

Partially Thrombosed Vertebral Artery Dissecting Aneurysm Presenting as Delayed Bulbar Compression after Lateral Medullary Infarction

Yuji Kato¹, Fumitaka Yamane², Takeshi Hayashi¹, Shinya Kohyama², Shoichiro Ishihara², Akira Uchino³ and Norio Tanahashi¹

Abstract

A 48-year-old man experienced lateral medullary infarction resulting from spontaneous vertebral artery (VA) dissection. Minimal fusiform dilatation was noted on basi-parallel anatomic scanning-magnetic resonance imaging; therefore, the patient was treated conservatively. Eight months later, he experienced deterioration of dysphagia and the onset of gait ataxia. Repeated imaging studies showed enlargement of the VA aneurysm with bulbar compression. Parent artery occlusion on the proximal side of the VA affected by the dissection relieved the patient's symptoms. Although the majority of dissected lesions stabilize within a few months, studies with longer observation periods and more frequent neuroimaging examinations are required.

Key words: vertebral artery dissecting aneurysm, lateral medullary infarction, medulla oblongata compression, basi-parallel anatomic scanning

(Intern Med 54: 427-429, 2015)

(DOI: 10.2169/internalmedicine.54.3347)

Introduction

Partially thrombosed vertebral artery (VA) aneurysms tend to become very large and thus induce symptoms of brainstem compression. However, symptomatic bulbar compression by VA aneurysms is rare, with only a few cases having been reported (1-4). Furthermore, to our knowledge, there are no previous cases of delayed bulbar compression due to a slowly growing VA dissecting aneurysm following lateral medullary infarction. We herein report a case of a VA dissecting aneurysm successfully treated with parent artery occlusion (PAO).

Case Report

A healthy 48-year-old man noticed left-side neck tenderness. Five days later, he experienced numbness of the right hand with vertigo and dysarthria. He was admitted to our

hospital, where the initial neurological examination findings were suggestive of Wallenberg syndrome. Diffusion-weighted magnetic resonance (MR) imaging performed on admission showed acute infarction in the left posterolateral medulla (Fig. 1A), and MR angiography indicated an indistinct flow in the left VA (Fig. 1B). Meanwhile, basi-parallel anatomic scanning (BPAS)-MR imaging (5) performed on day 6 showed fusiform dilatation of the outer contour at the narrowing portion of the left VA (Fig. 1C), with the source MR angiographic image showing a double lumen in the left VA (Fig. 1D). These findings were indicative of VA dissection. The patient was treated conservatively without antiplatelet therapy, as the MR and BPAS-MR imaging findings on day 27 indicated a stable size and morphology. The patient was subsequently discharged on day 34 and resumed a normal lifestyle after six months of rehabilitation.

Eight months after the initial medullary infarction, the patient experienced deterioration of dysphagia and the onset of gait ataxia. His blood pressure remained well controlled (<

¹Department of Neurology and Cerebrovascular Medicine, Saitama International Medical Center, Saitama Medical University, Japan, ²Department of Endovascular Neurosurgery, Saitama International Medical Center, Saitama Medical University, Japan and ³Department of Diagnostic Radiology, Saitama International Medical Center, Saitama Medical University, Japan

