

## Review Article

# Progress in treating ruptured infundibular dilatation at the origin of the intracranial posterior communicating artery

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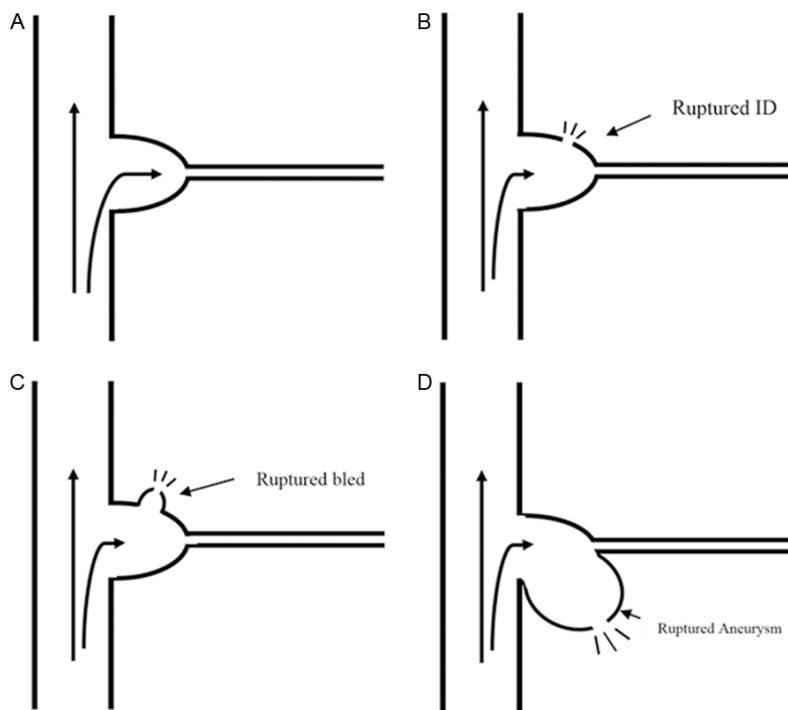
**Abstract:** Infundibular dilatation (ID) can occur at the origin of the intracranial posterior communicating artery. When this type of widening is less than 3 mm in diameter and the ID reaches the posterior communicating artery, it can be called posterior communicating artery infundibular dilatation (Pcom-ID). Currently, Pcom-ID is considered a normal anatomic variation, and the majority of Pcom-IDs are stable. However, in some cases, rupture occurs in a Pcom-ID; furthermore, Pcom-IDs can evolve into aneurysms. There are not many studies on Pcom-ID rupture hemorrhage; therefore, we performed a retrospective evaluation of published studies on Pcom-ID rupture hemorrhage and conducted a classification analysis for this condition. It is reasonable to classify Pcom-ID into three types: Type 1, direct rupture of the Pcom-ID; Type 2, bleb rupture of Pcom-ID; and Type 3, Pcom-ID rupture caused by an aneurysm. This type of classification can provide meaningful guidance for treating ruptured Pcom-ID. In addition, the present study also included a systematic review and summarization of the literature on each type. This investigation was aimed to improve the understanding of Pcom-ID rupture hemorrhage.

**Keywords:** Infundibular dilatation, rupture, treatment, posterior communicating artery

## Introduction

Dilatation may occur where the intracranial posterior communicating artery originates from the internal carotid artery. When a dilatation becomes obvious, forming a uniform conical or triangular shape on radiological image, this unusual arterial dilatation is regarded as posterior communicating artery infundibular dilatation (Pcom-ID). The diameter of an ID is often less than 3 mm, and the posterior communicating artery grows at the distal end of the ID [1, 2]. Ebina *et al.* reported (in a study in Japan in 1986) that the incidence of Pcom-ID was 7%-25% in brain digital subtraction angiography (DSA) [3]. Recently, Vlajković discovered that the incidence of Pcom-ID was 2.2% from an autopsy study on a Serbian population in 2015 [4]. It is suggested that the incidence of Pcom-ID may differ between the general population and the cerebrovascular-disease population receiving DSA. This difference between populations may also be a result of racial differ-

