Endovascular Occlusion of the Posterior Cerebral Artery in the Treatment of P2 Ruptured Aneurysms

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Summary

Four cases of posterior cerebral artery (PCA) aneurysms are described. The aneurysms were located at the P2 segment of PCA. All cases presented with a subarachnoid hemorrhage (SAH). Endovascular treatment was performed, with occlusion of the aneurysm and parent vessel, using platinum coils. Two patients developed a homonymus lateral hemianopia after treatment.

Introduction

Aneurysms of the posterior cerebral artery (PCA) are quite rare, accounting for about 1% of all intracranial aneurysms[4]. Among PCA aneurysms, P2 segment aneurysms develop distal to the junction of the posterior communicating artery with the posterior cerebral artery and are located at the level of the posterior part of the midbrain, within the ambient cistern; they may be saccular, fusiform or dissecting.

Endovascular or surgical treatment consists of selective occlusion of the aneurysm, when possible, or parent artery occlusion. There are only a few reports about endovascular treatment of PCA aneurysms[1,2,3,5,6,7].

Rewiewing our series of aneurysms presenting with SAH and treated by endovascular approach, we observed four P2 segment aneurysms, one saccular and three fusiform. Treatment with coil occlusion of the aneurysm and parent artery, results, complications, and follow-up studies are reported.

Material and Methods

Clinical Material and Diagnostic Examinations

Between 2001 and 2006 four patients were diagnosed having an acutely ruptured aneurysm of the PCA. The patients' age ranged between 19 and 54 years. Two patients were females and two were males. Upon admission, all four patients were in Hunt and Hess grade 1, and in GCS 15 to 13. Neurological examination did not show any visual field deficit. In all patients the CT findings demonstrated a Fisher grade 1 SAH.

Angiography demonstrated that three fusiform and one saccular aneurysm had been the cause of the hemorrhage. Three aneurysms were located at the P1-P2 junction, while one aneurysm was located at the P2-P3 junction of the PCA. Three aneurysms were large, while one was small.

Endovascular treatment of the lesions was performed in all cases using bare platinum coils (Table 1).

Endovascular Treatment and Follow-up Studies

All embolization procedures were performed via transfemoral approach, under general anesthesia and systemic heparinization. In all

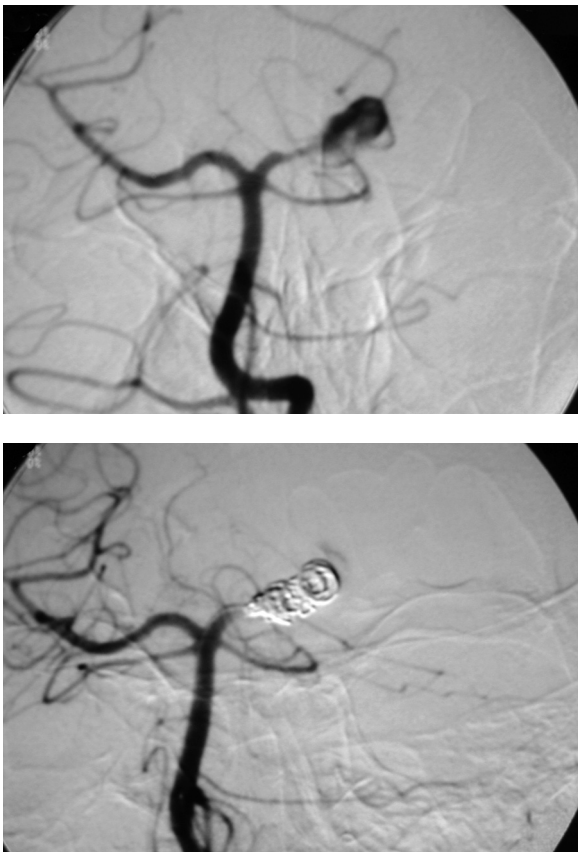

Figure 1 A) Angiogram showing a large fusiform aneurysm at the P2 segment of the left posterior cerebral artery. B) Aneurysm and parent vessel were occluded by depositing 9 GDC coils. The aneurysm is now excluded from the brain circulation.

cases a microcatheter was positioned in the aneurysm and platinum coils were delivered until aneurysm and parent vessel occlusion were achieved.

