Bilobed Wide Neck Posterior Cerebral Artery Aneurysm Associated with Fusiform Basilar Aneurysm, Subarachnoid Hemorrhage and Chronic Renal Failure

A Case Report*

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Key words: brain, aneurysms, SAH, renal failure

Summary

A 56 year-old woman presented with a ruptured bilobed wide neck aneurysm of the P2 segment of the PCA, atherosclerotic fusiform basilar artery aneurysm, subarachnoid bleeding with negative CT scan and chronic renal failure. She was managed by a cooperative approach involving neurosurgeons, neuroradiologist, neurointensivist, emergency room physicians, nurses and technicians.

She underwent operation by proximal clipping for the aneurysm of the PCA. Postoperative neurological deficits include homonymous hemianopsia and ipsilateral third nerve palsy. The operation was performed through a subtemporal approach. At surgery, the aneurysm was located in the distal of the P2 segment of PCA, bilobed up and down, no definitive neck with small distal branches, and was treated by proximal clipping of the PCA aneurysm. The fusiform basilar artery aneurysm was severely atherosclerotic and left untouched.

This is a rare case which required a high index of suspicion to detect subarachnoid bleeding from ruptured posterior fossa aneurysm,

accurate prediction of the site of bleeding and the location of aneurysm location by conventional angiogram, MRI and MRA, and careful planned surgical strategy with the right approach for the P2 segment of the PCA aneurysm, complicated post operative care with airway management, triple H therapy, nutrition, additional measures and multiple hemodialysis.

Introduction

A case with ruptured bilobed wide neck aneurysm of the P2 segment of the right posterior cerebral artery (PCA), atherosclerotic fusiform basilar aneurysm, SAH with negative CT scan and chronic renal failure is presented. This is a rare case which required a high index of suspicion to detect subarachnoid bleeding from ruptured posterior fossa aneurysm, accurate prediction of the site of bleeding and the location of aneurysm location, and careful planned surgical strategy with the right approach for the P2 segment of the PCA aneurysm, complicated post operative care with airway management, triple H therapy, nu-

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Figure 1 CT scan on the third day. A, B at the level of pons show a fusiform basilar aneurysm with wall calcification. C, D. at the level of mesencephalon show right perimesencephalic hemorrhage. The temporal horns are mildly dilated due to hydrocephalus.

trition and multiple hemodialysis. In this case report, the discussion of the post operative care is omitted.

Case Report

A 56-year-old woman with a history of hypertension on medication and chronic renal

Figure 2 A, B) Pre-operative vertebral angiogram shows a fusiform basilar aneurysm and multilobed right PCA aneurysm. C, D) Post operative vertebral angiogram shows a complete occlusion of the right PCA distal to proximal clipping. The fusiform basilar aneurysm was left untouched.

failure on three times weekly hemodialysis started nine months ago, presented with two weeks progressively worsening headache. Headache was aggravating in the last week and associated with neckache and stiffness, vomiting and fever. Three days before admission, she complained of abrupt generalized headache, mostly occipital, like "pulling" over the back of the neck. CT scan on the day of admission revealed a fusiform basilar aneurysm,

without findings of SAH. A fusiform basilar aneurysm was shown on four vessel catheter angiography, as was a multilobed wide neck aneurysm at P1-P2 segment of the right PCA. MRI and MR angiography on the second day revealed a fusiform aneurysm of the basilar artery compressing the right pons, suspicious findings of SAH in the right perimesencephalic cistern and some hemorrhage in the dependent posterior horn of the lateral ventricles. In

3D MIP (maximum intensity projection) reconstruction, the PCA aneurysm was not visible. The repeat CT scan on the third day confirmed the right perimesencephalic cistern and intraventricular hemorrhage (figure 1A, B, C, D). On the fourth day, right vertebral catheter angiography was repeated, and again showed the multilobed wide neck posterior cerebral artery aneurysm, reported at P1-P2 segment, 5x8 mm in diameter (figure 2A, B). The radiologist assumed that the SAH was caused by the ruptured aneurysm of the P1-P2 segment of the right posterior cerebral artery, and for this reason an operation was performed. At surgery, the ruptured aneurysm was located in the distal of the P2 segment of the right PCA, bilobed up and down, no definitive neck with small distal branches, and was treated by proximal clipping of the aneurysm. The fusiform basilar artery aneurysm was severely atherosclerotic and left untouched. Post operative CT scan showed right epidural hematoma at the site craniotomy and right occipital lobe infarction. Post operative angiogram (figure 2C, D) showed a complete occlusion of the right PCA distal to proximal clipping of the aneurysm. Post operatively she was treated in the Neuro Intensive Care Unit by airway management, triple H therapy, nutrition, additional measures and multiple hemodialysis. Right third nerve palsy and left homonymous hemianopsia were evident post-operatively and persisted. The patient was transferred to a High Dependency Unit two weeks later.

