTA. *Acta Neurochir (Wien)*. 2011;112(suppl):85-92.
18. Zhitao J, Yibao W, Anhua W, Shaowu O, Yunchao B, Renyi Z, et al. Microsurgical subtemporal approach to aneurysms on the P2 segment of the posterior cerebral artery. *Neurol India*. 2010;58:242-247.
19. Chang SW, Abla AA, Kakarla UK, Sauvageau E, Dashti SR, Nakaji P. Treatment of distal posterior cerebral artery aneurysms: a critical appraisal of the occipital artery-to-posterior cerebral artery bypass. *Neurosurgery*. 2010;67:16-25.
20. Wang WX, Xu BN, Wang FY, Wu C, Sun ZH. Microsurgical management of posterior cerebral artery aneurysms: a report of thirty cases in modern era. *Br J Neurosurg*. 2015;29:406-412.
21. Taylor CL, Kopitnik TAJR, Samson DS, Purdy PD. Treatment and outcome in 30 patients with posterior cerebral artery aneurysms. *J Neurosurg*. 2003;99:15-22.
22. Sanai N, Tarapore P, Lee AC, Lawton MT. The current role of microsurgery for posterior circulation aneurysms: a selective approach in the endovascular era. *Neurosurgery*. 2008;62:1236-1249.
23. Kim YB, Lee JW, Huh SK, Kim BM, Kim DJ. Outcomes of multidisciplinary treatment for posterior cerebral artery aneurysms. *Clin Neurol Neurosurg*. 2013;115:2062-2068.
24. Zeal AA, Rhoton AL. Microsurgical anatomy of the posterior cerebral artery. *J Neurosurg*. 1978;48:534-559.
25. Párraga RG, Ribas GC, Andrade SE, Oliveira E. Microsurgical anatomy of the posterior cerebral artery in three-dimensional images. *World Neurosurg*. 2011;75:233-257.
26. Saeki N, Rhoton AL. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. *J Neurosurg*. 1977;46:563-578.
27. Goehre F, Jahromi BR, Hernesniemi J, Elsharkawy A, Kivisaari R, von und zu Fraunberg M, et al. Characteristics of posterior cerebral artery (PCA) aneurysms: an angiographic analysis of 93 aneurysms in 81 patients. *Neurosurgery*. 2014;75:134-144.
28. Goehre F, Lehecka M, Jahromi BR, Lehto H, Kivisaari R, Hijazy F, et al. Subtemporal approach to posterior cerebral artery aneurysms. *World Neurosurg*. 2015;83:842-851.
29. Yasargil MG. *Microsurgery. Clinical Considerations, Surgery of the Intracranial Aneurysms and Results*. Stuttgart, Germany: Georg Thieme-Verlag; 1984.
30. Lavine SD, Meyers PM. Application of new techniques and technologies: stenting for cerebral aneurysm. *Clin Neurosurg*. 2007;54:64-69.
31. Bertrams J. Das periphere Aneurysma der A. cerebri posterior. *Dtsch Z Nervenheilkd*. 1968;194:243-251 [in German].
32. Drake CG, Amacher AL. Aneurysms of the posterior cerebral artery. *J Neurosurg*. 1969;30:468-474.
33. Hennerici M, Rautenberg W, Sitzer G, Schwartz A. Transcranial Doppler ultrasound for the assessment of intracranial arterial flow velocity—Part 1. Examination technique and normal values. *Surg Neurol*. 1987;27:439-448.
34. Cotroneo E, Gigli R, Guglielmi G. Endovascular occlusion of the posterior cerebral artery in the treatment of P2 ruptured aneurysms. *Interv Neuroradiol*. 2007;13:127-132.
35. Goehre F, Kamiyama H, Noda K, Ota N, Tsuboi T, Miyata S, et al. Technical description of the medial and lateral anterior temporal approach for the treatment of complex proximal posterior cerebral artery aneurysm. *World Neurosurg*. 2016;86:490-496.
36. Lawton MT, Hamilton MG, Morcos JJ, Spetzler RF. Revascularization and aneurysm surgery: current techniques, indications, and outcome. *Neurosurgery*. 1996;38:83-94.
37. Linskey ME, Jungreis CA, Yonas H, Hirsch WL Jr, Sekhar LN, Horton JA, et al. Stroke risk after abrupt internal carotid artery sacrifice: accuracy of preoperative assessment with balloon test occlusion and stable xenon-enhanced CT. *AJNR Am J Neuroradiol*. 1994;115:829-843.
38. Mathis JM, Barr JD, Jungreis CA, Yonas H, Sekhar LN, Vincent D, et al. Temporary balloon test occlusion of the internal carotid artery: experience in 500 cases. *AJNR Am J Neuroradiol*. 1995;116:749-754.
39. Origitano TC, al-Mefty O, Leonetti JP, DeMonte F, Reichman OH. Vascular considerations and complications in cranial base surgery. *Neurosurgery*. 1994;35:351-363.

40. Shen X, Xu T, Ding X, Wang W, Liu Z, Qin H. Multiple intracranial aneurysms: endovascular treatment and complications. *Interv Neuroradiol*. 2014;20:442-447.

41. Meisel HJ, Mansmann U, Alvarez H, Rodesch G, Brock M, Lasjaunias P. Cerebral arteriovenous malformations and associated aneurysms: analysis of 305 cases from a series of 662 patients. *Neurosurgery*. 2000;46:793-800.

42. Redekop G, Terbrugge K, Montanera W, Willinsky R. Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. *J Neurosurg*. 1998;89:539-546.

43. Zhou Q, Zhang M, Jiang Y. Intraoperative oculomotor nerve monitoring predicts outcome following clipping of posterior communicating artery aneurysms. *J Clin Neurosci*. 2012;19:706-711.

Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received 11 February 2016; accepted 21 March 2016

Citation: World Neurosurg. (2016) 92:521-532.
http://dx.doi.org/10.1016/j.wneu.2016.03.063

Journal homepage: ***www.WORLDNEUROSURGERY.org***

Available online: ***www.sciencedirect.com***