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Case Report

Subarachnoid Hemorrhage from a Dissecting Aneurysm of the Posterior Cerebral Artery in a Child : Rebleeding after Stent-Assisted Coiling Followed by Stent-Within-Stent Technique

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Pediatric patients with dissecting aneurysms usually present with ischemia rather than bleeding. We report a case of a 15-year-old boy with a dissecting aneurysm of the posterior cerebral artery (PCA) presenting with hemorrhage. He was first treated with stent-assisted coil embolization, in an attempt to avoid trapping of the PCA and preserve the perforators. After the procedure, he recovered well from general anesthesia, but rebleeding occurred from the same lesion 6 hours after the procedure, therefore endovascular segmental occlusion of the parent artery was performed secondarily. Apparently, a reconstructive method of stent-assisted coiling is worth trying to preserve the parent vessel and perforators, but it is not always efficient and durable for dissecting aneurysms.

Key Words : Subarachnoid hemorrhage · Dissecting aneurysm · Posterior cerebral artery · Children · Stent-assisted coil embolization.

INTRODUCTION

Intracranial aneurysms are rare in the pediatric population, with an incidence ranging from 0.5% to 4.6%[9,18,19,21]. Aneurysms in children are known to be different from those in adults in several aspects, such as male predilection and higher incidences of giant, traumatic, infectious, dissecting, and fusiform aneurysms. The most common location is the internal carotid artery (ICA) bifurcation, and aneurysms of the posterior circulation occur more frequently than in adults, comprising up to 17% of pediatric cases[3,13,18]. Pediatric cases account for 7% of all intracranial arterial dissections[25]. In a review of intracranial aneurysms in children, 45% were reported as dissections[15]. Posterior cerebral artery (PCA) dissecting aneurysms are rare and only 3% (1/34) were located on PCA in a case series of dissecting aneurysms[15]. Most dissecting aneurysms in children present with ischemic symptoms, and subarachnoid hemorrhage (SAH) is a rare mode of presentation, accounting for less than 2% of cases[8]. Therefore, a PCA dissecting aneurysm with SAH is very rare in the pediatric population.

The treatment of PCA dissecting aneurysm is a matter of debate. When the lesion is presented with ischemic symptoms, anticoagulation or conservative management is usually chosen. For ruptured dissecting aneurysms, surgical or endovascular intervention is considered[12]. Because of its anatomical location and its relationship to critical structures such as brain stem and cranial nerves, endovascular treatment is more feasible than surgery[29]. Previous series have shown surgical proximal ligation or trapping without revascularization and endovascular parent vessel occlusion (PVO) may be performed safely[10,11,26,28]. Nevertheless, PVO of PCA may result in ischemic complications because of obliteration of perforating arteries[1]. We report a case in a boy who presented with ruptured dissecting aneurysm of the PCA at the P2 segment.

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CASE REPORT

History and examination

A 15-year-old boy was admitted to our hospital after a sudden onset of loss of consciousness 8 days earlier. He denied of previous history of trauma, infection or vascular diseases. He was jogging at school when he suddenly lost consciousness and showed

tonic-clonic movements. He recovered consciousness on the way to a nearby hospital. Brain computed tomography (CT) revealed SAH. However, cerebral angiography failed to reveal any abnormalities (Fig. 1A, B, C). He was referred to our hospital for further evaluation and treatment. On admission, he showed no focal deficits. Angiography was repeated and a dissecting aneurysm at the P2 segment of the left PCA was demonstrated (Fig. 1D, E, F).

Endovascular treatment

To avoid the risk of thalamic and occipital lobe infarction, we performed a stent-assisted selective aneurysmal occlusion. First, a self-expandable stent (Neuroform III, 3 mm in diameter and 20 mm in length; Boston Scientific, CA, USA) was deployed over the dissected portion of the PCA. Then the aneurysm was occluded with three detachable coils. A remnant sac was noted on postembolization angiography, so another stent (Neuroform III, 3 mm in diameter and 15 mm in length) was deployed within the previous stent, obliterating the residual sac. The distal flow remained intact and the irregular arterial wall became smooth. Complications such as thromboembolism or bleeding were not noted during the procedure (Fig. 2A, B). Postprocedural, temporary anticoagulation with aspirin and clopidogrel was planned and they were taken by the patient 4 hours after the procedure.

