Case Report

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Acute Basilar Artery Dissection Treated by Emergency Stenting in a 13-Year-Old Boy

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Key Words
Basilar artery dissection · Brain stem · Cerebral infarction · Stenting

Abstract
We report a 13-year-old boy who presented with acute basilar artery occlusion due to traumatic arterial dissection. Because a grave prognosis was expected if left untreated, and the chance of neurological recovery was believed to be unlikely but not zero, given that emergency stenting for the dissection was performed within 6 h of ictus. Recanalization of the basilar artery with stent placement did not change the poor prognosis in this patient because there was extension of dissection into the posterior cerebral arteries.

Introduction

Ischemic stroke is a relatively infrequent occurrence in the pediatric population, but it is not rare. Such pediatric ischemic strokes have multiple possible etiologies. Arterial dissection is one of the causes and occurs either spontaneously or by trauma. Basilar artery (BA) dissection is rare compared to vertebral artery dissection [3, 7]. The prognosis of BA dissection varies from minimal neurological deficits to grave outcomes (including frequent death) [2, 7, 9, 14, 15, 17]. Herein, we report a 13-year-old boy who presented with a traumatic BA dissection and underwent emergency stenting on the day of ictus in an effort to recannulate the BA.

Case Presentation

This 13-year-old boy without a remarkable personal or family history of related medical problems developed an alteration in mental status when he was swimming a 100 m freestyle event during a regional junior high school swimming competition. During the last 25 m of the swim, he began to swim strangely before finally reaching the end of the pool. He was pulled out of the pool immediately and was found to be unconscious and hyperventilating.

He was referred to us 1 h after the ictus. At admission, he was comatose and tetraplegic with a Glasgow coma scale of 3 points. Both pupils were dilated and not reactive to light. The CT scan at the time of admission was normal (fig. 1). Fluid-attenuated inversion recovery MR images performed 2 h after ictus were normal except for a slightly high signal in the distal BA, suggesting either arterial dissection or a blood clot. Diffusion-weighted images showed acute ischemia in the cerebellum, pons, midbrain, bilateral thalami, and bilateral occipital lobes. MR angiography failed to demonstrate the BA. Laboratory data were within normal limits.

At this point, we thought the patient's condition was fatal if left untreated. We also thought that there was a small, but not zero,

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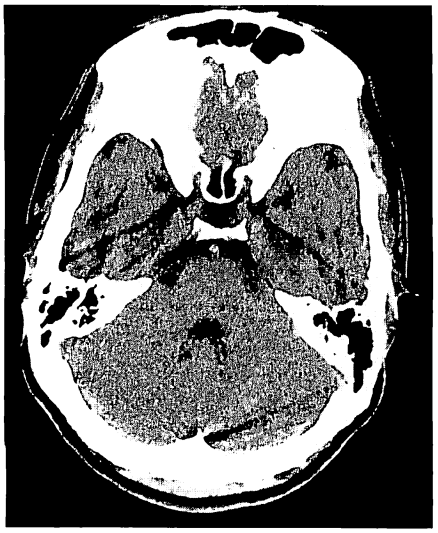

Fig. 1. Computed tomography at admission, which was interpreted as normal. One hour had elapsed from ictus until this examination.

Fig. 2. Left vertebral angiogram (AP view) on admission showing an occlusion of the basilar artery distal to the branch point of the right anterior inferior cerebellar artery (arrow).

chance of neurological recovery if the occluded BA was recanalized within a golden time. We, therefore, proceeded to perform catheter angiography and contemplate a possible intervention. Although diffusion-weighted images showed slightly high intensity lesions, we thought that the patient was still in the therapeutic window given that the time required for diagnostic and therapeutic angiography, i.e. within 6 h of ictus.

A written-informed consent for a diagnostic angiography and possible reperfusion treatment including stent deployment was obtained from the parents. We started diagnostic angiography at 4.5 h after the ictus to determine the etiology of the BA occlusion and allow for possible intervention. Catheter angiography and the following intervention were performed by the head author (M.K.), who is a board-qualified neuro-interventionalist in Japan. The angiography equipment used was Philips bi-plane digital subtraction angiography system (Integris Allura, Eindhoven).

