Dissection of Bilateral Intracranial Vertebral Artery with Basilar Artery Involvement: A Case Report of a Patient Free from Neurological Deficits

Noriko Hagiwara¹,2, Masahiro Kamouchi¹, Tooru Inoue¹, Setsuro Ibayashi³, Mitsuo Iida² and Yasushi Okada¹

Abstract

We report a patient with dissection of the bilateral intracranial vertebral artery (VA) that did not present any symptoms other than occipital headache, which was probably associated with sleeping overnight in a car seat with unsteady head position. Although cerebral angiography revealed extensive dissection of the bilateral VA after branching of the posterior inferior cerebral artery, retrograde flow to the basilar artery (BA) via the right posterior communicating artery contributed to preserved posterior circulation. These findings indicate that even in patients without neurological deficits, the involvement of BA cannot be excluded and that accurate evaluation using radiological techniques should be considered.

Key words: dissection, vertebral artery, basilar artery, headache, anticoagulation, cerebral angiography

Introduction

Dissection of the intracranial vertebral artery (VA), which has previously been considered rare, is now recognized as a primary pathogenesis and has been drawing increased attention among young and middle-aged patients (1-5). In general, involvement of the basilar artery (BA), typically occurring via antegrade progression of VA dissection, is a serious condition that greatly affects the clinical course and prognosis (1-5). Here, we describe a rare case with dissection of the bilateral intracranial VA accompanied by BA involvement, who did not present any neurological deficits. Thus, it is critical to correctly diagnose extension of vertebrobasilar artery dissection even in patients who present pain alone or only minor neurological symptoms.

Case Report

A 48-year-old man without vascular risk factors was admitted to our hospital complaining of a pain in the occipital region. Two weeks earlier, he had slept overnight in a car seat with his head tilted backwards. Bilateral posterior headache was evident upon awakening (day 1) and rapidly worsened after habitual exercises including neck flexion and rotation. The pain was sharp and throbbing in nature, spread to the shoulder. He had never experienced such a severe headache. Although he was prescribed with an analgesic (diclofenac potassium, 75 mg/day) at a local clinic, the pain hardly improved and he was referred to our hospital for further evaluation.

Upon admission (day 7), his blood pressure was 186/98 mmHg and his pulse rate was regular at 77 beats per minute. He was alert and had no neurological deficits. A physical examination revealed no abnormal findings other than hypertension. Findings of blood tests, electrocardiography, chest radiography and transthoracic echocardiography were all normal.

Cranial computed tomography (CT) and magnetic resonance imaging (MRI) including diffusion-weighted image upon admission revealed no abnormalities other than hypertension. Findings of blood tests, electrocardiography, chest radiography and transthoracic echocardiography were all normal. Cranial computed tomography (CT) and magnetic resonance imaging (MRI) including diffusion-weighted image upon admission revealed no abnormalities in the brain parenchyma. However, fat-suppressed T1-weighted images showed an intimal flap of the left intracranial VA and visualized flow within the true and false lumens (Fig. 1A), indi-

¹Department of Cerebrovascular Disease, Cerebrovascular Center and Clinical Research Institute, National Hospital Organization Kyushu Medical Center, Fukuoka and ²Department of Medicine and Clinical Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka

Received for publication April 4, 2007; Accepted for publication June 7, 2007

Correspondence to Dr. Noriko Hagiwara, hagiwara@intmed2.med.kyushu-u.ac.jp
Figure 1. Magnetic resonance imaging and MR angiography upon admission (A-C) and day 21 (D, E). (A) Fat-suppressed T1-weighted image shows an intimal flap of the left VA (arrow) and flow within true and false lumens. (B) An intense eccentric crescentic signal surrounding the narrow lumen of the right VA (arrow) was also documented. (C) MR angiography upon admission shows disappearance of signal flow with filling defects in the bilateral VA and basilar artery (BA). (D) On day 21, the bilateral VA was recanalized although the left VA was markedly stenotic and indistinct compared with the right VA (arrowheads). (E) Three-dimensional MRA on day 21 revealed severe stenosis of the left VA (arrow) and wall irregularity of the right VA and the lower BA (arrowheads).

cating dissection. An intense crescentic signal surrounding the narrowed lumen of the right intracranial VA (Fig. 1B), suggesting an intraluminal hematoma due to dissection, was also demonstrated. Magnetic resonance angiography (MRA) showed a partially obscured flow signal at the vertebrobasilar junction with a filling defect that was finally absent in the lower BA (Fig. 1C). This was confirmed by subsequent cerebral angiography on day 9 that revealed tapering occlusion of the left VA immediately distal to the origin of the posterior inferior cerebellar artery (PICA) branch (Fig. 2A) and dilatation distal to the tapering lesion in the right VA (Fig. 2B). The BA and main branches were visible via the right posterior communicating artery from the anterior circulation (Fig. 2C, D). However, retrograde flow into BA could not be observed from the proximal site of the anterior inferior cerebellar artery (AICA) branching, which suggested that VA dissection extended to the lower BA. Flow velocity examined by Doppler sonography was reduced in the bilateral VA, indicating occlusion after branching of the PICA. Based on these findings, he was diagnosed as having bilateral dissections of the intracranial VA. Cerebral blood flow (CBF) in the cerebellum visualized by single photon emission computed tomography (SPECT) imaging using $^{99m}$Tc-ECD was barely preserved at rest. However, vasoreactivity during acetazolamide challenge was negative as the CBF was decreased by 30%, suggesting the steal phenomenon.