Received for publication May 26, 2014; Accepted for publication July 9, 2014

Correspondence to Dr. Yuji Kato, yujik@saitama-med.ac.jp

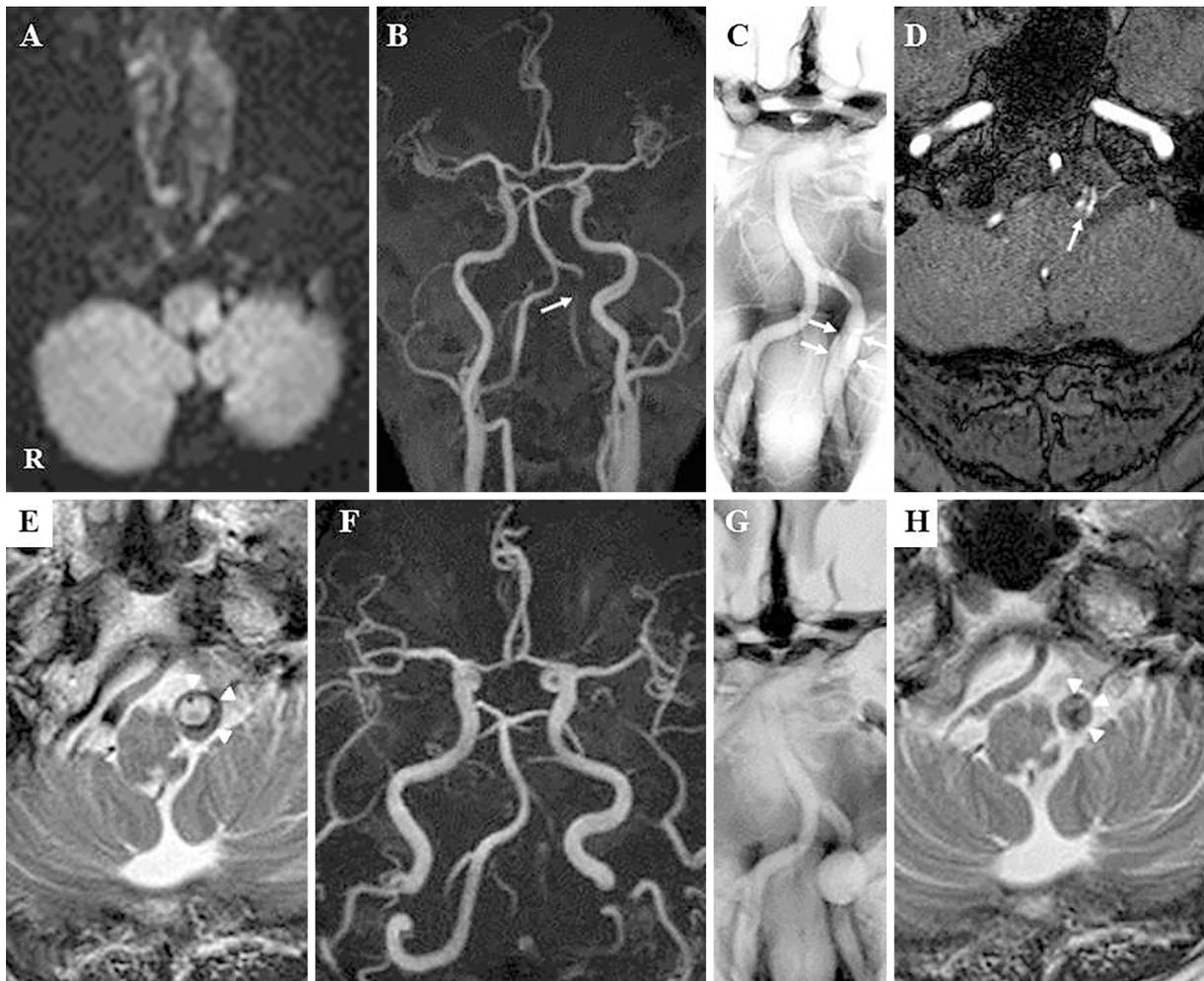


Figure 1. A: Diffusion-weighted MR image obtained on admission showing a high-intensity lesion in the left posterolateral medulla oblongata. B: MR angiography image showing an indistinct flow in the left VA (arrow). C: BPAS-MR image obtained on day 6 showing fusiform dilatation of the outer contour in the narrowed segment of the left VA (arrows). D: MR angiographic source image revealing the double lumen in the left VA (arrow). E: T2-weighted MR image obtained eight months after onset showing a heterogeneous mass associated with a low-intensity signal rim (10 mm in diameter) compressing the medulla oblongata adjacent to the left anterior side, suggestive of a partially thrombosed VA aneurysm (arrowheads). F: MR angiography image showing narrowing of the left VA. G: BPAS-MR image demonstrating obvious growth of the aneurysm. H: T2-weighted MR image obtained six months after PAO showing a decrease in the size of the aneurysm (arrowheads). MR: magnetic resonance, BPAS-MR: basi-parallel anatomic scanning-MR, VA: vertebral artery

140/90 mmHg) with imidapril during this period. T2-weighted MR imaging suggested enlargement of the partially thrombosed VA aneurysm compressing the medulla from the left anterior side (Fig. 1E). Although the findings of MR angiography showed narrowing of the left VA (Fig. 1F), BPAS-MR imaging performed at that time demonstrated significant aneurysmal enlargement at the site of VA dissection (Fig. 1G). Due to growth of the lesion and deterioration of the patient's neurological symptom, we performed PAO at the proximal region of dissection in order to preserve the perforators from the aneurysmal dome. A partially thrombosed aneurysm was successfully occluded using platinum coils (Fig. 2). Thereafter, the patient's clinical symptoms gradually improved, and MR imaging, including

BPAS, performed six months after the procedure revealed a decrease in the size of the aneurysm (Fig. 1H).

Discussion

It remains unclear how long dynamic changes in dissecting aneurysms persist after symptom onset. Studies have indicated that aneurysm enlargement is observed only in the relatively early phase, at three weeks in most cases (6, 7). Furthermore, the degree of fusiform dilation usually remains unchanged, even resolving in some patients (7). The course of the present case, on the other hand, was not in line with that of previous reports. It is not possible to precisely determine how long the lesion should be monitored based on the

