Received: 30.10.2010 / **Accepted:** 24.11.2010
DOI: 10.5137/1019-5149.JTN.3830-10.0

Endovascular Coil Embolization of Parent Artery for Giant Intracranial Basilar Artery Dissection: A Case Report

Dev İntrakraniyal Basiler Arter Diseksiyonu için Ana Arterin Endovasküler Sarmal Embolizasyonu: Bir Olgu Sunumu

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ABSTRACT

Isolated dissection of the basilar artery (BA) is rare and still a big challenge regarding treatment. The suitable therapeutic strategies for dissection of the BA remain controversial. We report the case of a young patient with a giant unruptured dissection of the BA successfully treated by endovascular coil embolization. A 14-year-old male who underwent computed tomography (CT) scan following a minor head trauma was incidentally found to have a lesion located anterior to brain stem. Further imaging with magnetic resonance imaging (MRI), computed tomography angiography (CTA) and digital subtraction angiography (DSA) were consistent with a diagnosis of giant unruptured dissection of the BA. The patient was initially observed conservatively. The follow-up DSA obtained 3 months later revealed extension of the dissection inferiorly to involve the left VA. The dissection expanded at the conjunction of VAs in the shape of ball. Therefore, coil embolization to occlude BA at the bottom of aneurysm was performed as a further treatment. Follow-up DSA 6 months later demonstrated complete obliteration of the dissection and good compensative perfusion from extensively collateral circulation. For young patients with isolated dissection of the BA, coil embolization to occlude BA at the bottom of aneurysm might be tolerable and effective.

KEYWORDS: Basilar artery, Giant, Dissection, Endovascular embolization, Treatment

ÖZ

Baziler arterin (BA) izole diseksiyonu nadirdir ve tedavisi halen zordur. BA diseksiyonu için uygun terapötik stratejiler tartışmalıdır. Dev bir rüptüre olmamış BA diseksiyonu bulunan ve endovasküler sarmal embolizasyonuyla başarıyla tedavi edilen genç bir hasta sunuyoruz. Küçük bir kafa travması sonrasında bilgisayarlı tomografi (BT) yapılan 14 yaşında bir erkek hastada beyin kökünün önünde bir lezyon saptandı. Manyetik rezonans görüntüleme (MRG), bilgisayarlı tomografi anjiyografi (BTA) ve dijital subtraksiyon anjiyografisi (DSA) ile yapılan ileri görüntüleme sonuçları rüptüre olmamış dev BA diseksiyonu tanısıyla uyumluydu. Hasta başlangıçta konservatif olarak izlendi. Üç ay sonra çekilen takip DSA, diseksiyonun inferiora uzanıp sol VA'yı da olaya kattığını gösterdi. Diseksiyon VA'ların birleşiminde bir top şeklinde genişlemişti. Bu nedenle ileri tedavi olarak anevrizma dibinde BA oklüzyonu için sarmal embolizasyonu yapıldı. Altı ay sonra yapılan kontrol DSA diseksiyonda tam obliterasyon ve yaygın kollateral dolaşımdan iyi kompanse edici perfüzyon gösterdi. İzole BA diseksiyonu olan hastalarda anevrizma dibinde BA oklüzyonu için sarmal embolizasyonu tolere edilebilir ve etkin olabilir.

ANAHTAR SÖZCÜKLER: Baziler arter, Dev, Diseksiyon, Endovasküler embolizasyon, Tedavi

INTRODUCTION

Isolated dissection of the BA is a rare but increasingly recognized entity because of the advancement of medical imaging techniques for diagnosis. However, owing to limited data about its pathogenesis and natural course, the suitable therapeutic strategies for dissection of the BA remain controversial (2,7,14,20,21,25). Unlike dissection involving both VA and BA, isolated BA dissections are generally asymptomatic and rarely cause subarachnoid hemorrhage (2,3,5,7,25). As such, conservative management is recommended in most cases. However, surgical interventions are essential for those patients with progressive deterioration secondary to rupture or bleeding (5,7,14,24). The current therapeutic options mainly include endovascular treatment, wrapping to reinforce the dissected wall, direct surgical ligation or clipping and arterial reconstruction (14,20,21,25). The safety and efficacy of these techniques are not well documented except for a few anecdotal descriptions.

