Posterior Communicating Artery Aneurysms
Technical Pitfalls

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Objective: The posterior communicating artery aneurysms correspond on 25% of all ruptured aneurysms. The clinical course is typically a subarachnoid hemorrhage and third nerve palsy. We intend to introduce a new classification for PComA aneurysms to help neurosurgeons in day-to-day practice present. We review our experience in PComA aneurysms and discuss the main factors involving morbidity, mortality, signs and symptoms, and prognosis of these aneurysms.

Material and Methods: We reviewed historical records, images, surgical videos, and CDs of 46 surgically clipped aneurysms in 39 patients from June 2000 to July 2009, in 2 Institutions: Hospital São Camilo and Santa Paula, São Paulo, Brazil. They were classified in 2 groups, the A group composed by patients who presented subarachnoid hemorrhage in acute phase and the B group composed by incidental aneurysms carriers. All patients were classified according to Hunt-Hess scale.

Results: The average age found was 53.6 years old (min 28 to Max 92). The incidence was higher among women (3.6:1). Worse outcomes were observed in group A. The mortality rate was 20% in group A and zero cases in group B. Similar rate was found for rupture cases (20% in A group vs zero in B group). Morbidity was similar for both groups. The mean aneurismal size for A group was 6 mm (ranging from 5 to 25 mm) and 5.3 mm (ranging from 3 to 10 mm) for B group.

Conclusions: Posterior communicating artery aneurysms occurred 3 to 4 times more frequently in women than man. Oculomotor palsy associated with severe headache were commonly related to posterior circulation aneurysms. Type II aneurysms (temporal) were the most frequently found in our study. The worst prognosis in cases with acute bleeding occurred with fetal variant circulation. Intratentorial aneurysms, mainly those with increased Hunt-Hess, have the worst prognosis. Infundibular aneurysms had the best results with surgical clipping.

Key Words: intracranial aneurysms, posterior communicating artery, surgical technique

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Intracranial aneurysms affect approximately 2% to 5% of the population and those that rupture present typically with subarachnoid hemorrhage (SAH). Twenty-five percent of the ruptured aneurysms are classified as posterior communicating artery aneurysm.[1]

The Posterior Communicating Artery (PComA) arises from the posteromedial aspect from the carotid artery (segment C4) midway between the origin of the ophthalmic artery and the terminal bifurcation and course in a posterior direction medial to the oculomotor nerve to join the posterior cerebral artery (PCA). The oculomotor nerve is an important anatomic landmark in surgical anatomy, which enters the dura lateral to the clinoid process. The PComA on its course gives off an average of 2–10 branches, which begin approximately 2 to 3 mm from the origin, and they penetrate the tuber cinereum and premmamilary part of the floor of the third ventricule, mammilary bodies, subthalamus, posterior hypothalamus, anterior thalamus, the optic tract, and the internal capsule[2–5] (Fig. 1). The PComA frequently serves as a vital physiologic collateral between the anterior and posterior cerebral circulation, helping in the maintenance of adequate cerebral perfusion and the completeness of the circle of Willis.

The posterior communicating artery is commonly described as the adult configuration if the diameter of the precommunicating part of the posterior cerebral artery P1 is larger than the diameter of the posterior communicating artery itself (PCA). The finding that the diameter of PCA can be clearly larger than diameter of the P1 is designated as the fetal variant circulation. This nomenclature refers to the embryonic situation in which a large branch arising from the internal carotid artery is the major source of blood supply to the occipital lobes.[6] The overall incidence of the fetal-type posterior circulation has been reported to occur in 4% to 29% of patients, bilaterally occurring in 1% to 9% of patients.[7–11]

