Delayed Thrombosis of the Basilar Artery after Stenting for a Basilar Trunk Dissection Aneurysm

A Case Report and Review of the Literature

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Summary

A 30-year-old man was referred in our department for treatment of a midbasilar trunk aneurysm. His presenting symptoms included headache and dizziness. A CT scan at another hospital showed no significant findings whereas a digital subtraction angiogram disclosed a dissecting aneurysm in the midbasilar trunk, and there was severe stenosis in the basilar artery. After discussion, we planned to use stent-assisted-coil embolization technique. During the procedure, a LEO stent (Balt, Montmorency, France) was implanted into the basilar artery across the aneurysm neck, but fearing acute basilar artery occlusion because of stent collapse or thrombus we did not fill coils into the aneurysm.

After the procedure, the completion angiography demonstrated considerably decreased flow into the aneurysm, with stasis persisting into the venous phase of angiography. The patient awoke from general anaesthesia after the procedure and had no additional neurological symptoms, he was discharged three days later and used clopidogrel and aspirin for antiplatelet therapy. Six months later when he was admitted for a recheck, a DSA showed the basilar artery was occluded completely and the aneurysm had disappeared even though the patient remained neurologically normal.

Introduction

Aneurysms located in the middle or lower basilar artery are difficult to reach for surgical clipping because of the complicated anatomical structures in this area, so endovascular treatment has gradually become the main method to manage these aneurysms during the last decade¹⁻⁴. Stent-assisted detachable coil embolization has offered new reliable options to many patients with wide-necked aneurysms who were previously treated with surgical clipping or vessel sacrifice^{1,5}. Several cases have reported that using double stent, telescoping stent, stent-within-stent techniques, or balloon-expanding stents alone can promote thrombosis in predominantly dissecting aneurysms^{3,6-8}, but chronic basilar artery occlusion caused by stent placement had rarely been reported. We report a case of delayed thrombosis and complete vessel occlusion after stenting of midbasilar artery dissection aneurysm with no clinical symptoms.

Case Report

A 30-year-old man who presented with headache and dizziness in two years aggravated in two months was admitted to our hospital. A CT scan at another hospital scan showed no significant findings whereas a digital subtraction angiogram disclosed a dissecting

Figure 1 Injection from the left internal carotid artery. The basilar artery and the aneurysm are filled by contrast medium.

Figure 2 Left vertebral injection. The AICA was involved in the aneurysm (arrow).

aneurysm in the midbasilar trunk between the right anterior inferior cerebellar artery (AICA) and the vertebrobasilar junction, the right AICA was involved, the aneurysm could be filled from the left vertebral artery injection and left internal carotid artery (ICA) injection, with a diameter of approximately 9.7 mm x 7.7

Figure 3 Right vertebral artery injection. The contrast medium did not fill the basilar artery but reversed to fill the left vertebral artery (arrow) and the left PICA (arrowhead), implying that the direction of the blood stream was reversed.

mm and neck length of 5.3 mm (Figures 1 and 2). In the right vertebral artery injection the basilar artery was not filled, the contrast medium reversed to fill the left vertebral artery and the left posterior inferior cerebellar artery (PICA) (Figure 3), and from the left ICA injection the basilar artery and the aneurysm could be filled completely (Figure 2). This demonstrated that the blood flow in the basilar artery was reversed. Left vertebral artery injection also revealed aplasia of the P1 segment on the right side (Figure 5). Open surgical treatment was thought to carry an unacceptably high risk and unlikely to achieve a definitive treatment, so endovascular treatment was selected to prevent the possible bleeding of the aneurysm. Considering the length of the neck and the risk associated with protrusion of the coils, a decision to perform stent-assisted coil embolization was made. The patient was administered aspirin 100 mg and clopidogrel 75 mg Qd antiplatelet therapy for three days before the procedure. The procedure was performed under general anaesthesia. The patient was administered 4,000 units of heparin intravenously during the procedure to maintain an activated clotting time of 250-350 after the femoral sheath was introduced. A 6-French guiding catheter (Cordis Corp, NJ, USA) was placed in the vertebra dentate level of the left vertebral artery. Through