We report the case of a 14-year-old male with isolated dissection of the BA that was first observed conservatively, followed by endovascular coil embolization to occlude the BA at the bottom of the dissection. Complete obliteration of BA was demonstrated by follow-up DSA obtained 6 months later without any neurological complication, suggesting that this method might be a practical approach in young patients.

CASE REPORT

Clinical Presentation

A 14-year-old boy admitted with minor head trauma underwent CT scan of the head. An oval-like hyperdense lesion with a diameter of 14mm (Figure 1A), which enhanced after contrast (Figure 1B), was incidentally detected. The neurological examination was completely normal. MRI showed slight hypointensity with scattered hyperintensity on T1-weighted image and isointensity or hypointensity on T2-weighted image (Figure 1C, D). An intimal flap (Figure 1E, pointed by right arrow), as well as a thrombus (Figure 1E, pointed by left arrow), in the dilated lumen of BA could also be seen when observed on the FLAIR image. CTA of the brain depicted a large fusiform enlargement with eccentric filling defects situated in the upper and middle portion of BA and extended to the top of the BA, followed by an irregular and slight dilatation at its proximal part. The fusiform lesion was about 28mm in length, with 8.6 mm maximum in its anteroposterior diameter and 5.2 mm maximum in its transverse diameter (Figure 1F). Further examination using DSA performed on day 5 demonstrated double lumen of the BA in the coronal section (Figure 1G) and retention of contrast medium in the venous and the later period of arterial phases