The patient was free of deficits after the embolization. However, 6 hours after the procedure, he suddenly became comatose and his systolic blood pressure rose to 230 mmHg. Pupil sizes were isocoric, 3 mm on both sides, but light reflexes were sluggish. Rebleeding of the aneurysm was suspected and brain CT revealed acute intraventricular hemorrhage and SAH (Fig. 2C). External ventricular drainage (EVD) was conducted and the opening pressure was over 30 cm CSF. Immediately after EVD insertion, his mental status improved to Glasgow Coma Scale (GCS) E3M5VE, being able to withdraw from painful stimuli and light reflex became prompt on both sides. Angiography revealed extension of the PCA dissection in length and depth (Fig. 2D). PVO with detachable coils was performed (Fig. 2E). Post-embolization angiography of the left ICA showed middle cerebral artery (MCA) collaterals to the PCA territory (Fig. 2F). No oral anticoagulation medication was given.

Postembolization clinical course

After PVO, his mental status improved and right hemiparesis of grade 2 was noted. Diffusion-weighted MR imaging revealed

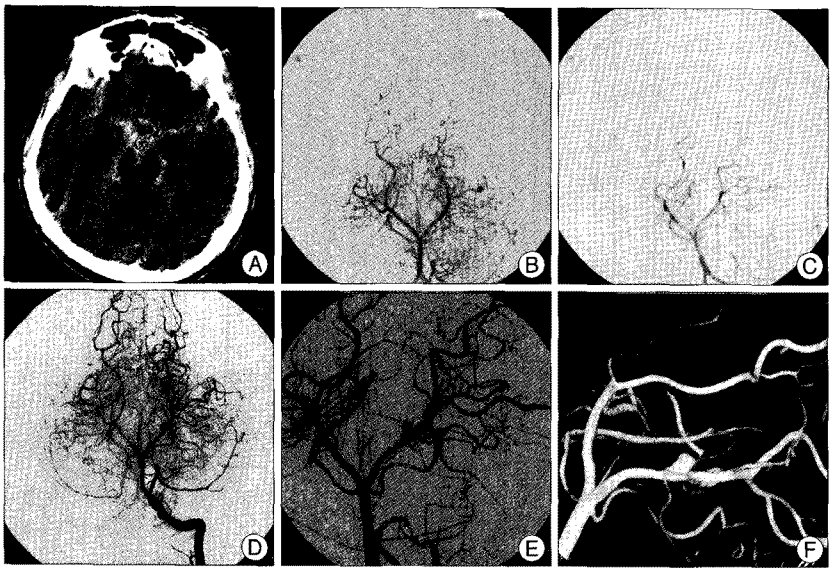

Fig. 1. A : Brain computed tomography at presentation showing thick subarachnoid hemorrhage of basal cistern. Initial vertebral angiogram (B : Anteroposterior view, C : Oblique view) revealing no definite evidence of intracranial aneurysm. Follow-up angiogram (D, E and F : Anteroposterior view, oblique view, and 3D view, respectively) 8 days after symptom onset demonstrating dissecting aneurysm and segmental dissection of the left posterior cerebral artery at the P2 segment.