In one case (case n° 1) a balloon test occlusion of the PCA (proximal to the aneurysm) was performed prior to the induction of general anesthesia. In this case, clinical examination during the test occlusion showed no deficits. In the remaining three cases no test occlusion was performed.

Results

Complete occlusion of the aneurysm and parent vessel was achieved in all four cases (figure 1A,B). No technical complications were encountered.

The immediate post treatment clinical examinations showed no clinical sequelae in two cases (Cases 1 and 4). In the remaining two cases (Cases 2 and 3), however, a homonymous lateral hemianopia was detected which persisted at the follow-up studies (Table 1). Long term clinical follow-up studies in these four cases showed no episode of rebleeding.

Follow-up studies were performed at one month, six months, and 12 months. The one and six month studies consisted of clinical and MRA examinations. Digital subtraction angiography was performed at the 12 month follow-up in three cases. In the remaining case

Table 1 **Patient's characteristics, treatment type, and follow-up studies**

Patient no. Sex, Age, (y)	Aneurysm Type, Side and Size	Presenting Symptom	Test	Treatment Type	Clinical Outcome and Follow–up findings
1/M/19 2001	Fusiform P2 L	SAH HH1 GCS 15 Large	Angiography + balloon occlusion	GDC	Excellent, no deficit; at 1-y Angiography, no recanalization
2/F/45 2004	Fusiform P2 L Large	SAH HH1 GCS 14	angiography	GDC	right lateral hemianopsia, at 1-y Angiography, no recanalization
3/F/54 2006	Fusiform P2-P3 L Large	SAH HH1 GCS 13	angiography	GDC	right lateral hemianopsia at 9-mo Angiography, no recanalization
4/M/41 2006	Berry P2 L Small	SAH HH1 GCS 13	angiography	GDC+ Micrus coils	Excellent, no deficit at 8-mo Angiography, no aneurysm recanalization pca recanalization

(case n° 4) 30 day, three month and 12 month angiographic follow-ups were performed, which showed a recanalization of the parent artery and persistent occlusion of the aneurysm (figure 2A-C).

Discussion

The most common clinical presentation of PCA aneurysms is subarachnoid hemorrhage SAH [3]. Clinical presentation of P2 fusiform aneurysms with SAH, however, is rare [6,7]. Drake and Amacher [4] reported that the most common site of origin of PCA aneurysms is at the first major branch, beyond the junction with the PCoA. Aneurysms, however, can occur at any site along the course of PCA. PCA aneurysms are classified as those of the P1 segment, P1-PCoA junction, P2, P3, or P4 segments.

As to the largest published series on the endovascular treatment of PCA aneurysms, Hallacq et Al. reported ten cases of P2 segment aneurysms, of which only two with clinical history of SAH [6], while Van Rooij et Al [7] reported 22 PCA aneurysms, of which only two were non-saccular and presented with a clinical history of SAH.

Endovascular approach for the treatment of P2 aneurysms is an accepted form of treatment. Previous reports described the occlusion of the aneurysm and the parent artery as the most efficacious kind of endovascular treatment [1,2,3,5,6,7].

In our series we report four P2 aneurysms (three fusiform and one saccular) with a clinical history of SAH. No symptoms were reported in our cases before the subaracnoid hemorrage. All aneurysms were occluded with coils including the P2 main trunk. After the first patient (Case 1, vide supra), we did not consider preoperative test occlusion in the remaining three patients. This was because other series reported a well tolerated PCA occlusion by virtue of the rich collateral leptomeningeal arterial network [6,7]. We did not consider the use of intracranial stents because all cases had a clinical history of recent SAH. In addition, there have been no reports to date on the use of stents in the P2 segment of PCA.