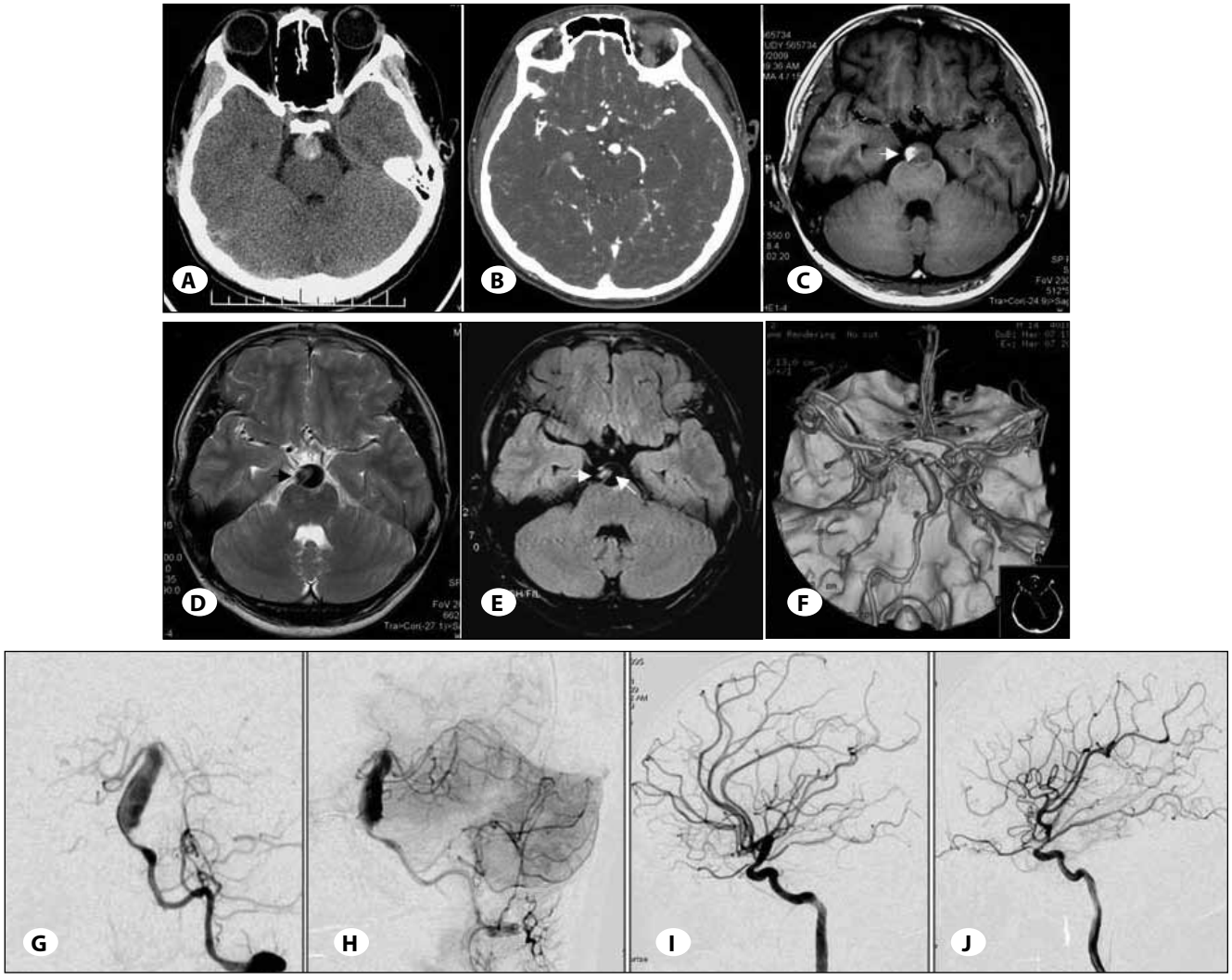

Figure 1: Preoperative imaging examinations **A)** Unenhanced computed tomography (CT) scanning shows an oval-like hyperdensity lesion anterior of the brain stem. **B)** Enhanced CT scanning shows eccentric intensification of the lesion. **C-E)** Magnetic resonance imaging (MRI) shows the lesion presenting slight hypointensity with scattered hyperintensity on T1-weighted image and isointensity or hypointensity on T2-weighted image (C-D); on FLAIR imaging, the intimal flap (E, right arrow) and thrombi (E, left arrow) in the dilated lumen of BA are seen. **F)** Brain computed tomography angiography (CTA) depicts a severe and fusiform enlargement in upper and middle portion of BA with eccentric filling defects extended to the top of the BA, followed by an irregular and slight dilatation at its proximal part. **G-J)** Digital subtraction angiography (DSA) demonstrates double lumen of the BA in the coronal section (G), retention of contrast medium in the venous and the later period of arterial phases (H), bilaterally tenuous posterior cerebral arteries as well as strong posterior inferior cerebellar arteries on VAs angiography, and bilaterally strong posterior communicating arteries providing perfusion to its distributional areas via posterior cerebral arteries (I-J).

(Figure 1H). Angiograms of VAs revealed tenuous posterior cerebral arteries as well as strong posterior inferior cerebellar arteries, and angiograms of internal carotid artery (ICA) showed bilaterally strong posterior communicating arteries providing perfusion to its distributional areas via posterior cerebral arteries (Figure 1 I-J). The imaging results were consistent with unruptured dissection of the BA. Routine laboratory parameters such as blood cell counts, erythrocyte sedimentation ratio, rheumatoid factor, C-reactive protein, prothrombin time (PT), actived partial thromboplastin time (APTT) were within the normal range.