The left vertebral angiogram showed BA occlusion at the proximal BA distal to the branch point of the right anterior inferior cerebellar artery (fig. 2). The right carotid angiogram showed minimal collateral blood flow to the right posterior cerebral artery through the small right posterior communicating artery. The left carotid angiogram showed no collateral flow to the left posterior cerebral artery. After diagnostic angiography, a 6F-guiding catheter was introduced into the left cervical vertebral artery under full heparinization. Then, a microcatheter (RapidTransit, Cordis, Miami, Fla., USA) was navigated to the BA in order to probe the lesion. Selective slow injection of the contrast at the proximal BA showed a 'double lumen' sign in the left P1–2 segments and in the right P1 segment of the posterior cerebral arteries, indicating the propagation of arterial dissection from the BA to the bilateral posterior cerebral arteries (fig. 3). Clot formation, if any, was not the major cause of the arterial occlusion.

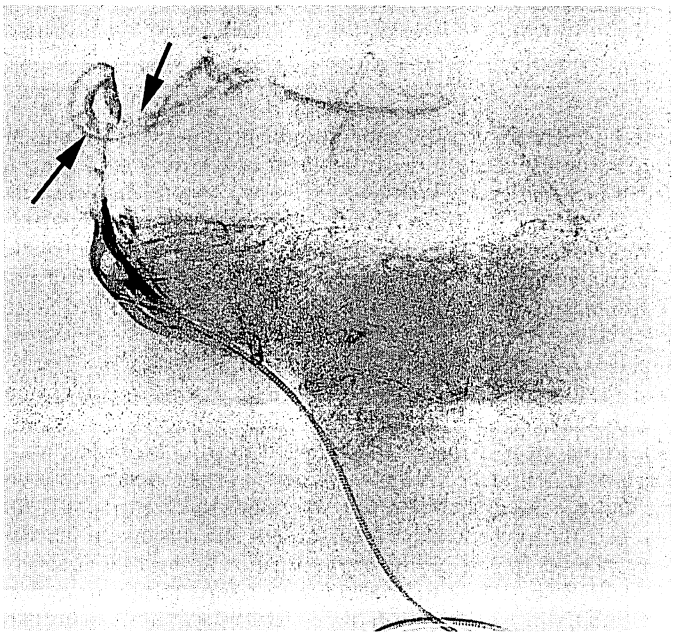

Fig. 3. Selective slow injection (lateral view) through a microcatheter placed at the proximal basilar artery showing 'double lumen' sign at the bilateral posterior cerebral arteries (arrows), being more prominent on the left side.

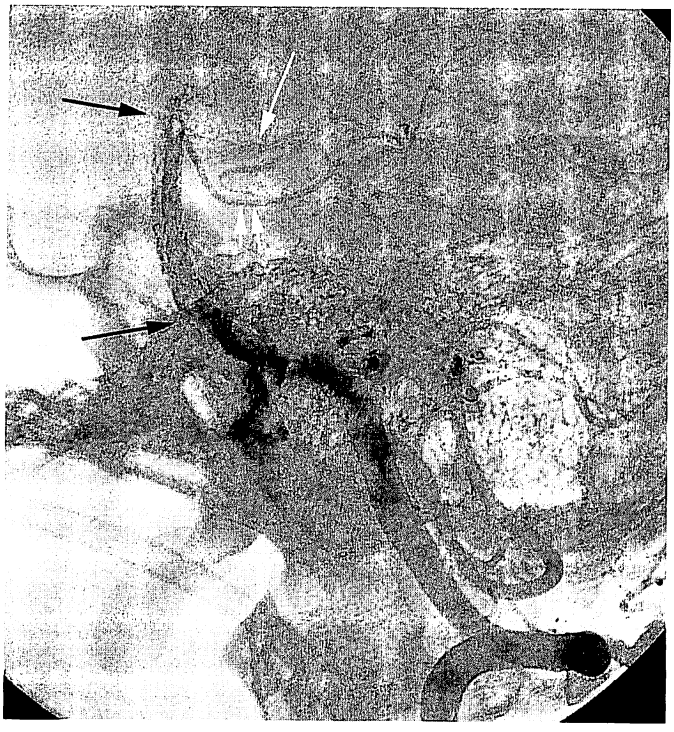

Fig. 4. After the second stent placement, the left posterior cerebral artery (white arrow) and left superior cerebellar artery (white double arrows) are visualized, but the right posterior cerebral and right superior cerebellar arteries are not shown. Black arrows indicate the proximal and distal ends of the two stents. Laminar flow was caused by a slow flow due to incomplete recanalization of the posterior cerebral arteries.