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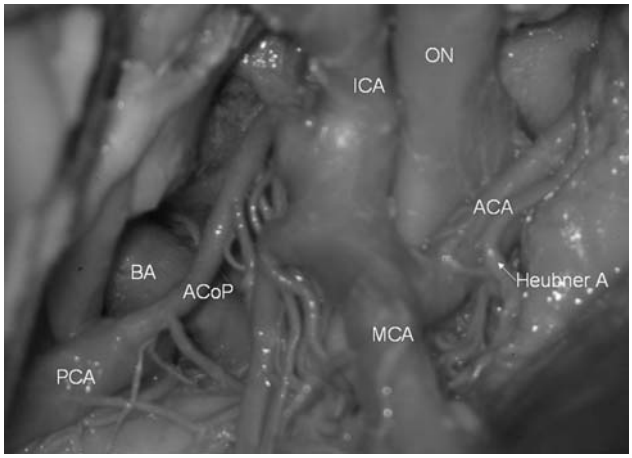


FIGURE 1. Anatomy of posterior communicating artery (PCoA). ACA indicates anterior cerebral artery; ACop, anterior communicating artery; BA, basilar artery; Heubner A, Heubner artery; ICA, internal carotid artery; MCA, middle cerebral artery; ON, optic nerve; PCA, posterior cerebral artery.

ICA-PComA aneurysms, commonly refered to as PComA aneurysms, are tipically not aneurysms of the PComA itself but instead are aneurysms of the ICA occurring at the origin of the PComA. True posterior communicating artery aneurysms are extremely rare with incidence ranges from 0.1% to 2.8% of all aneurysms.[12,13] According to Horikoshi et al in 2002, a greater relative frequency in ICA-PComA aneurysms has been reported to occur in patient with fetal variant circulation.[14] Fetal variant vessels were significantly more common in women, and they carry a greater risk profile in regard to injury to the fetal PCA vessel.[15]

The posterior communicating artery aneurysms are sacular and take place in the posterior wall of the carotid artery, near from the beginning of the PComA. They can arise bellow the tentorium and to the temporal lobe.[16]

Giant aneurysms are also described for posterior circulation. They represent approximately 5% of all aneurysms.[17] The diameter is, by definition, 25 mm or more.[18,19] If untreated, the 2-year mortality rate for persons with giant aneurysms is between 60 and 100%.[20]

Currently evaluation, typically using size alone, is the mainstay of applied aneurysm ruptured risk assessment, something in day-to-day clinical practice.

Data from international study of unruptured intracranial aneurysms (ISUIA) calculated a much lower risk of rupture (0% and 0.1% per year) in aneurysms less than 6 mm in diameter and 7 to 12 mm in diameter when the patient is asymptomatic. The limit of the size that can be considered safer has been revised from 9 to 6 mm in the ISUIA.[21]

Ruptured intracranial aneurysms typically cause SAH and its sequelae, resulting in significant morbidity and mortality.[22] The symptoms may be mild, such as headaches, or manifestation may be more severe, such as signs related to subarachnoid hemorrhage, and the palsy of third nerve.[23–25] Seizures have not been usually ascribed to the aneurysms of the PComA. Onset may be acute by sudden increase in aneurysm volume as a result of aneurysm wall dissection with or without accompanying SAH. In a minority of patients, CN III palsy is the only clinical presentation of a PComA aneurysm. Other clinical presentations mimic an acute subdural hematoma[25] and abducens nerve palsy.[26,27] Of the patients who had SAH from a ruptured aneurysm, about 20% have more than one aneurysm at the time of presentation and have a continuing risk of developing new aneurysms. Smoke and female gender are known risk factors for new aneurysm formation and SAH.[28]

Complicating factors of surgery are age, commorbidities, fetal posterior communicating artery, severe vasospasm, low grade of Hunt Hess at admission and high grade of cistern blood.[29,30] A well-recognized complication of surgical or endovascular obliteration of PComA aneurysms is the inadvertent injury to the PComA itself, or to related perforating arteries, which may result in ischemic injury to dependent regions. This scenario is especially true in the case of a fetal variant circulation, where inadvertent occlusion of the dominant feeder to these regions can be deleterious, causing potential infarction of the midbrain, thalamus, and occipital region.[5,31] Other fatal complications have been associated with neurogenic pulmonary edema,[32] fistula, and cavernous sinus.[33]

The aim of this study is to propose a new classification for the PComA aneurysms based on the authors experience and in extensive literature research, to help neurosurgeons with the decision about the best surgical treatment for these aneurysms. The analysis focused also on morbidity, mortality, risk of rupture, and evolution of each case.