Therapeutic Managements

The patient was first observed conservatively, but the follow-up DSA examination obtained 3 months later, revealed a further enlargement in the proximal portion of the BA dissection which had extended to the left VA, along with stenosis in its upper portion with a newly discovered tortuous configuration compared with previous angiography (Figure 2A, B). Therefore, we decided to perform coil embolization to occlude BA without performing a balloon test occlusion at first. Proper working angle was determined based on immediate DSA. Microcatheter (Echelon™-14) was navigated into the BA dissection with the guide of a microguidewire (SilverSpeed™-14; ev3 Endovascular Inc.). Next, MicroPlex18 20 mm × 50 cm was deployed as an initial framing coil to obtain stable frame (Figure 2C), and then five other coils, MicroPlex18 18 mm × 44 cm, 16 mm × 39 cm, 16 mm × 39 cm, 14 mm × 34 cm, 13 mm × 32 cm were packed in turn to embolize the artery. Postembolization angiographies showed no filled aneurysm image of aneurysm in angiogram of VA (Figure 2D). Bilateral ICA angiography displayed compensative perfusion from posterior communicating arteries via posterior cerebral arteries (Figure 2E, F).

Outcome

During treatment, the patient had an excellent neurological outcome without any deficit. No complication was seen after coil embolization. Follow-up DSA 6 months later demonstrated complete obliteration of the dissection by showing a large signal void in the BA area (Figure 3A, B), compensative perfusion from strong posterior inferior cerebellar arteries and posterior communicating arteries via posterior cerebral arteries and extensive collateral circulation in the peripheral area of the occluded BA (Figure 3C, D).