Discussion

The etiology and pathogenesis of intracranial aneurysms are not congenital and clearly multifactorial, with genetic factors, acquired and hemodynamic factors playing an increasingly recognized role. Intracranial aneurysms have been associated with numerous heritable connective tissue disorders, which account for at least 5% of cases^{1,2,3}. Of these disorders, the most important are Ehlers-Danlos syndrome Type IV, Marfan's syndrome, neurofibromatosis Type 1, and autosomal dominant polycystic kidney disease. The association with intracranial aneurysms, however, has been firmly established only for polycystic kidney disease³. There was a significant association between the presence of multiple aneurysms and hyperten-

sion, cigarette smoking, family history of cerebrovascular disease, female sex and postmenopausal state in female patients⁴. Multiple intracranial aneurysms have been detected in up to one third of patients with cerebral aneurysms. Aneurysms of the middle cerebral, internal carotid and pericallosal arteries are more frequently associated with multiple intracranial aneurysms, only 6% of the aneurysms were in the vertebrobasilar tree⁵. The multiple intracranial aneurysms in this patient could be related to hypertension, atherosclerosis, female sex and postmenopausal state, but there was no evidence of polycystic kidney disease.

The presentation of this patient with abrupt generalized headache for three days before admission, negative CT scan for SAH on the day of admission and perimesencephalic hemorrhage on the third day of CT scan merit some discussion. The most common presentation of an intracranial aneurysm is SAH, which is the first manifestation in 90% of cases. In addition, most (>80%) nontraumatic SAH are caused by aneurysm rupture. Patients typically complain of 'the worst headache of my life', which may or may not be associated with nausea and vomiting. Patients with a severe SAH rapidly deteriorate to a comatose state or have a respiratory arrest from extremely high intracranial pressure which induces a profound reduction in cerebral perfusion pressure. Of note, approximately 40-50% of patients have been reported to experience warning signs caused by a minor leak or expansion of the aneurysm prior to rupture. These warning signs are often followed by aneurysm rupture in 10-20 days⁸. SAH typically is confirmed by CT scan. When CT scan is done within hours after the event, the sensitivity in aneurysmal SAH is very high and may approach 95 percent. In two to five to percent of patients, subarachnoid blood has completely "washed out" on CT scans within 24 hours. The proportion of patients with SAH and negative CT scans increases in the following week to approximately ten percent on day three and 50 percent on day seven. In many patients, no subarachnoid blood is shown on CT scans after ten days, but intraventricular and intraparenchymal hematomas take more time to resolve. Reportedly negative CT scans in patients in whom SAH is strongly suspected must be carefully scrutinized for subtle signs of subarachnoid

blood. When results of CT scanning are negative, a lumbar puncture should be performed. Blood-stained cerebrospinal fluid (CSF) may have a traumatic origin. The number of patients with a negative CT scan but blood pigments in the CSF after SAH is very small, and the probability that bloody CSF represents a traumatic puncture is higher. Traumatic lumbar puncture and true SAH can be differentiated by centrifuging CSF and inspecting the supernatant for xanthochromia¹⁸. Considering hemorrhage in the perimesencephalic cistern, a benign form of SAH was reported in which bleeding was confined to the cisterns around the midbrain without evidence of an aneurysm in the posterior circulation. This CT scan feature was termed "perimesencephalic hemorrhage". The CT scan characteristic of a perimesencephalic hemorrhage includes blood clots in the cisterns surrounding the midbrain, often with the center of the bleeding immediately anterior to the mid-brain. Typically, the blood does not extend to the lateral sylvian fissures or to the anterior interhemispheric fissure. Some extension to the basal part of the sylvian fissure is possible when CT scanning is performed very early. Intraventricular hemorrhage is absent except for some sedimentation in the dependent posterior horns. Rupture of a dilated vein in the prepontine cistern is a possible explanation. Perimesencephalic hemorrhage may closely mimic a ruptured basilar artery aneurysm, and therefore a cerebral angiogram is warranted. Unusual aneurysms, such as a P2 aneurysm, may manifest as a perimesencephalic SAH¹⁸.