MATERIAL AND METHODS

The investigators reviewed historical records, images, surgical videos, and CDs of 46 surgically clipped aneurysms in 39 patients from June 2000 to July 2009, in 2 Institutions: Hospital São Camilo and Santa Paula, São Paulo, Brazil.

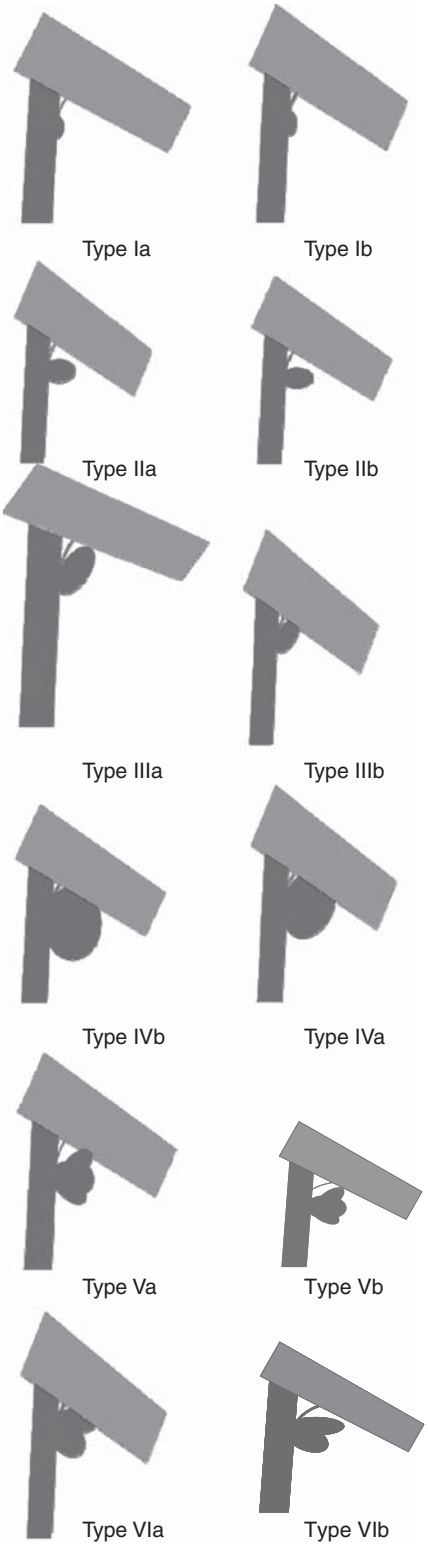
They were classified in 2 groups: (1) The A group consisted of 15 patients who presented SAH and were clipped in acute phase (22 aneurysms in total; 16 PComA aneurysms); and (2) The B group consisted of 11 patients with incidental PComA, in a total of 24 aneurysms. All patients were classified according to Hunt-Hess scale.

The investigators suggest a new classification for PComA according to: Temporal or tentorial localization, the presence or absence of fetal variant circulation, aneurysm size and aneurysm shape (sacular, infundibular, or giant). The following classification is elaborated in two different views (Figs. 2, 3).

RESULTS

The average age found was 53.6 years old (28 to 92). The incidence was higher among women (3.6:1).