It is the author's opinion that this is suggestive for the two following reasons:

1) Post treatment neurological deterioration was observed in two out of four cases. Previous reports, on the contrary, indicated that the occlusion of the PCA does not lead to neurologi-

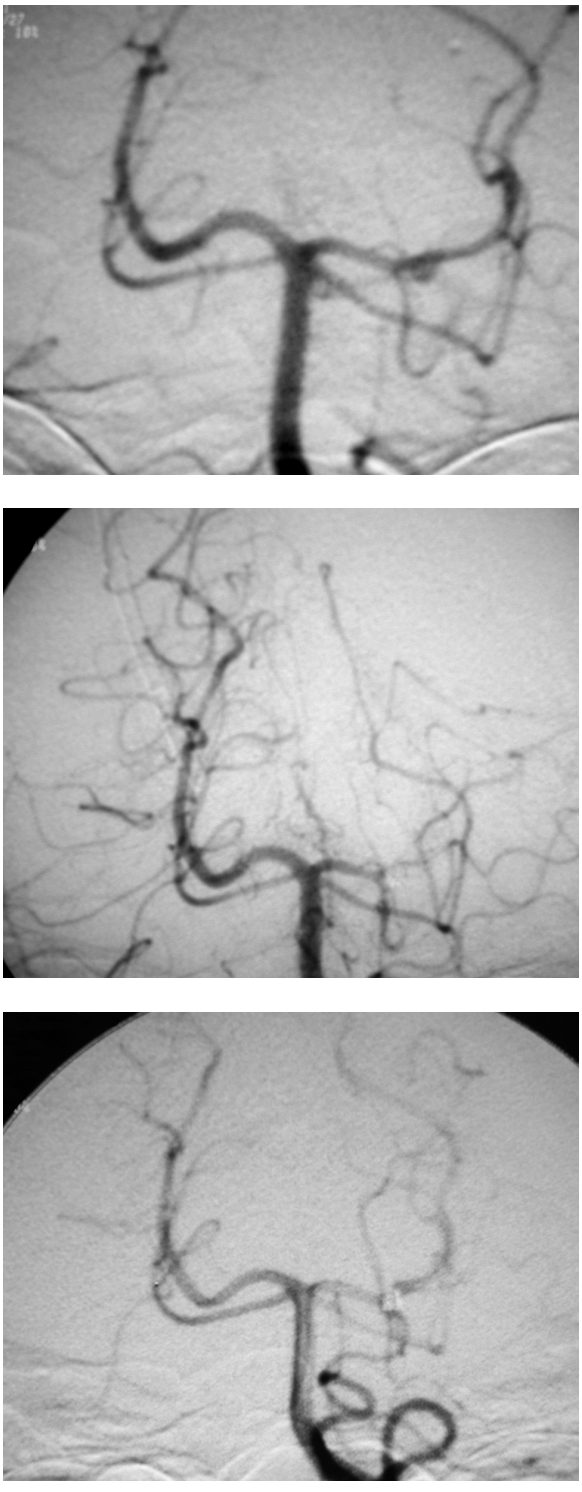

Figure 2 A) Angiogram showing a small, saccular aneurysm arising from the P2 segment of the left posterior cerebral artery (arrow). B) Aneurysm and parent vessel were occluded by depositing 3 platinum coils. C) This 30-day follow-up angiogram shows persistent occlusion of the aneurysm and repermeabilization of the entire posterior cerebral artery vascular tree (arrows) (see text).

cal deterioration [6,7]; this represents a discrepancy with our clinical results. Nevertheless, in a recent report, Andreou et Al [1] described six cases of PCA aneurysms treated with parent vessel occlusion: in two of these six cases, post-treatment campimetric deficits were observed. Furthermore, Ciceri et Al [3] reported that in seven similar cases two patients were affected by post-treatment campimetric deficits. Moreover, Eckard et Al [5] reported post-treatment campimetric deficits in one out of three cases treated with PCA occlusion.

2) In one of our four cases (case n° 4) treated with aneurysm and PCA coil occlusion the artery resulted reopened at the 30 day follow-up angiogram. Occlusion of the aneurysm and patency of the parent vessel were confirmed at the three-month as well as at the 12-month follow-up angiograms. Recanalization of the parent vessel could possibly be due in this case to a modification of the structure of the thrombus with "de novo" re-endothelialization of channels within the thrombus, with consequent revascularization of the artery. Persistent aneurysm occlusion will have to be confirmed in this case with a longer term follow-up angiogram.