ences. The incidence of Pcom-ID in the general population has not been reported due to the lack of large-scale surveys. The main motivation to study Pcom-ID is that rupture and hemorrhage can be found clinically in Pcom-ID, and this dilatation is suggested to gradually evolve into aneurysms [5, 6]. Currently, the understanding of the natural history of Pcom-ID has been very limited. In early 1959, Saltzman performed a related study on Pcom-ID rupture hemorrhage. Studies regarding Pcom-ID hemorrhage and its progression to aneurysms have continued up to this day [7]. Particularly, Pcom-ID-induced hemorrhage has confused clinical physicians, particularly, the judgment of whether subarachnoid hemorrhage (SAH) is caused by ID, which type of ID easily progresses into an aneurysm, and what treatment to use for ID after hemorrhage occurs. In 2010, the author of this article has encountered a case of Pcom-ID and achieved good results with treatment [8]. In addition to the articles published during these years on Pcom-ID hemorrhage, it is nec-



**Figure 1.** Illustration of the Pcom-ID classification. A. Anatomy of a normal Pcom-ID; the arrows represent the blood flow in the blood vessel. B. Type 1, direct ruptured Pcom-ID; the arrow points to the direct bleeding point. C. Type 2, Pcom-ID with a ruptured bleb; the arrow points to the ruptured bleb. D. Type 3, Pcom-ID evolving into a ruptured aneurysm.

essary to review the literature on Pcom-ID-induced hemorrhage and improve the understanding of Pcom-ID rupture hemorrhage.

#### Classification of hemorrhage type Pcom-ID

For asymptomatic Pcom-ID, the general view is that the Pcom-ID is a normal variation of cerebral blood vessels that lacks clinical significance [9, 10]. In this case, neither intervention nor follow-up is necessary. However, in some cases, due to the diagnosis and treatment of other intracranial vascular diseases by DSA, Pcom-ID is also discovered. In addition, after a certain period, rupture hemorrhage occurs from a Pcom-ID or rupture occurs from the aneurysm developing from a Pcom-ID, indicating that certain Pcom-ID cases are unstable [5, 6]. This fact allows us to place more attention on Pcom-ID and even to re-study this condition. For hemorrhage type Pcom-ID, no multi-case systematic research has been conducted, and the currently available data are only limited to case reports with scattered data, which do not adequately classify and summarize Pcom-ID

hemorrhage. This situation is not ideal for further elucidating this type of Pcom-ID. The only report mentioning Pcom-ID classification is a case study from Fischer in 2011. In this report, Pcom-ID is classified into three types including the no-change type, rupture hemorrhage type, and a type evolving to cyst aneurysms [1]. This classification method roughly categorizes Pcom-ID conditions but fails to consider bleb rupture of Pcom-ID; this type of case has also been reported. The bleb is similar to a blood blister such as an aneurysm and has its own characteristics, requiring a separate classification. Therefore, after a literature review and based on the author's understanding of Pcom-ID, hemorrhage-type Pcom-ID is better classified into the following three categories, which would provide good guidance for diagnosing and treating this type of Pcom-ID:

Type 1: The Pcom-ID exhibits direct rupture, causing SAH, with the rupture point on the blood-clot-blocked ID visible from craniotomy probing and with no aneurysmal change; Type 2: There is a bleb on the Pcom-ID. The bleb ruptures, causing SAH, and the bleb is similar to intracranial blood blisters such as aneurysms; Type 3: During follow up for a period after Pcom-ID, typical posterior communicating aneurysms occur and rupture. The details of this classification are shown in **Figure 1**. Concerning the method for this classification on Pcom-ID, in 1995, Endo *et al.* reviewed 32 cases of Pcom-ID without rupture. These cases all underwent craniotomy because of other diseases, and Pcom-ID was discovered at the same time. The classification of unruptured Pcom-ID was also mentioned in their study and was similar to that in the present study [11]. Therefore, it should be more reasonable for this study to evaluate the literature retrospectively according to the above three categories.