Regarding group A, the mortality rate was 3 cases in 15 patients who presented intraoperative rupture and developed severe ischemia after clipping. All of those cases had Fisher cistern grade III-IV, and 2/3 were admitted in



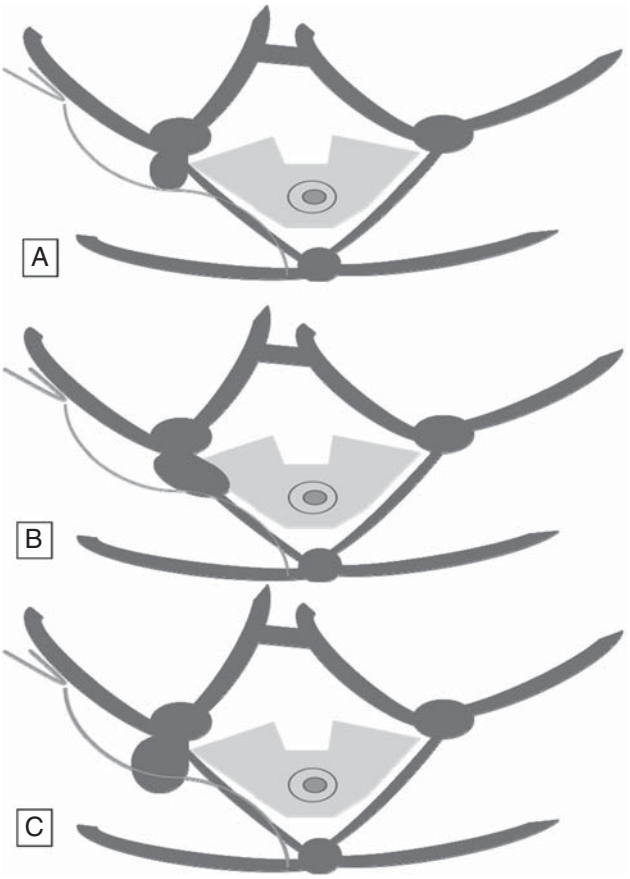


FIGURE 3. Diagram of superior view and relationship of tentorium and the different types of the posterior communicanting aneurysms. A- Type II, B- Type III and C: type IV.

Hunt-Hess scale IV. The morbidity rate showed 1 patient with aphasia and hemiparesis, who suffered embolic ischemic stroke in left side basal ganglia in the post-operative period. The mean size of group A aneurysms was 6 mm (ranging from 5 to 25 mm).

Severe vasospasm was found in 2 cases, moderate in 1 case and mild in 3 cases. The diagnosis was based on transcranial Doppler (Tables 1 and 2).

FIGURE 2. Types of posterior communicating aneurysms by Aguiar e cols. Type IA: infundibular aneurysms without fetal variant circulation; Type Ib: infundibular aneurysms with fetal variant circulation; Type IIA: temporal aneurysms without fetal variant circulation; Type IIB: temporal aneurysms with fetal variant circulation; Type IIIA: tentorial aneurysms without fetal variant circulation; Type IIIB: tentorial aneurysms with fetal variant circulation; Type IVA: giant aneurysms without fetal variant circulation; Type IVB: giant aneurysms with fetal variant circulation; Type VA: multilobulated aneurysms without fetal variant circulation; Type VB: multilobulated aneurysms with fetal variant circulation; Type VIA: intratentorial multilobulated aneurysms without fetal variant circulation; Type VIB: intratentorial multilobulated aneurysms with fetal variant circulation.

TABLE 1. Morbility, Mortality and Hunt-Hess Score in SAH Cases

	n = 22	Mortality	Morbity	Hunt Hess IV/V
Type Ia	1	N	N	N
Type Ib	0	N	N	N
Type IIa	10	1	1	1
Type IIb	0	N	N	N
Type IIIa	4	0	1	0
Type IIIb	0	N	N	N
Type IVa	2	0	0	0
Type IVb	1	1	0	1
Type Va	2	0	0	0
Type Vb	2	1	0	1
Type VIa	0	N	N	N
Type VIb	0	N	N	N

N indicates none; n, number of cases.

The pterional approach followed by a subfrontal route and carotid optic cistern dissection was the operation chosen for those aneurysms. External ventricular drainage was necessary in 3 cases of 15. Temporary clipping was used in the 3 cases with intraoperative rupture and mortality. The B group, with incidental aneurysms, presented no mortality and 1 case of disability with transient third nerve palsy. The mean arterial size of Group B was 5.3 mm (ranging from 3 to 10 mm). There was no intraoperative rupture. There was no postoperative vasospasm. The main approach was the pterional approach followed by subfrontal acess to aneurysm neck, and dissection of basal cisterns. There was no necessity of transient clipping (Figs. 4, 5).