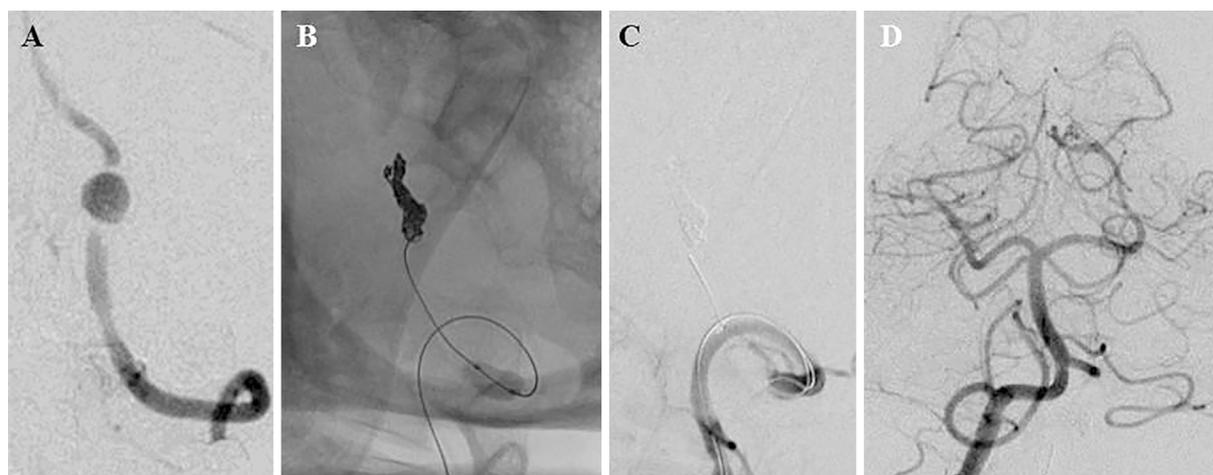


Figure 2. A: Preoperative left VA angiogram showing the aneurysm. B: Radiograph revealing the shape of the coil mass packed into the aneurysm. C: Postoperative left VA angiogram showing complete obliteration of the aneurysm. D: Postoperative right VA angiogram showing retrograde filling to the distal end of the left VA. VA: vertebral artery

first angiographic manifestations. Although the majority of dissected lesions stabilize within a few months (7), a longer observation period and more frequent neuroimaging studies may be required in some cases.

The pathogenesis of aneurysms growth remains poorly elucidated. The proposed mechanism accounting for the slow growth of VA aneurysms involves the “two-stage dissection theory,” in which the initial insult is macroscopic arterial dissection, a process later coupled with the development of multiple sites of mural microdissection secondarily to underlying weakness of the internal elastic lamina (8). In the present case, thrombus formation at the false lumen may have resulted in parent artery narrowing as well as the formation of the mass causing bulbar compression. We confirmed the patient’s vascular condition using noninvasive BPAS-MR imaging (5), which can be employed to detect the outer contour of aneurysms even if the VA is occluded. Because aneurysms can grow even after the occurrence of complete thrombotic changes (2), providing careful follow-up, including BPAS-MR imaging, is necessary.

In summary, we herein reported a case of a partially thrombosed VA aneurysm compressing the medulla oblongata. Slow growth of the aneurysm after lateral medullary infarction may cause symptoms owing to bulbar compression. The administration of endovascular treatment, such as PAO, should be considered as soon as possible in cases of unruptured VA dissection associated with growing aneurysmal dilation during the follow-up period.

The authors state that they have no Conflict of Interest (COI).

References

1. Bohl J, Fenske A, Reulen HJ, Schindler E. Giant aneurysm of the vertebral artery causing compression of the lower medulla oblongata. *J Neurol* **214**: 289-293, 1977.
2. Aoki N, Sakai T, Oikawa A, Takizawa T. Giant unruptured aneurysm of the vertebral artery presenting with rapidly progressing bulbar compression--case report. *Neurol Med Chir (Tokyo)* **37**: 907-910, 1997.
3. Sato K, Ezura M, Takahashi A, Yoshimoto T. Fusiform aneurysm of the vertebral artery presenting hemifacial spasm treated by intravascular embolization: case report. *Surg Neurol* **56**: 52-55, 2001.
4. Shiraishi S, Fujimura M, Kon H, et al. Thrombosed vertebral artery aneurysm presenting with hemorrhage and bulbar compression: report of two cases. *Clin Neurol Neurosurg* **107**: 123-127, 2005.
5. Nagahata M, Abe Y, Ono S, Hosoya T, Uno S. Surface appearance of the vertebrobasilar artery revealed on basiparallel anatomic scanning (BPAS)-MR imaging: its role for brain MR examination. *AJNR Am J Neuroradiol* **26**: 2508-2513, 2005.
6. Kitanaka C, Tanaka J, Kuwahara M, et al. Nonsurgical treatment of unruptured intracranial vertebral artery dissection with serial follow-up angiography. *J Neurosurg* **80**: 667-674, 1994.
7. Nakagawa K, Touho H, Morisako T, et al. Long-term follow-up study of unruptured vertebral artery dissection: clinical outcomes and serial angiographic findings. *J Neurosurg* **93**: 19-25, 2000.
8. Mizutani T. A fatal, chronically growing basilar artery: a new type of dissecting aneurysm. *J Neurosurg* **84**: 962-971, 1996.