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**Table 1.** Type 1: Data summary of Pcom-ID with direct rupture

No	Year	Author	Age	Gender	Location	ID	Hemorrhage	Treatment	Prognosis	Confirmation	Combined with
1	1978	Archer [14]	55	M	Right side	Triangular (3 mm)	SAH in suprasellar cistern	Conservative treatment	Dead	Autopsy	Right middle cerebral artery aneurysm
2	1981	Yoshida [13]	58	F	Right side	Orbicular-ovate (2.5 mm)	SAH toward the right side	Perpendicularly clipping	Good	Craniotomy	None
3	1994	Ohyama [15]	57	M	Left side	Orbicular-ovate (2.8 mm)	Diffuse SAH	Conservative treatment	Dead	Autopsy	Intracranial arterial stenosis
4	2007	Coupe [16]	51	M	Left side	Triangular	Perimesencephalic SAH, hydrocephalus	Perpendicularly clipping	Good	Craniotomy	None
5	2010	Yu [8]	35	M	Right side	Orbicular-ovate (3 mm)	SAH twice	Coiling	Good	DSA and CT	None

**Table 2.** Type 2: Data summary of Pcom-ID with ruptured bleb

No	Year	Author	Age	Gender	Location	ID	Hemorrhage	Treatment	Prognosis	Confirmation	Combined with
1	2001	Kuwahara [23]	67	F	Right side	Bleb below the ID	Diffuse SAH	Clipping + retain ID	Good	Clipping and DSA	None
2	2009	Nashimoto [24]	63	M	Left side	Bled beside the ID	SAH more in left Sylvian fissure	Clipping + retain ID	Good	Clipping and DSA	None

**Table 3.** Type 3: Data summary of Pcom-ID evolving into ruptured aneurysm

No	Year	Author	Age	Gender	Observation time	Location	Aneurysm	Hemorrhage	Treatment	Prognosis	Confirmation	Combined with
1	1970	Stuntz [25]	38	M	9 years	Left side	Berry	SAH, Hydrocephalus, Temporal lobe and brain stem hematoma	Conservative treatment	Dead	Autopsy	Contralateral posterior communication artery ID
2	1983	Patrick [27]	43	F	9 years	Left side	Lobulated	SAH	Clipping	Good	Surgery and DSA	Contralateral posterior communication artery ID
3	1983	Itakura [26]	33	F	7 years	Right side	Lobulated	SAH	Clipping	Good	Surgery and DSA	Bifurcation of internal carotid artery
4	1998	Marshman [30]	54	F	5 years	Left side	Column	SAH, hydrocephalus	Clipping	Dead	Surgery and DSA	Middle cerebral artery aneurysm
5	1999	Su [28]	43	F	10 years	Right side	Berry	SAH, ventricular hemorrhage	Clipping	Good	Surgery and DSA	Contralateral posterior communication aneurysm
6	2002	Martins [5]	54	F	11 months	Right side	Berry	SAH	Clipping	Good	Surgery and DSA	Multiple aneurysms (including the left posterior communication artery)
7	2004	Cowan [31]	23	M	5 years	Left side	Berry	Bloody cerebrospinal fluid	Clipping	Good	Surgery and DSA	Apical aneurysm of basilar artery + Alagille Syndrome
8	2006	Takahashi [29]	54	F	10 years	Right side	Wide-necked	SAH	Clipping	Good	Surgery and DSA	Contralateral posterior communication aneurysm
9	2006	Radulovic [33]	45	F	11 years	Right side	Berry	SAH	Clipping	Good	Surgery and DSA	ID discovered after the first SAH
			48	F	10 years	Right side	Berry	SAH	Clipping	Good	Surgery and DSA	ID discovered after the first SAH
10	2014	Karekezi [6]	60	F	10 years	Right side	Berry	Diffuse SAH	Conservative treatment	Dead	DSA	Left posterior communication and right anterior choroidal artery aneurysms

### Type 1: Direct ruptured Pcom-ID

This type of Pcom-ID hemorrhage is the most difficult for diagnosis because a DSA examined after a SAH can only detect the Pcom-ID that is considered a normal anatomic variation, in which case, the SAH is not suggested to be caused by the Pcom-ID rupture. The SAH distribution from brain CT was found to be of some value in predicting the location of the bleeding. For example, Karttunen found that the location of the concentrated blood clots on CT mostly indicate the location of the aneurysm [12]. Due to the anatomic location of Pcom-ID on CT, the bleeding was observed more concentrated in the origin of the suprasellar cistern and sylvian fissure. This characteristic is similar to that of aneurysm hemorrhage. Even so, some perimesencephalic hemorrhages are similar to SAH caused by Pcom-ID rupture, which is difficult to distinguish. Therefore, it is speculated that many Pcom-ID rupture hemorrhage cases were misdiagnosed and were diagnosed as SAH negative instead. A retrospective analysis was performed on five cases with complete data between 1978 and 2010 (see **Table 1**) [8, 13-16]. In two cases, the diagnoses of SAH were confirmed from the autopsy, indicating the difficulty of diagnosing Pcom-ID rupture based on SAH [14, 15].