DISCUSSION

The posterior communicating artery is responsible for feeding the tuber cinerium, posterior perforated substance, optic chiasm, posterior hypothalamus, and posterior limb of internal capsule.[34] The PComA courses in the posteromedial direction toward the interpeduncular fossa and joins the posterior cerebral artery, marking the beginning of the P2 segment.[6] A superolateral course of the artery toward the oculomotor been reported when the fetal configuration is present.[35]

TABLE 2. Morbidity, Mortality and Hunt-Hess Score in Incidental Cases

	n = 26	Mortality	Morbility	Folow-up
Type Ia	6	0	1*	4/4
Type Ib	0	N	N	
Type IIa	10	0	0	9/10
Type IIb	2	0	0	2/2
Type IIIa	2	0	0	1/2
Type IIIb	2	0	0	
Type IVa	1	0	1	1/1
Type IVb	2	0	1*	2/2
Type Va	0	N	N	
Type Vb	1	0	0	1/1
Type VIa	0	N	N	
Type VIb	0	N	N	

N indicates none; n, number of cases.

Injury or occlusion of the fetal PComA may result in occipital infarcts and subsequent clinical complications such as homonymous hemianopsia, alexia, aphasia, and hemichromatopsia.[13] Perforators originating from the PComA may also be compromised secondary to inadvertent clipping of the parent PComA vessel, causing midbrain or thalamic injury as reported by Zada.[36]

The posterior communicating artery may have a fetal pattern in 14.6% in health persons and 33% in patients with aneurysms.[1] In those patients with aneurysms the clipping should preserve the fetal artery, otherwise the brainstem circulation might be compromised. The posterior communicating artery aneurysms as a rule have their origin superiorly and laterally to the posterior communicating artery and are projected to tentorial surface or to temporal lobe.[16]

The usually mentioned types of aneurysms of posterior circulation in literature are fusiform, microaneurysms, giant and sacular aneurysms by Sugita (1981).[37,38]

Recent studies have indicated that intracranial aneurysm size may be a primary determinant of rupture probability[39] and many earlier series have implicated size as an important factor in aneurysm rupture.[40]

The incidence of SAH in Japan is 17 to 96 per 100,000 population per year, which is higher than in other developed countries.[41]

Reported critical sizes at which the incidence of rupture increases for aneurysms are from 4 to 10 mm. Zacks[42] stated that unruptured aneurysms <10 mm have a good prognosis without surgical treatment. Wiebers[43] and others from the Mayo clinic claim that there is a critical diameter of 10 mm below which aneurysms rarely rupture.

The recommendations of the American Heart Association recommendations show there is an apparent low risk of hemorrhage from incidental small (<10 mm) aneurysms.[44]

According to retrospective ISUIA data.[45] the rate of rupture of aneurysms less than 10 mm diameter was less than 0.05% per year in patients with no SAH history and 0.5% per year in patients with previous history of SAH. Additionally, in prospective ISUIA results,[46] the annual rupture rates for patients without a history of SAH with aneurysms located in the posterior circulation were 0.5% for 7-mm aneurysms diameter and 2.9% for 7 to 12 mm diameter. These results suggest that small aneurysms are relatively safe and not likely to rupture easily.

Many other studies also reported rupture rate of unruptured intracranial aneurysms: Juvela et al found 1.3% annual risk of rupture for unruptured aneurysms.[47,48] Morita reported a 2.7% risk of rupture.[41] In our study, ruptured aneurysms were most identified among temporal aneurysms without fetal variant circulation.