PCA aneurysms are unusual, accounting for approximately 1% of all intracranial aneurysms^{6,7,8}. Zeal and Rhoton defined the P2 region of the PCA as a segment spanning from the posterior communicating artery (PCoA) to the point at which the PCA enters the quadrigeminal cistern. The P2 segment was subdivided into anterior (P2a) and posterior (P2p) portions, divided by the posterior aspect of the cerebral peduncle⁹. Most PCA aneurysms arise from P2-related sites, including the P1-P2 junction, P2a, and P2p^{6,10,11}. The usual saccular type accounts for about 80% of PCA aneurysms, with fusiform and giant aneurysms accounting for about 10% each¹². The PCoA plus P1 is the caudal division of the internal carotid artery (ICA) with its diencephalomesencephalic territory (which includes the cere-

bellum). It extends to the upper basilar artery postsegmental portion (distal to the trigeminal remnant) and influences the anterior superior cerebellar artery (ASCA) pattern. The P2-P3-P4 are the true PCA system (mainly telencephalic, partially choroidal and incidentally diencephalic). From embryologic considerations, although definition of PCA segments is unsatisfactory and incorrect, it is useful for practical purposes³. There has also been some confusion, and discrepancies exist in the neuroanatomic, microsurgical and neuroradiological nomenclature used to define specific segments of the PCA, especially regarding the location of the P2-P3 junction. Originally, the P2-P3 junction was described as occurring at the take-off of the major inferior posterior temporal trunk. In Zeal and Rhoton's definition, it was described as occurring at the entrance point of the PCA into the quadrigeminal cistern. As a result of the extensive variations that occur in the course and branching pattern of the PCA, these two definitions may not coincide in many cases. Therefore, the surgical approach to the P2 segment and to the P2-P3 junction of the PCA ought not to be based on specific guidelines, but rather on detailed study of the preoperative cerebral angiogram and, if possible, magnetic resonance imaging in each individual case to identify the specific location of the aneurysm and its relation to the midbrain, cisterns, vessels, and mesial temporal lobe structures¹⁴.

The angiography of this case provided exact details on the site, size and morphology as multilobed wide neck PCA aneurysm as well as the fusiform basilar aneurysm. The detection of multilobed aneurysm in this case with multiple aneurysm and perimesencephalic bleeding is very important to define the site of rupture^{15,16}. However, the angiography failed to give information of the exact segment of the PCA aneurysm and its relationship with the mesencephalon. The aneurysm was reported at the P1-P2 segment of the PCA, but at surgery it was located at the distal P2 segment which needed further dissection. Although MRI and MRA were done, the MRA failed to show the PCA aneurysm. The PCA aneurysm was not visible in MRA, because the 3D TOF MRA technique has inherent limitations in the depiction of intracranial aneurysms. The perception of aneurysms accompanied by spin saturation

tion resulting from slow flow or turbulent flow is severely limited¹⁶. Alternative MRA methods, such as flow independent or contrast enhanced MRA, which localizes vessels due to the relaxation characteristics of blood rather than to its flow properties, may be necessary in this case¹⁷.

The P2 segment of PCA aneurysms may be difficult to reach during surgical procedures. The potential for morbidity and mortality is considerable with surgery, because the perforating branches are closely related to the cerebral peduncle¹⁴. In the surgical series of Terasaka et al¹¹, four of 14 patients had a disability after surgery. Compared with surgical clip placement, endovascular treatment may have notable advantages in this subset of P2 segment aneurysms. Endovascular treatment is not associated with manipulation of the surrounding tissues. Therefore, the risks of brain infarction due to retraction or removal are theoretically reduced. If it is possible to treat an aneurysm in this location with proximal occlusion of the PCA, then such an approach may be preferable to an open surgical technique^{6,14}. In Hallack et al's retrospective review of a ten year series⁶, endovascular occlusion of the PCA for the treatment of P2 segment aneurysms appears to be safe as no transient or permanent complication related to the procedure was noted. The potential collateral supply and hemodynamic balance between the anterior and posterior choroidal arteries, pericallosal vessels, and anterior and middle cerebral vessels to the distal PCA made P2 occlusion safe, because the aneurysm occurred after the thalamoperforating vessels arose from the P1 segment. In the endovascular series of Ciceri et al⁷, fourteen (66%) of the 21 aneurysms were successfully treated with preservation of the parent artery. In the remaining seven (33%), the parent artery was permanently occluded. The overall complication rate in this series was 15%, with a permanent morbidity rate of 10% and a 0% mortality rate. They conclude that endovascular treatment of these aneurysms is relatively safe and effective with low mortality and morbidity.