the guiding catheter, a Vasco 21+ microcatheter (Balt, Montmorency, France) was navigated on a 0.014 guidewire (Balt, Montmorency, France) to the parent vessel distally to the aneurysmal neck. The delivery system of the Leo stent (Balt, Montmorency, France) was then introduced inside the Vasco microcatheter. The 3.5 mm x 25 mm stent was successfully deployed in the basilar artery (Figure 4). Control angiography performed immediately after stent placement demonstrated stagnation of the contrast medium due to significantly decreased intra-aneurysmal flow (Figure 5), allowing for acute basilar artery occlusion because of stent crevasse or thrombus, no coil packing was performed. After the procedure, the patient awoke from general anaesthesia and no additional neurological symptoms emerged. He was discharged three days later and used clopidogrel and aspirin for antiplatelet therapy. He was admitted again one month later and a DSA showed no change in the morphology and size of the aneurysm (Figure 6), and the basilar artery remained. So we still did not fill coils into the aneurysm, and let the patient stop using clopidogrel and continue to use aspirin. Six months after his first admission, a DSA showed the basilar artery was occluded and the aneurysm had also disappeared (Figures 8-10), but the patient had no clinical symptoms and was discharged.

Discussion

Regardless of its presentation, acute basilar artery dissection seems to have a worse prognosis than a dissection limited to the vertebral artery⁹⁻¹², but little is known about the clinical course and prognosis. A literature review by Masson et Al.¹⁰ disclosed 38 cases of basilar artery dissection, including 18 cases of isolated basilar artery dissection. Of the 38 patients, 30 (78.9%) died. Yoshimoto et Al.¹² reported ten patients with basilar artery dissection (four with ruptured and six with unruptured dissections). Of the four patients with ruptured basilar artery dissection, one died and two were severely disabled, whereas four of the six patients with unruptured basilar artery dissection were severely disabled because of infarct progression. The clinical symptoms of our patient might have been caused by mass effect or mild ischemia. Although he had experienced neither SAH nor infarction in the CT scan, allowing for

Figure 4 Fluoroscopy after implantation of the LEO stent.

Figure 5 After stent implantation, stasis of contrast medium in the aneurysm (arrow), the white part in the aneurysm was because the stasis of contrast medium was subtracted.

Figure 6 Angiogram one month after the first procedure.

the probably poor prognosis, we decided to treat the lesion aggressively.

Aneurysms located in the basilar trunk are among the most difficult to manage surgically. Anatomically, the BA trunk is located in the depth of the valley surrounded by the bilateral bones. Surgical access to this area is hampered by a dense collection of vital cranial nerves and perforating arteries to the brain stem. Furthermore, the difficulty in treating these aneurysms

Figure 8 Left vertebral artery injection, the basilar artery was occluded completely.

Figure 7 Right vertebral artery injection six months later, the left vertebral artery and the PICA were no longer filled.

is compounded by their rarity, which limits the acquisition of experience in treating these lesions. However, an endovascular approach to this area is comparatively easy because of the simple access to the BA. In particular, the use of stents has allowed the treatment of wide-necked saccular, fusiform or dissecting aneurysms with detachable coils placed through the interstices of the stent, preventing coil prolapsed into the parent vessel^{1,4,5,13}. Furthermore, the metallic scaffolding along the intimal surface of the artery may reconstruct the parent artery by remodeling blood flow and stimulating endothelial growth ideally to promote stasis, platelet adhesion and thrombosis within the aneurysm^{4,14}.

Thromboembolic complications and intimal hyperplasia that cause hemodynamically significant in-stent stenosis are well documented after metallic stent placement in the intracranial vasculature^{1-5,14}. The prevention of acute thrombotic complications entails the routine use of systemic heparin (activated coagulation test, 2-2.5 x baseline) during endovascular procedures. The inherent thrombogenicity of metallic stents mandates antiplatelet therapy as a pre and postprocedure supplement to heparinization in stent-assisted cases. Our standard protocol is an antiplatelet regimen (75 mg of clopidogrel and 100 mg of ASA daily) for three days before the procedure and dual antiplatelet

Figures 9 and 10 Injection of the two common carotid arteries.

therapy (75 mg of clopidogrel and 100 mg of ASA daily for one month after stent placement, followed by 100 mg ASA indefinitely).