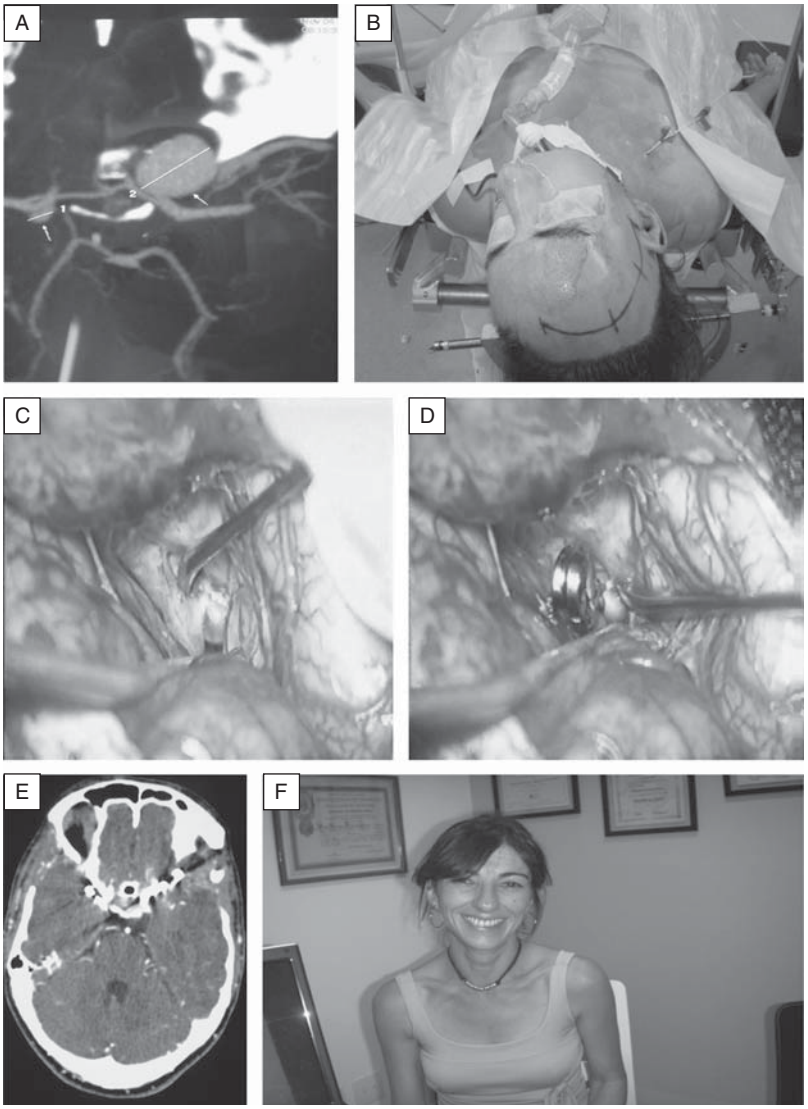


FIGURE 4. A, Magnetic resonance angiography showing a giant posterior communicating artery aneurism Type IVA; (B) preoperative positioning. C, Visualization of the aneurysm after lateral fissure opening. D, Aneurysm view after clipping; (E) CT after surgery. F, Patient after aneurysm clipping.

Van der Ark et al 1972 showed an anatomical study that 35% are projected posteriorly, 24% are projected superiorly in direction to tentorial notch, 13% supero medially, and only 2% inferiorly and medially.[16]

Management of large PComA aneurysms is still controversial. Parent vessel preservation or sacrifice has been debated, and many approach techniques have been proposed. They impose morphologic and accessibility difficulties, either surgical, endovascular, or combined.[49–60]

In this study, we propose a new classification for the PComA aneurysms considering their development, anatomical and surgical peculiarities and the implication of fetal pattern for the PComA. We intend to make a more detailed and specific classification to stratify risks based on the variety of aneurysms shape, not just size, and the different directions they assume intratentorial.

CONCLUSIONS

Posterior communicating artery aneurysms occurred 3 to 4 times more frequently in women than man, as mentioned previously by other authors.