Based on the above findings, we considered him to have risk for ischemic complication such as brain infarction. To prevent the progression of brain stem infarction, we started continuous intravenous infusion of sodium heparin at 10,000 units per day for 7 days followed by warfarin. His pain was gradually improved and completely disappeared on day 14.
Figure 2. Cerebral angiography on day 2 (A-D) and day 37 (E-H). (A) Left vertebral angiography on anteroposterior view shows string sign of the left VA after branching of the posterior inferior cerebellar artery (asterisks) and finally occlusion (arrow). (B) Right vertebral angiography shows dilatation of the BA (arrow) distal to the tapering lesion of the right VA (arrowheads). (C, D) Right carotid angiography shows retrograde filling to the BA and its main branches via the right posterior communicating artery on anteroposterior view (arrow) and lateral view (arrowheads), respectively. (E) On day 37, the left VA is recanalized to the union with poor opacification of the BA. (F) The occlusion of the right VA was resolved although its wall irregularity still remained. The BA lumen was reestablished with antegrade flow from the right VA. (G, H) Retrograde flow into BA disappeared on anteroposterior and lateral views, respectively.

MRA on day 21 showed recanalization of the bilateral VA to the union (Fig. 1D). On three-dimensional MRA, high-grade stenosis of the left VA still remained and wall irregularity of the right VA and the lower BA was still present with slight dilatant change (Fig. 1E). Repeated angiography on day 37 showed antegrade flow of the BA, although the “pearl and string” sign persisted in the bilateral VA (Fig. 2E, F). Retrograde flow into BA from anterior circulation disappeared (Fig. 2G, H). Cerebral hemodynamics on SPECT images showed improved vasoreactivity to acetazolamide with 10% increased CBF from rest time. With improvement of pain, his blood pressure level returned to normal range and was well-controlled during hospitalization without an antihypertensive. The patient did not develop any neurological deficits during the course of treatment.

Discussion

The main aspects of this report are that a patient developed dissection of the bilateral intracranial VA with no neurological deficits due to the collateral filling to the BA, and further that accurate evaluation of the cerebral hemodynamic process is necessary to select the appropriate therapeutic strategy for each patient.

The clinical presentations of intracranial VA dissection are quite varied, depending on the individual. In particular, involvement of the BA is generally a determinant factor for the severity of brain ischemia (1-5). However, the present patient presented with headache and no neurological deficits, since the brain stem was supplied with sufficient retrograde flow via the posterior communicating artery from the anterior circulation.

Evident factors in the diagnosis of arterial dissection in this patient were: occurrence in middle-age, tapering occlusion and later recanalization of VA within a short period, intimal flap and crescent sign on MRI, and lack of atherosclerosis and cardioembolic sources (1). It has been reported that headache may precede other manifestations of VA dissection for several hours to days (6), and that occasionally the stroke is not complete at its onset but proceeds in a stepwise fashion (7). Thus in this case, the 7-day history of occipital headache after sleeping in a car seat might have been a warning sign of severe VA dissection. Subsequent exercises of the neck repeatedly triggered off deterioration of pain probably by progression of the dissection. Since the VA near the PICA branching is often compressed by neighboring structures including bones, muscles, ligaments, and fascial tissues (1, 5), neck rotation and flexion may give physi-
cal stretch to the fragile portion of the VA. Such mechanical stimulation could enhance extension of the intimal tear of arterial wall and enlarge intramural bleeding of the dissecting VA. In the present case, we could not confirm the initial dissecting site of VA because the bilateral VA was already been involved at the time of hospitalization. Considering the limited lesion around the vertebrobasilar junction and the difference of the recanalizing process, we speculated that the left VA dissection might have extended into the contralateral VA with the lower BA involvement. However, another possibility that the bilateral VA might have been dissected simultaneously could not be excluded completely.

Intracranial extension of the dissection is usually regarded as a contraindication of anticoagulation, considering the potential risk of subarachnoid hemorrhage (1, 8). However, with this patient we ventured to choose anticoagulation to prevent the progression of brain stem ischemia under appropriate control of blood pressure and radiological examination to exclude aneurysmal changes of the dissected arteries. As the result, the unusual hemodynamics in posterior circulation returned to normal satisfactorily with radiological confirmation.

In conclusion, when managing patients with intracranial VA dissection, involvement of the BA cannot be excluded even in cases who are asymptomatic except for headache or minor symptoms. This case also indicates that early detection, which leads to early treatment, may significantly reduce serious neurological complications. Further studies are needed to establish the precise management of intracranial VA dissection.

References