Previously reported cases of PCA aneurysms treated with trapping or proximal clipping are considered to be relatively safe especially in children and young adults because of the remarkable collateral circulation of the PCA. However, there are cases in which persi-

stent neurological deficits occur postoperatively. Neurological deficits such as hemiparesis, hemihypesthesia or hemianopsia during the postoperative period, even though they are transient, suggest that ischemic events do take place in the area perfused by the perforating arteries from the PCA. Batjer¹⁹ stated that to date no effective angiographic or neurophysiological criteria are available to assist the surgeon in preoperatively or intraoperatively predicting individual tolerance. Whereas young age and brisk retrograde bleeding after proximal clipping provide some degree of security, the seriousness of the potential deficits and the diminished autoregulatory capacity after SAH make routine sacrifice of this vessel an unsettling practice. Although the distal posterior cerebral territory can be well supplied in many patients by the middle cerebral and the anterior cerebral watershed, the critical branches and perforators from the proximal P-1 and P-2 segments that may inadvertently be included in the trapped segment have essentially no collateral supply. The posterior thalamoperforates, thalamogeniculates, and peduncular perforating vessels must be clearly seen before decisions can be made about final clip placement. Various possible revascularization techniques can be beneficial in the treatment of unclipable aneurysms in this region^{11,12,19}.

There are few absolute contraindications to endovascular treatment: the presence of fresh clot within the aneurysm sac and fusiform shape (i.e. no neck), are probably the only absolute contra-indications²⁰. Because of the severe atherosclerosis of the vertebral arteries and fusiform aneurysm of the basilar artery in this patient and the chronic renal failure and to make sure that the SAH was caused by ruptured aneurysm of the P2 segment of the right posterior cerebral artery, an open surgical technique was chosen. Although proximal clipping of the P2 aneurysm of the PCA is considered to be relatively safe because of the remarkable collateral circulation of the PCA, this patient is one of the few case in which persistent homonymous hemianopia due to occipital lobe infarction occurred postoperatively. In this difficult and challenging surgery, the right third nerve palsy without hemiparesis or hemihypesthesia that occurred postoperatively, seemed to be an acceptable morbidity.

Fusiform aneurysms may be caused by disor-

ders of collagen and elastin, infections, dissection, and, very rarely, neoplastic invasion of the arterial wall. Drake et al²¹ reviewed 120 surgically treated patients with giant fusiform intracranial aneurysms. Twenty-five aneurysms were located in the anterior and 95 in the posterior circulation. Six patients had atherosclerosis and only three others had a known arteriopathy. The remaining 111 patients presented with aneurysms resulting from an unknown arterial disorder; these patients were much younger than those harboring atherosclerotic aneurysms. Mass effect occurred in only 50% of cases and hemorrhage in 20%²¹. Neurosurgeons consider dissecting aneurysms and fusiform aneurysms to be different entities and distinguishing between them is very important to determine their course of treatment and prognosis. Differential diagnosis, however, is very difficult. Turbulent flow, laminar flow, or intraarterial thrombus in fusiform aneurysms may mimic the findings for dissecting aneurysms on angiographic or magnetic resonance images. Angiographic findings, such as pearl and string signs and retention of contrast medium, or magnetic resonance imaging findings, such as an intimal flap and a double lumen, do not necessarily indicate a dissecting aneurysm. Angiographically determined fusiform aneurysms displaying such characteristics, which have been clinically diagnosed as dissecting aneurysms, have later been diagnosed as fusiform aneurysms during autopsy²². Hunterian proximal occlusion or trapping were dominant among the treatment methods. Good outcome occurred in 76% of patients with aneurysms in the anterior circulation, two of the six cases with poor results included patients who were already hemiplegic. Ninety percent of patients with posterior cerebral aneurysms fared well.

Only 67% of patients with basilar or vertebral aneurysms had good outcomes, although more (17%) of these patients were in poor condition preoperatively because of brainstem compression. The atherosclerotic fusiform aneurysm of the basilar artery as in this patient, proves to be the most difficult to treat and usually involves the length of the basilar artery, even when good PComA exist. Successful thrombotic occlusion of this aneurysm results in almost inevitable massive brainstem infarction from occlusion of the anterior inferior cerebellar artery (AICA) and the innumerable perforating vessels arising from the basilar artery²¹. The fusiform basilar artery aneurysms are essentially untreatable. Multiple perforators arise from this vessel, the wall is frequently calcified, and the location for reconstruction is suboptimal. If the vessel thromboses, unforeseen results may occur. Proximal occlusion does not appear to be an option in this case²³.

Conclusion

This is a rare case with ruptured bilobed wide neck P2 segment of PCA aneurysm and atherosclerotic fusiform basilar artery aneurysm, SAH with warning signs and negative CT scan, perimesencephalic hemorrhage and chronic renal failure, which required a high index of suspicion to detect subarachnoid bleeding from ruptured posterior fossa aneurysm, accurate prediction of the site of bleeding and the location of aneurysm location by conventional angiogram, MRI and MRA, and careful planned surgical strategy with the right approach for the P2 segment of the PCA aneurysm, complicated post operative care with airway, triple H therapy, nutrition, additional measures and multiple hemodialysis.

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