The analysis of five cases in **Table 1** revealed that the age of onset was from 35 years to 58 years, including four males and one female. The bleeding of SAH mainly concentrated around Pcom-ID, and one case was diagnosed with combined hydrocephalus. According to the clinical characteristics of these SAH cases, Pcom-ID rupture could not be diagnosed. Even so, except for the two dead cases, some clinical characteristics with significant implications were discovered after a careful analysis on the three remaining cases. For example, in the cases reported by Yoshida *et al.* in 1981, a DSA examination was performed after SAH, and only the right side of the Pcom-ID was observed. No intracranial aneurysms were found; consequently, conservative treatment was administered. Three weeks after DSA, a pseudoaneurysm was found on the Pcom-ID in time, which indirectly confirmed that the initial SAH was caused by Pcom-ID rupture hemorrhage. Therefore, craniotomy-clipping treatment was

conducted and yielded a satisfactory therapeutic effect. During the operation, the ruptured bleeding point from the Pcom-ID apex was also observed [13]. This case provided a suggestion to neurosurgeons that when DSA is performed after SAH and Pcom-ID is diagnosed, it is not recommended to simply diagnose it as angiography negative SAH. Instead, Pcom-ID rupture should also be considered. In addition, a timely follow-up check is critical because after a Pcom-ID ruptures, its morphology may change within a certain period. This change confirms that a Pcom-ID rupture hemorrhage existed in the first place and that a remedial treatment can be introduced.

In a case reported by Coupe *et al.* in 2007, the SAH characteristics were in line with the perimesencephalic hemorrhage from the CT image; however, the bleeding deviated toward the left side, and the DSA exam could only reveal Pcom-ID on the left side. The SAH was suggested to be the result of Pcom-ID rupture hemorrhage. Therefore, exploratory craniotomy surgery was performed, and the rupture point was observed on the apex of Pcom-ID during the operation, which confirms that the SAH was caused by Pcom-ID rupture [16]. The article by Coupe *et al.* did not mention how they confidently reached the decision for an exploratory craniotomy. Due to various reasons leading to this type of SAH, an exploratory craniotomy can only confirm whether the hemorrhage is caused by Pcom-ID, and the procedure might not be the best choice. We reported cases (Yu) in 2010, in which we discovered a left-sided Pcom-ID by DSA, after an SAH occurred. Although the SAH was accumulated on the periphery of the Pcom-ID, no diagnosis was confirmed. Therefore, the patient was placed under observation. A secondary SAH occurred during hospitalization, which suggested that the SAH was caused by Pcom-ID rupture. A satisfactory prognosis was achieved after a subsequent treatment [8]. Based on a retrospective analysis of the cases reported by Yoshida and Yu, we recommend that if Pcom-ID is observed during a DSA examination after SAH, the possibility of Pcom-ID rupture may be considered. If an exploratory examination cannot be performed, a timely DSA may also be chosen. If SAH occurs repeatedly, Pcom-ID rupture hemorrhage is more likely to be the case.

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From the review of the three cases in the literature regarding the treatment for Pcom-ID rupture, in 2 cases, a craniotomy-clipping operation was performed. Because the Pcom-ID appeared in a conical or triangular shape, to occlude the rupture point, clipping could only be performed perpendicularly to the posterior communicating artery. The posterior communicating artery was blocked by Pcom-ID clipping. Because, in both cases, the communicating artery was not in the embryonic form and was not well developed, postoperative complications were not observed. Therefore, perpendicular clipping of the posterior communicating artery on Pcom-ID is a good treatment option. The case by Yu was treated by combined interventional embolization. Microcatheters were placed into the Pcom-ID through both the internal carotid artery and the posterior communicating artery, which also achieved an ideal treatment effect. If Pcom-ID rupture hemorrhage can be determined, in addition to craniotomy-clipping operation and endovascular embolization, treatment using a flow diversion device can also be considered. In 2014, Kameda-Smith *et al.* reported a case using the Pipeline® flow guide device to treat an aneurysm near a Pcom-ID. The Pipeline® device covered the entire Pcom-ID, and the Pcom-ID disappeared by the 3-month follow-up examination. Therefore, it is possible to use this device in an effort to treat Pcom-ID. If Pcom-ID rupture hemorrhage is confirmed as the cause of SAH, interventional therapy is also a choice. Due to the relatively few cases within this type, their clinical features may not be representative. Although two patients' courses were complicated with other diseases, such as intracranial aneurysms at other locations and intracranial arterial stenosis, these cases may have no special clinical significance. No good method is currently available to determine whether an SAH hemorrhage is caused by Pcom-ID, but the author proposed some ideas that may help to determine whether a hemorrhage is caused by Pcom-ID. This idea is to use high-resolution MRI examinations. MRI can reveal permeability changes in the surrounding walls of a ruptured aneurysm. Therefore, if a Pcom-ID is ruptured, the ID walls may also demonstrate permeability changes. This observation is helpful for the diagnosis but must be confirmed by further research [17, 18].