DISCUSSION

Dissections involving BA generally originates from dissection

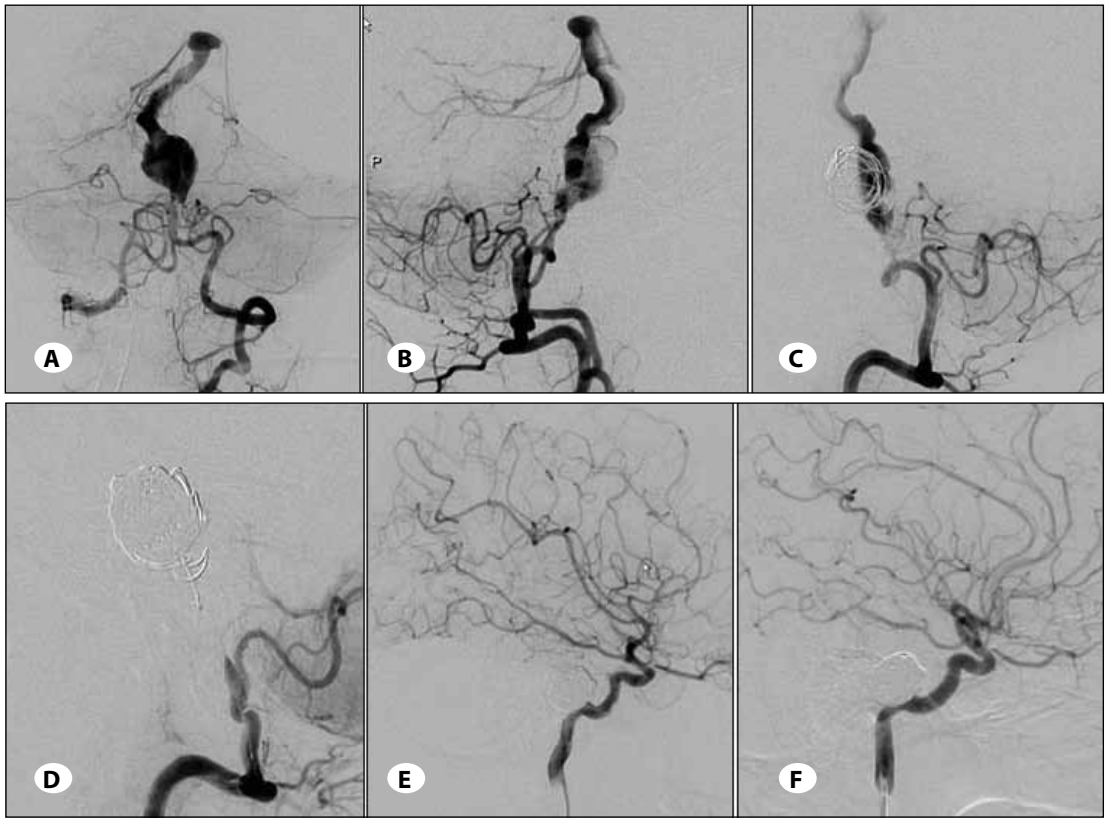

Figure 2: Coil embolization: **A, B)** DSA reveals more remarkable enlargement in the proximal portion of BA which have extended to left VA; **C)** MicroPlex18 20 mm × 50 cm is deployed as an initial framing coil to obtain stable frame; **D)** after five other coils have been packed in turn to embolize the artery, angiographies show no filled aneurysm image on left VA angiography; **E, F)** angiogram of bilateral internal carotid arteries shows good compensative perfusion from posterior communicating arteries via posterior cerebral arteries.

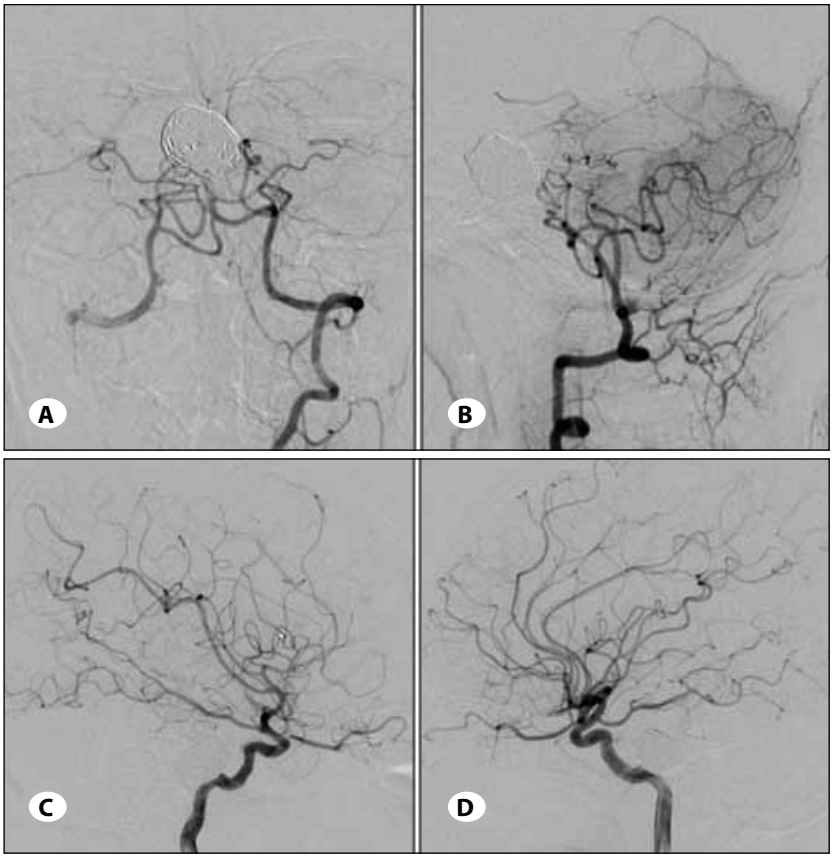

Figure 3: Postembolization examination: **A,B)** DSA demonstrates complete obliteration of the dissection by showing a large signal void in the basilar artery area; **C, D)** good compensative perfusion from strong posterior inferior cerebellar arteries and posterior communicating arteries via posterior cerebral arteries and extensive collateral circulation in the peripheral area of the occluded BA.

of vertebral arteries (VA) with upwards extension. Dissections of the BA with subsequent progression to involve VAs are extremely rare (2,14,21). BA dissection in patients over the age of 60 years tends to be of atherosclerotic in nature (5). Apart from atherosclerois, various causes such as trauma, fibromuscular dysplasia, sepsis, infective endocarditis, rheumatoid arthritis and hypercoagulability can also lead to dissection (5,15,22). Dissection in young adults below 18 years are extremely rare.