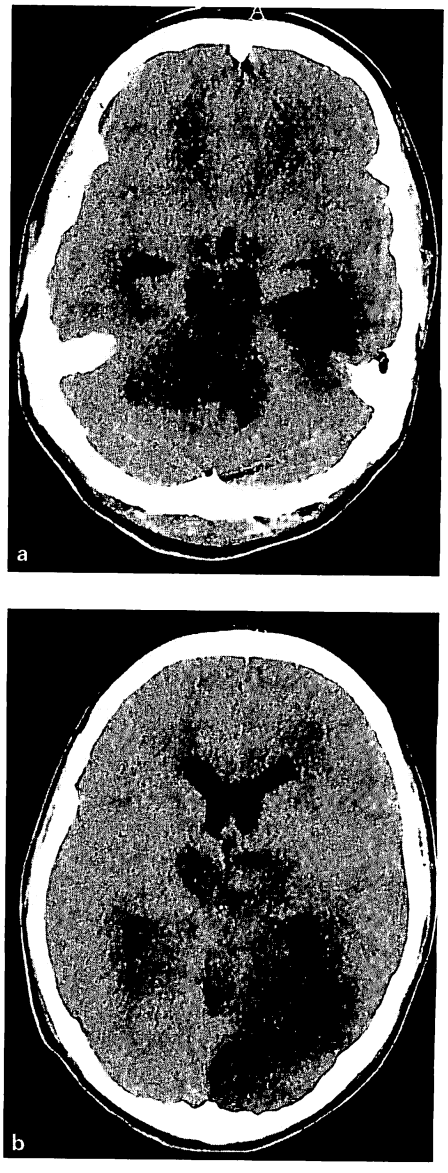

Fig. 5a, b. Computed tomography images on day 5 showing subacute infarction in the pons, cerebellum, midbrain, bilateral thalami, and bilateral occipital lobes, which are supplied by the basilar artery, cerebellar arteries, posterior cerebral arteries as well as the thalamoperforating arteries.

The microcatheter was carefully advanced to the left posterior cerebral artery through the true lumen of BA dissection using a 0.014-inch guidewire (Transend-Ex floppy, Boston Scientific, Fremont, Calif., USA), which was then exchanged with a stiffer 0.014-inch guidewire (déjà-vu, Cordis Japan, Nagoya) to obtain stable stent navigation. A S-670 coronary stent (3.5 × 12 mm) (Medtronic, Minneapolis, Minn., USA) was deployed at the mid-BA. Since control angiography did not fill the distal BA, another S-670 stent (3.5 × 15 mm) was deployed at the distal BA with some overlap with the first stent in the telescoping fashion. Up to this point, 5.5 h had passed from the ictus. Stenting resulted in improved visualization of the left posterior cerebral and left superior cerebellar arteries, but the flow in the BA was very slow. The right superior cerebellar and posterior cerebral artery were not visualized (fig. 4). We stopped the procedure because further reperfusion of the BA branches was impossible due to propagation of the arterial dissection. Local fibrinolysis was not considered since the occlusion was determined to be due to arterial dissection and not clot formation.