### Type 2: Pcom-ID with ruptured bleb

A Pcom-ID can directly rupture, which has been discussed in detail in the previous section. In addition, there is a special type of Pcom-ID. Some small and thin blebs grow on the Pcom-ID surface, and these blebs can rupture, which constitutes Type 2 Pcom-ID. The blebs have an anatomical structure that differs from a normal berry aneurysm. From observations during surgery on cases of ruptured-bleb-type Pcom-ID, the bleb is similar in appearance to intracranial blood blisters such as aneurysms, with the loss of the normal intima and media; only the remaining thin layer of adventitia remained [11, 19, 20]. In 1995, Endo *et al.* reviewed 32 cases of Pcom-ID without rupture, including four cases with a reddish bleb in the lateral wall that protruded slightly in a posterolateral direction, which was suggested to be a preaneurysmal condition and to indicate a future event of rupture. This early change is only a small bleb on Pcom-ID, which is difficult to observe on DSA [11]. If the bleb is relatively large, it can be detected incidentally before it ruptures [21]. This situation is similar to the case reported by Pereira *et al.* in 2003, in which a 55-year-old female with headache was found on DSA to have a Pcom-ID bleb associated with a middle cerebral artery aneurysm. Clipping treatments were administered for both conditions [22].

When this type of bleb ruptures, local pseudoaneurysms often form, and a DSA examination can reveal a bulge on the Pcom-ID. We have reviewed the literature on Type 2 Pcom-ID with ruptured blebs. Only two cases with complete clinical data are summarized in **Table 2**. One of the cases was reported by Kuwahara *et al.* in 2001, in which a 67-year-old female suffered from permissive SAH. Her DSA displayed Pcom-ID on the right side and a bleb originating from the Pcom-ID. Considering the possibility of bleb rupture, bleb clipping was performed while retaining the funnel and distal posterior communication artery aneurysm, which achieved a satisfactory result [23]. A similar case was reported by Nashimoto *et al.* in 2009, in which a 63-year-old male patient suffered from SAH with relatively heavy left-lateral-fissure bleeding. DSA revealed a 1.5-mm bulge at the posterolateral side of the left Pcom-ID. The Pcom-ID was retained by clipping surgery, and the outcome was remarkable [24]. Therefore, after

the bleb on the Pcom-ID ruptures and causes SAH, a DSA examination often reveals the ruptured bleb on the Pcom-ID, which is clinically distinctive. Based on the literature review, we found that Type 2 Pcom-ID is relatively rare, lacking relevant information. Therefore, the incidence and time for the occurrence of the bleb growth on Pcom-ID has not been clarified.