Several factors may have contributed to the delayed stent thrombosis in the case presented here. We could see the stenosis of the proximal end of the dissecting aneurysm in the figures (Figure 2), when the stent was placed in the basilar and vertebral artery lumen, the stenosis was still not propped open (Figure 5). In addition, both of the two posterior communicating arteries were well developed, and the basilar artery could be filled from the left internal carotid artery, so there was a hemodynamic balance between the two PcomA and the two vertebral arteries. Stagnation of contrast medium in the aneurysm after stent implantation implied that the blood stream was very slow in the aneurysm, and when the clopidogrel was discontinued, actual thrombosis might occur in the stagnant arterial bed¹⁵, and then lead to occlusion of the narrow basilar artery.

We have found only two cases similar to ours in the English literature^{16,17}. One was a 19-month-old infant with tuberous sclerosis who developed a giant aneurysm of the midbasilar artery. The authors used detachable coils to fill and occlude the aneurysm and the involved segment of the basilar artery. After the procedure, the child had transient peripheral fifth cranial nerve palsy and no permanent neurologic deficits¹⁶. The other case was a 30-year-old

man presenting with signs of brain stem compression three years after initial presentation of brain stem infarction. The CTA showed a large asymmetric pseudoaneurysm in the basilar trunk. The authors placed two overlapping stent. After stent deployment, DSA showed flow redirection through the stent and the basilar artery with stasis of contrast media in the pseudoaneurysmal sac. A 4-month follow-up vertebral artery angiogram and right internal carotid artery angiogram revealed occlusion of the basilar artery and hypertrophied right posterior communicating artery supplying the distal basilar artery and its branches¹⁷.

Basilar artery occlusion has long been recognized as a dramatic clinical event^{15,18,19}, but like the case presented here, patients were free of clinical symptoms even though the BA was totally occluded. We speculate the following reasons.

The area critical to consciousness in man is the paramedian pontine tegmental gray matter immediately ventral to the ventricular system, extending continuously from the posterior hypothalamic reticular formation rostrally to approximately the lower third of the pontine tegmentum caudally. This area is supplied by the penetrating branches of the basilar artery and the thalamoperforating branches of the posterior communicating and posterior cerebral arteries. The acute loss of consciousness in the majority patients with basilar embolism

may be caused by a temporary or permanent occlusion of the rostral part of the basilar artery¹⁹. Both sides of the posterior communicating arteries in our patient were well developed, the rostral part of the basilar artery was well protected, so the patient did not experience consciousness disturbances. Furthermore, both sides of the PICA and the SCA were well developed, and the occlusion of basilar artery had not affected them, in the capillary phase, both hemispheres of the cerebellar were filled completely.

The basilar artery has two sets of pontine branches¹⁵: 1) a minute median set arteries at right angles to the parent artery, and enters the median sulcus of the pons along its length and, 2) a transverse set, which runs laterally and subdivides into smaller branches which penetrate the ventral surface of the pons in a segmental fashion. Although considered end-arteries, collateral channels, which are functional

as well as morphologic entities, have been demonstrated between adjacent vascular territories. These anastomoses could ensure the blood supply when one or more of the vessels were occluded for any reason, but the compensation course needs time, if the patient had an acute basilar artery occlusion, the clinical outcome might be different.

Conclusions

Basilar artery occlusion has long been recognized as a dramatic clinical event, but our patient who experienced chronic basilar artery occlusion not only presented without neurological symptoms, but the dissecting aneurysm also disappeared. Through this case we could conclude that in some circumstances, sacrificing the basilar artery to manage some kind of hard-to-treat midbasilar aneurysm is possible, but that conclusion needs further investigation.

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