The post-procedural CT showed no subarachnoid hemorrhage indicative of vessel rupture. Subsequent CT images on day 5 showed subacute infarction of the brain stem, cerebellum and thalami, and occipital lobes (fig. 5a, b). Follow-up angiography on day 10 showed an occlusion of the BA at the distal part of the stents, probably due to the initial incomplete revascularization of the posterior cerebral arteries and subsequent clot formation (fig. 6). The patient was on controlled ventilation for the initial 14 days, and then weaned from the mechanical ventilation. Due to tracheal malacia, however, continuous controlled ventilation was required thereafter. At 4 months after ictus, this patient was still on controlled ventilation and tube-

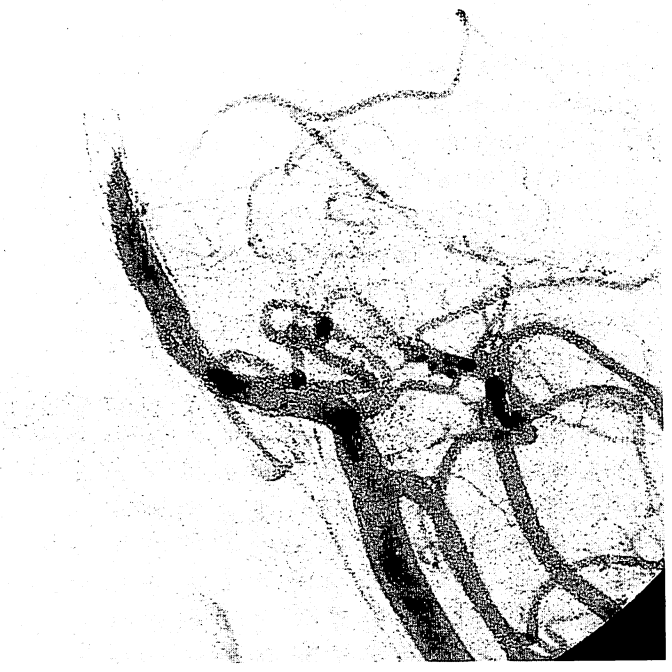

Fig. 6. Follow-up angiogram (lateral view) on day 10 showing an occlusion of the basilar artery at the distal part of the stents probably due to the initial incomplete revasculization of the posterior cerebral arteries and the subsequent thrombus formation.

fed. Neurologically, he was in the vegetative state. Retrospectively, stenting procedure in the acute stage had not changed the natural course of extensive BA dissection in this patient.

Discussion

There are a variety of etiologies of ischemic stroke in children [4, 6, 8, 10, 16] – etiologies that are strikingly different from those in adults. They are grouped into vascular, intravascular, and embolic causes. Vascular causes include arteriopathies, vasospastic disorders, vasculitis, systemic vascular disease, and trauma. Intravascular causes include hematological disorders, acquired prothrombotic states, congenital prothrombotic states, and metabolic disorders. Embolic stroke is attributed to congenital heart disease, acquired heart disease, and trauma.

The predisposing factors of vertebrobasilar dissection in adults include trauma, atherosclerosis, hypertension, cystic medial degeneration, fibromuscular dysplasia, syphilis, contraceptive drugs, and migraine. In children, however, the common causes of the vertebrobasilar ischemia include traumatic or spontaneous dissection of the cervical or intracranial vertebral artery and the BA, minor head injury causing perforator ischemia, and migraine [7, 8, 10, 12, 17]. Minor or trivial trauma and sport exercise are often associated with arterial dissection, as happened in our patient [5].

Clinical presentation of the BA dissection is either a brain stem ischemia or, less frequently, subarachnoid hemorrhage [15]. BA dissection may be limited to the BA itself, but it is occasionally caused by the extension of vertebral artery dissection [3, 14]. More rarely, BA dissection can extend to its branches, such as the cerebellar arteries and posterior cerebral arteries [1, 2, 9, 11]. The management of BA dissection is controversial, and its prognosis varies from minimal neurological deficits to severe disabilities or death [2, 7, 9, 14, 15, 17].

BA dissection with favorable outcomes are usually limited to the localized short BA segment with enough antegrade or rich collateral blood flow. Thus, these patients can be treated conservatively with anticoagulation or antiplatelet therapy [5, 12, 17], although there remains a small possibility of subarachnoid hemorrhage developing from the dissection. In other words, aggressive interventional treatment is not indicated [7]. On the contrary, BA dissections with poor or fatal outcomes are associated with poor collateral blood flow to the brain stem [13]. In this situation, we believe that aggressive intervention is warranted [18]. To our knowledge, there has been no report of acute revascularization for total BA occlusion due to arterial dissection.