### Type 3: Pcom-ID evolving to ruptured aneurysm

If the bleb Pcom-ID does not rupture, its continued growth may develop into a posterior communication artery aneurysm, that is, Type 3 Pcom-ID. The rupture of a Pcom-ID after it evolves into an aneurysm has been reported more frequently. Although in previous case reports, a retrospective study of the previous literature was included, these reviews were mostly limited to the particular case-report-related content, which was not very systematic. In this study, we collected 11 cases of Pcom-ID from 1970 to 2014 with complete data from each case of ruptured aneurysms and performed a retrospective analysis on these clinical data, as summarized in **Table 3**. The age of the 11 patients ranged from 23 to 60 years, with an average age of 45 years. There were more women (nine cases) than men (two cases). The period between the identification of Pcom-ID to the evolution into aneurysms was found to be 11 months to 10 years, with an average of 7.8 years. The most distinctive feature was that seven out of the 11 patients suffering from Pcom-ID with evolution into aneurysms were diagnosed with a contralateral Pcom-ID or posterior communicating artery aneurysm. Therefore, bilateral Pcom-ID and Pcom-ID with contralateral posterior communicating artery aneurysms are suggested to develop into aneurysms more easily [5, 6, 25-29]. In addition, five out of 11 cases were also diagnosed with aneurysms in other positions such as middle cerebral artery aneurysms, internal carotid artery bifurcation aneurysms, basilar artery apex aneurysms, and choroid anterior aneurysms [5, 26, 30, 31]. Therefore, it is obvious that Pcom-ID combined with multiple aneurysms poses a higher risk of evolving into an aneurysm [32].

Except for the cases with combined multiple aneurysms, Type 3 may also be a result of a

previous history of SAH. For example, Radulovic *et al.* reported two cases in 2006 in which DSA was performed because of SAH; a Pcom-ID was discovered, which was 10 and 11 years prior to the occurrence of the posterior communicating artery aneurysm [33]. Therefore, it is suggested that a past history of SAH may be a risk factor for the evolution of Pcom-ID into a posterior communicating artery aneurysm, but more research is required to confirm this hypothesis. In addition, a case reported by Coan in 2004 involved Alagille syndrome combined with an apical aneurysm of the basilar artery [31]. Alagille syndrome is a rare autosomal dominant disorder that affects multiple systems and organs. Cases combined with multiple intracranial aneurysms have also been reported [34]. However, more evidence is necessary to support the suggestion that Alagille syndrome makes it easier for a Pcom-ID to evolve into an aneurysm.

In a review of the cases in **Table 3**, bilateral Pcom-ID was often found to evolve into hemilateral posterior communicating artery aneurysms, and cases of bilateral Pcom-ID with evolution to bilateral posterior communicating artery aneurysm were rare. After a literature review, we found that only Itakura *et al.* reported a case of bilateral Pcom-ID combined with an internal carotid artery bifurcation aneurysm, found by DSA. Seven years after the administered internal carotid artery bifurcation aneurysm clipping treatment, both sides of the Pcom-ID evolved into aneurysms. One side ruptured, and a clipping treatment was administered. Another unruptured aneurysm was left under observation [26]. The treatment for Type 3 Pcom-ID with evolution into ruptured communicating artery aneurysms is hardly different from the conventional treatment for ruptured communicating artery aneurysms. The literature in **Table 3** reveals that craniotomy and clipping operations achieve a good therapeutic effect. Therefore, after summarizing Type 3 Pcom-ID evolving into ruptured communicating artery aneurysms, we found that when Pcom-ID is combined with contralateral posterior communicating artery aneurysms or other intracranial aneurysms, precautions against Pcom-ID evolution into aneurysms may be necessary. For Pcom-ID that has evolved into a ruptured posterior communicating artery aneurysm, active treatment should be engaged, and a

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subsequent good prognosis can often be achieved.

## Summary

According to the literature review on Pcom-ID rupture, this condition can reasonably be classified into three types, as follows: Type 1, direct ruptured Pcom-ID; Type 2, bleb ruptured Pcom-ID and Type 3, Pcom-ID evolving into a ruptured aneurysm. This classification provides meaningful guidance for treating ruptured Pcom-ID. For Type 1 cases, the most difficult thing is to confirm the diagnosis. Based on the literature review, we discovered that when an SAH is concentrated around a Pcom-ID, the possibility of Pcom-ID hemorrhage should be considered. If the diagnosis cannot be established, a timely follow up DSA may deliver meaningful evidence for Pcom-ID rupture. For Type 2 cases, when the SAH is concentrated around the Pcom-ID, if DSA reveals blebs on the Pcom-ID, the possibility of SAH caused by a ruptured bleb should be considered. For Type 3 cases, the treatment is no different from the conventional treatment for a communicating artery aneurysm. However, when Pcom-ID is combined with a contralateral posterior communicating artery aneurysm or aneurysms at other locations, the possibility of Pcom-ID evolving into aneurysms may be considered, and it is necessary to conduct a radiographic follow-up.

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## Disclosure of conflict of interest

None.

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