The radiological features of the BA dissection are rather complicated. They may show fusiform dilatation with proximal or distal stenosis or a double lumen in the coronal section or retention of contrast medium in the venous phase. Presence of double lumen is thought to be the most reliable imaging for diagnosis (20,24). However, this finding is seen in only about 13% of cases (11). These features along with a detectable intimal flap on FLAIR imaging of MRI is highly characteristics of a dissection (5,25). All these findings were present in our case.

Clinical manifestations of the BA dissection are diverse, headache being the most common early symptom (7). However, growing dissections leads to two clinical scenarios based on two typical pathological features (2,25). In those cases where dissection occurs between intima elastic lamina and media, a growing intramural thrombosis might lead to stenosis or obliteration of BA leading to brain stem infarct, In the second case scenario where dissection involves subadventitial tissue, fatal subarachnoid hemorrhage may occur due to the rupture of the vessel wall. Thus the clinical manifestations of the BA dissection is mainly, if not fully, depend on the pathogenetic plane of dissection (5,20).

In this article, we describe a case of BA dissection that was detected accidentally. Imaging examinations confirmed the definitive diagnosis of dissection of the BA. Serial DSA showed that the dissection grew in the subadventitial plane to reach a maximum size of 28 mm in length, 8.6 mm maximum in anteroposterior diameter and 5.2 mm maximum in transverse diameter. The patient, however, remained asymptomatic despite the size of the lesion.

In our report, the patient was initially treated with conservation. He was subsequently treated with coil embolization to occlude BA due to uncontrollable expansion of the lesion. Endovascular intervention as a treatment for VA dissection has been performed previously to prevent the occurrence of life-threatening rupture (8,9,18). DSA revealed a large BA dissection with a fragile wall. Little is still known about the ideal therapeutic measures for the BA dissection, so conversative treatment was performed first on a clinical basis (7,14,24). However, having observed the continually downward extension and dilatation of the lesion, coil embolization was inevitable to avoid potentially serious consequences caused by fatal rupture.

Other modalities to treat BA dissection mainly include craniotomy (such as wrapping to reinforce the dissected

wall and arterial reconstruction by direct surgical ligation or clipping) and endovascular treatment (such as embolization of parent vessels to reduce or reverse blood flow) (1,21,22). Surgical options are still limited because they might result in potential complications. Kitanaka et al. reported serious postoperative complications in 29% of cases, and the study by Mizutani et al. showed a similar high complication rate of 31.3% (6,10,12,13,19).

To date, endovascular approach of the BA dissection frequently relies upon the occlusion of the VA(s), which can be performed as selective unilateral occlusion of VA to reduce the blood pressure, and in turn, minimize the risk of re-bleeding, or perform a bilateral occlusion of VAs to induce flow reversal (8,16,21). However, there are several limitations to both these approaches. On one hand, growth of the lesion might be observed due to incomplete obstruction of unilateral occlusion. On the other hand, bilateral intervention may cause disastrous consequences if the thrombus in the dissected lumen collapses the true lumen, resulting in standstill of retrograde flow. Based on a retrospective review of the literature focusing on spontaneous BA occlusion, the authors were aware of several cases with favorable outcomes following complete obliteration of BA (4,17). Our patient therefore received coil embolization to completely obstruct the BA. Routine DSA obtained 6 months postoperatively showed a large signal void which implied complete obliteration of the dissection, good compensative perfusion from strong posterior inferior cerebellar arteries and posterior communicating arteries via posterior cerebral arteries, and extensive collateral circulation in the peripheral area of the occluded BA.

For young patients with isolated dissection of the BA, coil embolization to occlude BA at the bottom of the aneurysm might be a tolerable and effective approach.

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