Conclusions

Our results confirm that parent vessel occlusion is a viable therapeutic option for distal posterior cerebral aneurysms. However, a homonymous lateral hemianopia may develop after treatment. All therapeutic options should be considered on a case-by-case basis.

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EDITORIAL COMMENT

Aneurysms of the P2 segment of the posterior cerebral artery are typically dissecting aneurysms no matter whether their aspect is "saccular" or "fusiform". Intradural spontaneous dissections (SDs) are different from cervical or extradural SDs, although they may be associated with the same underlying diseases.

On histopathology, the elastic lamina is destroyed and only some elastic fragments remain. The dissection typically appears between the media and the adventitia, however, the internal elastic lamina can also be disrupted. At the site of rupture, neovascularization and subadventitial dissecting hemorrhage are present. A disruption of the entire arterial wall can also be present. Depending on how many layers of the vessel wall are affected, an aneurysm might develop; alternatively, the blood clot within the vessel wall can protrude against the vessel lumen, leading to narrowing of the vessel with a pseudostenosis. Therefore neuroimaging features may vary: the most frequent angiographic demonstration is regular or irregular fusiform dilatation. However, ruptured dissections may not have an obvious "aneurysmal" dilatation, and stenotic vessel segments might also lead to hemorrhagic events.

Spontaneous hemorrhagic intracranial dissection has an unfavorable prognosis and a high rebleeding rate. Depending on the cited source, 1-10% of all intracranial non-traumatic SAH are caused by ruptured intracranial dissection, and in young patients, this rate may rise up to 5-20%. Treatment should be targeted at excluding the damaged vessel wall segment from the circulation, that for the P2 location can be best reached endovascularly by sacrificing the diseased vessel. This vessel sacrifice is clinically in most instances well tolerated because of the rich anastomotic vascular network of the PCA: Since embryologically, the PCA is a diencephalomesencephalic artery, which gathers its telencephalic supply from the distal annexation of the anterior choroidal artery territory, one possible collateral supply is present between the posterior and anterior choroidal arteries that may actually form a rete close to the lateral geniculate body. In addition, splenial branches of both anterior cerebral arteries with both posterior cerebral arteries also contribute to the collateral vascular network. Thirdly, leptomeningeal participation from the ipsilateral anterior and middle cerebral artery may provide additional collateral supply.

Whereas the method of treatment, is - at this point in time and in most institutions - not controversially discussed, timing of treatment is, since two points have to be taken into account: the therapeutic result and the tolerance to the vessel sacrifice is obviously dependant on the collateral supply. Probably, chronic dissections allow for better collateral connections as described above to occur in time, while acute dissections may not have induced a sufficient leptomeningeal network of arteries to take over. Although this argument would favour an conservative approach once a dissection of the P2 segment is found, a second, clinical more important argument has to be discussed: acutely ruptured dissections are unstable and have a tendency to rebleed with the rebleeding rate being reported to be as high as 70%. The mortality rate of these re-bleeds is as high as 50%. As a rule, the closer the

time after initial hemorrhage, the higher the rebleeding risk in the acute phase. 70% of rebleedings occur within the first 24 hours after initial SAH and 80% of rebleedings occur within the first week after SAH. As time elapses, the rebleeding rate decreases considerably after the first week following initial hemorrhage. A ruptured dissecting aneurysm enters into a healing stage approximately one month after the initial SAH, therefore only 10% of rebleeding occurs more than one month after the initial hemorrhage. The histopathology of the healing response following dissection reveals covering of the entire area of the disrupted arterial wall with neointima. This repair occurs from the disrupted ends of the media toward the ruptured portion.

The healing mechanism may be delayed under several conditions such as aneurysms with extensive defects of the aneurysmal wall in the ruptured portion (i.e. large aneurysms), aneurysms with abundant thrombus in the ruptured portion (since neointima may appear in accordance with retraction of the thrombus), or aneurysms in which the media is completely separated from the adventitia.

Since, for a given patient, the "chronicity" of the dissection can not be easily assessed, balloon occlusion may be a viable option to test whether vessel sacrifice will be tolerated. However, since it is our opinion, that - once a dissecting aneurysm has become symptomatic with hemorrhage - therapy is indicated, a test balloon occlusion only makes sense if a surgical bypass to the distal PCA can be performed.

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