Stenting may not be indicated, however, when the extent of BA dissection is extensive, i.e. propagating beyond the basilar apex to bilateral posterior cerebral arteries because stents that can be brought to the intracranial peripheral arteries are not available. In such situations, stenting limited to the BA is not enough to re-establish adequate blood flow to the major branches of the BA, as occurred in our case. For the performance of intracranial stent deployment, well-trained professionals with proper neuro-interventional technique as well as high quality digital subtraction angiography equipment are required.

In conclusion, BA dissection with minimal or moderate neurological deficits can be treated conservatively. BA dissection limited to the BA itself and causing profound brain stem ischemia can be treated with stent deployment. However, BA dissections extending distal to the posterior cerebral arteries are not candidates for revasculization using stenting techniques at present.

References

1 Amin-Hanjani S, Ogilvy CS, Buonanno FS, et al: Treatment of dissecting basilar artery aneurysm by flow reversal. Acta Neurochir (Wien) 1997;139:44–51.

2 Berger MS, Wilson CB: Intracranial dissecting aneurysms of the posterior circulation: Report of six cases and review of the literature. J Neurosurg 1984;61:882–894.

3 Caplan LR, Baquis GD, Pessin MS, et al: Dissection of the intracranial vertebral artery. Neurology 1988;38:868–877.

4 Chabrier S, Husson B, Lasjaunias P, Landrieu P, Tardieu M: Stroke in childhood: outcome and recurrence risk by mechanism in 59 patients. J Child Neurol 2000;15:290–294.

5 Chung YS, Han DH: Vertebrobasilar dissection: A possible role of whiplash injury in its pathogenesis. Neurol Res 2002;24:129–138.

6 de Veber G: Arterial ischemic strokes in infants and children: an overview of current approaches. Semin Thromb Hemost 2003;29: 567–573.

7 James CA, Glasier CM, Angtuaco EE: Altered vertebrobasilar flow in children: angiographic, MR, and MR angiographic findings. AJNR Am J Neuroradiol 1995;16:1689–1695.

8 Kieslich M, Fiedler A, Heller C, Kreuz W, Jacobi G: Minor head injury as cause and co-factor in the aetiology of stroke in childhood: a report of eight cases. J Neurol Neurosurg Psychiatry 2002;73:13–16.

9 Kurihara S, Nakayasu H, Aoyama Y, et al: A young adult case of spontaneous basilar artery dissection. Clin Neurol 2002;42:970–973.

10 Lotze TE, Paolicchi J: Vertebral artery dissection and migraine headaches in children. J Child Neurol 2000;694–696.

11 Manz HJ, Luessenhop AJ: Dissecting aneurysm of intracranial vertebral artery: case report and review of literature. J Neurol 1983; 230:25–35.

12 Marietta M, Bertesi M, Rozzi N, Cano MC, Vallone S, Cappi C, Pozzi S, Torelli G: Low-molecular-weight heparin for vertebral artery dissection. Clin Appl Thromb Hemost 2002;8: 179–181.

13 Moscow NP, Newton TH: Angiographic implications in diagnosis and prognosis of basilar artery occlusion. AJR Am J Radiol 1973;119: 597–604.

14 Nakatomi H, Nagata K, Kawamoto S, et al: Basilar artery occlusion due to spontaneous basilar artery dissection in a child. Acta Neurochir (Wien) 1999;141:99–104.

15 Pozzati E, Andreoli A, Padovani R, et al: Dissecting aneurysms of the basilar artery. Neurosurgery 1995;36:254–258.

16 Roach ES: Stroke in children. Curr Treat Options Neurol 2000;2:295–303.

17 Schols G, Van Havenbergh T, Parizel PM, Delrue F, Ceulemans B, Jorens PG: Traumatic basilar artery dissection in a child: need for anticoagulation? Neuropediatrics 1998;30:328–330.

18 Willing SJ, Skidmore F, Donaldson J, et al: Treatment of acute intracranial vertebrobasilar dissection with angioplasty and stent placement: Report of two cases. AJNR Am J Neuroradiol 2003;24:985–989.