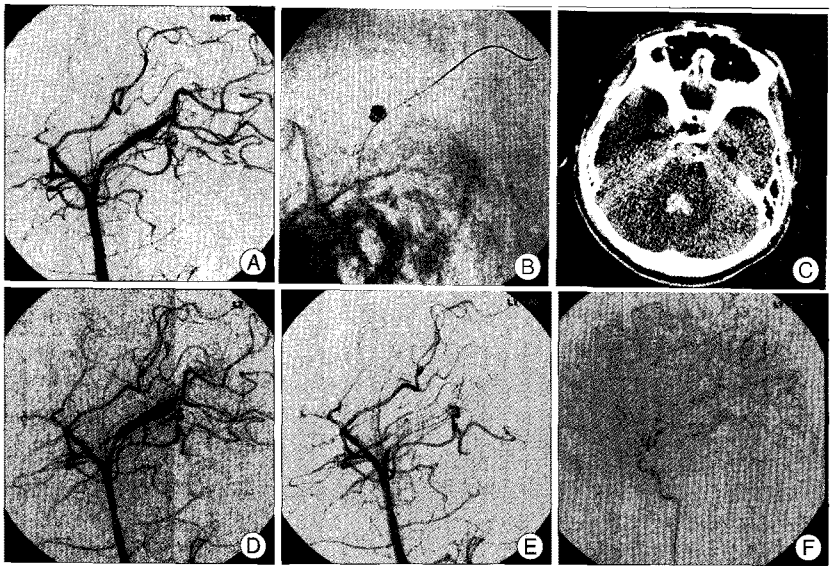

Fig. 2. A and B : Vertebral angiogram oblique views showing two stents deployed in the left PCA and selectively occluded aneurysm with coils. C : Brain CT performed after clinical deterioration, revealing intraventricular and subarachnoid hemorrhage. D : Repeated angiogram oblique view revealing widening of the stented segment. E : The previously stented P2 segment was completely occluded. F : Left carotid angiogram lateral view showing collaterals to the PCA territory from the anterior circulation. PCA : posterior cerebral artery.

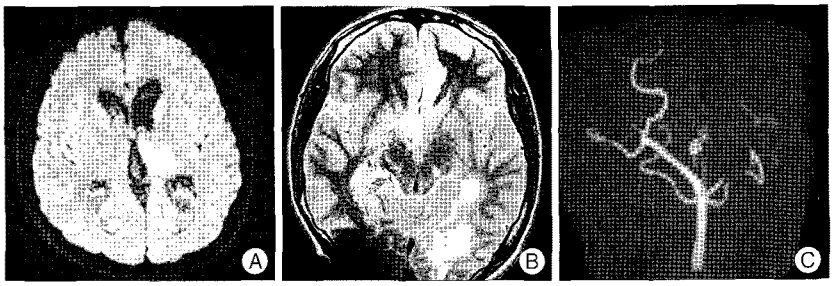

Fig. 3. A : Diffusion MRI performed one day after the parent vessel occlusion (PVO) showing acute infarction of the left thalamus and posterior limb of internal capsule. B, C : Old infarction of the left occipital lobe is noted on the T2-weighted image, axial MRI view (B) and occlusion of the PCA beyond the P1 segment is shown on MRA (C) taken 8 months after PVO. PCA : posterior cerebral artery.

acute infarction in the left posterior thalamus and posterior limb of the internal capsule (Fig. 3A). His consciousness improved gradually. Routine daily follow-up with transcranial doppler sonography was conducted and when vasospasm was suspected, follow-up angiography was performed. Vasospasm was detected twice after PVO, and intra-arterial infusion of nimodipine was done. After removal of the EVD catheter, progressive hydrocephalus was noted. Therefore, ventriculoperitoneal shunting was performed. By the time the patient was transferred to the department of rehabilitation medicine, he was alert and right-side motor weakness had improved to upper extremity grade 3 and lower extremity grade 4.

Follow-up MRA conducted 8 months after treatment showed complete occlusion of the left PCA beyond the P2 segment. The old infarction in the left occipital lobe was also noted (Fig. 3B, C). On the last follow-up, 18 months after the bleeding, the patient was back in school, with some deficits in physical activities. He could walk up and down stairs alone, but was still clumsy when writing. He had fully recovered from right-side hypesthesia, which he had been complaining of at discharge. Visual field was evaluated with Goldman perimetry; his left eye showed no visual field defect, while his right eye had insufficient vision to assess field status because of a subretinal calcification that seems to have been present long before the SAH. The small subretinal calcification has been static in size on follow-up MRI taken 1.5 years since rupture of aneurysm.