Symptoms were unspecific, nevertheless oculomotor palsy associated to severe headache were commonly related to posterior circulation aneurysms. The Vb type, multilobulated, may present carotid walls fragility and had higher rates of thrombosis. Type II aneurysms, temporal types, were the most frequently founded in our study. The worst prognosis in cases with acute bleeding was shown in fetal variant circulation as

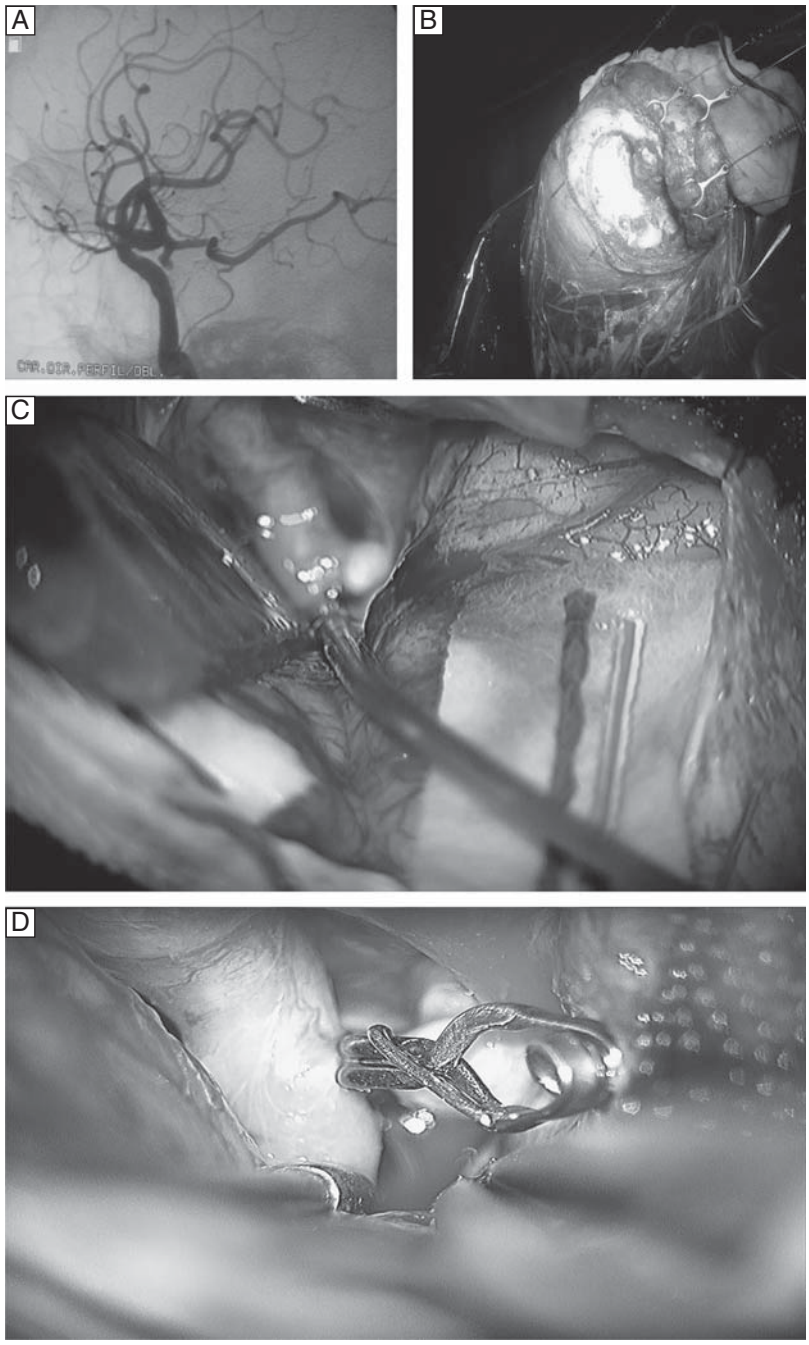


FIGURE 5. A, Arteriography evidence an infundibular PComA aneurysm Type VA; (B) pterional approach; (C) microsurgical visualization of the aneurysm; (D) microsurgical view after clipping.

described in previous researches. Intratentorial aneurysms, mainly those with increased Hunt-Hess, have the worst prognosis. Infundibular aneurysms had the best results with surgical clipping.

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