DISCUSSION

Treatment of dissecting aneurysm depends on its presentation. In cases presenting with stable ischemic symptoms, anticoagulation or conservative management is recommended[26]. Patients with ruptured dissecting aneurysms or progressive neurologic deficits require treatment by surgical or endovascular means[12]. Surgical treatment includes trapping or proximal ligation with or without revascularization. Reconstruction of the PCA can be achieved by using donor tissue from the superficial temporal artery, occipital artery, or superior cerebellar artery[4,23,29,31]. However, a surgical approach to the PCA aneurysms may be difficult, with potential morbidity of up to 28.5%[29]. Therefore, endovascular procedures are generally preferred, and for PCA dissecting aneurysms, PVO is usually performed[10,11,16,28]. Previous reports have shown that proximal ligation or trapping of a PCA aneurysm can be performed safely without revascularization[10,16,22,30]. This is mostly because of the rich collaterals of the PCA territory from anterior choroidal artery, superior cerebellar artery, MCA, and anterior cerebral artery[7,10]. However, complications may occur and cannot be overlooked[1,22]. Occipital lobar or thalamic infarcts can occur after PVO if there is insufficient collateral blood flow, which may result in hemianopsia or hemiparesis[22,30]. To estimate the collateral supply before PVO, a balloon test occlusion (BTO) can be performed, but this test may give false-positive or false-negative results. Moreover, in cases of dissection, the test procedure may cause further arterial injury. Therefore, most patients usually do not undergo BTO before PVO[1,5,16]. Overall, the difficulty of surgical access and the absence of a single method to ascertain the tolerability of PVO make treatment of PCA dissecting aneurysm a matter of debate.

Recently, interesting endovascular techniques for wide-neck or dissecting aneurysms have emerged, including stent and coil placement and "remodeling technique"[20]. Stent-assisted intraaneurysmal coil embolization is an attractive alternative for patients who cannot tolerate a PVO because of perforators of the parent artery[14,17,27]. The stent may work as a scaffold, providing mechanical support for coil embolization and assisting reconstruction of the diseased parent vessel[32]. The concept of flow diversion, implying stent changes the hemodynamics of the parent vessel and the dissected wall, decreasing the blood flow into the aneurysm allowing spontaneous thrombosis, exists. To divert more blood flow from the dissecting aneurysm sac, a technical advance of overlapping the stent struts has been achieved, thus decreasing the porosity of the stent and straightening the parent vessel[6,27].

In our case of a PCA dissecting aneurysm, stent-assisted coil embolization was chosen first. The patient was 15 years of age, 8 days had already passed since the initial bleed at the time of treatment, and the patient was clinically stable and free of neurologic deficits. Even in the presence of rich collaterals, some have advised the avoidance of PVO in P1 and P2 segments whenever possible, because numerous perforators to the brainstem arise from those segments[30]. It seemed reasonable, if only technically feasible, to avoid taking the risk of ischemia of the PCA territory in such a young and clinically stable patient. Two stents were used to reinforce the unstable wall of the dissected parent vessel and to obliterate the residual aneurysmal sac. The patient was well and without any deficits for 6 hours after the procedure, but showed sudden deterioration because of progression of the dissection and rebleeding. After this, we had no choice but to con-

duct a PVO. Though his mental status recovered fully, he suffered from infarction of the left thalamus and occipital lobe. Although risk of embolism may be present, oral anticoagulation medication was not given because the patient suffered from rebleeding. Regarding the cause of dissecting aneurysm, possible relation has been suggested with trauma, infection such as syphilis, migraine headaches, vasculopathies such as cystic medial necrosis, mixed connective tissue diseases, fibromuscular dysplasia, and homocystinuria[2,24]. The patient in this case report had no connection with the causative factors, suggesting it was a spontaneous dissecting aneurysm presenting with SAH.

CONCLUSION

From the present case, it is obvious that PVO of a PCA dissecting aneurysm is not always safe and complications cannot be completely avoided. Preserving the parent artery and occluding the dissecting aneurysm selectively might be the ideal treatment. However, stent-assisted coiling and second stent placement may not be safe for PCA dissecting aneurysms. Further technical advances in stents, allowing effective flow diversion, may provide us with a better treatment modality.

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