Case Report

A Case of Vertebral Artery Dissection Associated with Morning Blood Pressure Surge

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We report a case of a middle-aged man who suffered a cerebral infarction resulting from dissection of a vertebral artery associated with morning blood pressure surge. A 56-year-old man was transferred to our hospital with dizziness and vomiting in the early morning on a cold day in winter. He reported that he had been standing in front of the sink after bathing when he suddenly felt dizzy and fell down. He did not lose consciousness, and by the time he reached the hospital by ambulance, his dizziness had subsided, but he complained of severe headache and vomited 3 times. On admission, he was alert, and there were no neurological or radiological abnormalities (CT, MR angiography) in the brain. However, infarction in the left cerebellar hemisphere was detected by brain MRI on the 5th day of hospitalization. String sign of the left vertebral artery was noted by angiography, confirming the diagnosis of dissection of the left vertebral artery. Ambulatory blood pressure monitoring was performed after discharge. Although the mean 24-h blood pressure was in the normal range, a marked morning blood pressure rise was observed. We speculated that the acute rise of blood pressure in the early morning might have contributed to the dissection of the vertebral artery. (*Hypertens Res* 2005; 28: 847–851)

***Key Words*: vertebral artery dissection, morning blood pressure surge, cerebellar infarction**

Introduction

Intracranial vertebrobasilar artery dissection is a rare cause of ischemic stroke, being seen in about 0.5–3 cases per year (*1*). Major clinical complications of vertebral artery dissection include subarachnoid hemorrhage and cerebral ischemia. The most common symptom is a severe headache, which usually occurs immediately before other clinical manifestations. Several predisposing factors have been reported (*2*), but there is controversy as to whether hypertension is one of them (*3*). We report a case of a middle-aged man who suffered a cerebellar infarction due to dissection of a vertebral artery which might be caused in association with morning blood pressure (BP) surge.

Case History

A 56-year-old man was admitted to the Shioya General Hospital with dizziness and vomiting. He had been diagnosed with arrhythmia several years previously, but otherwise he did not have any prior cardiovascular events. His self-monitored BP was about 140/70 mmHg. There was no family history of hypertension or stroke. He smoked 1 pack/day and

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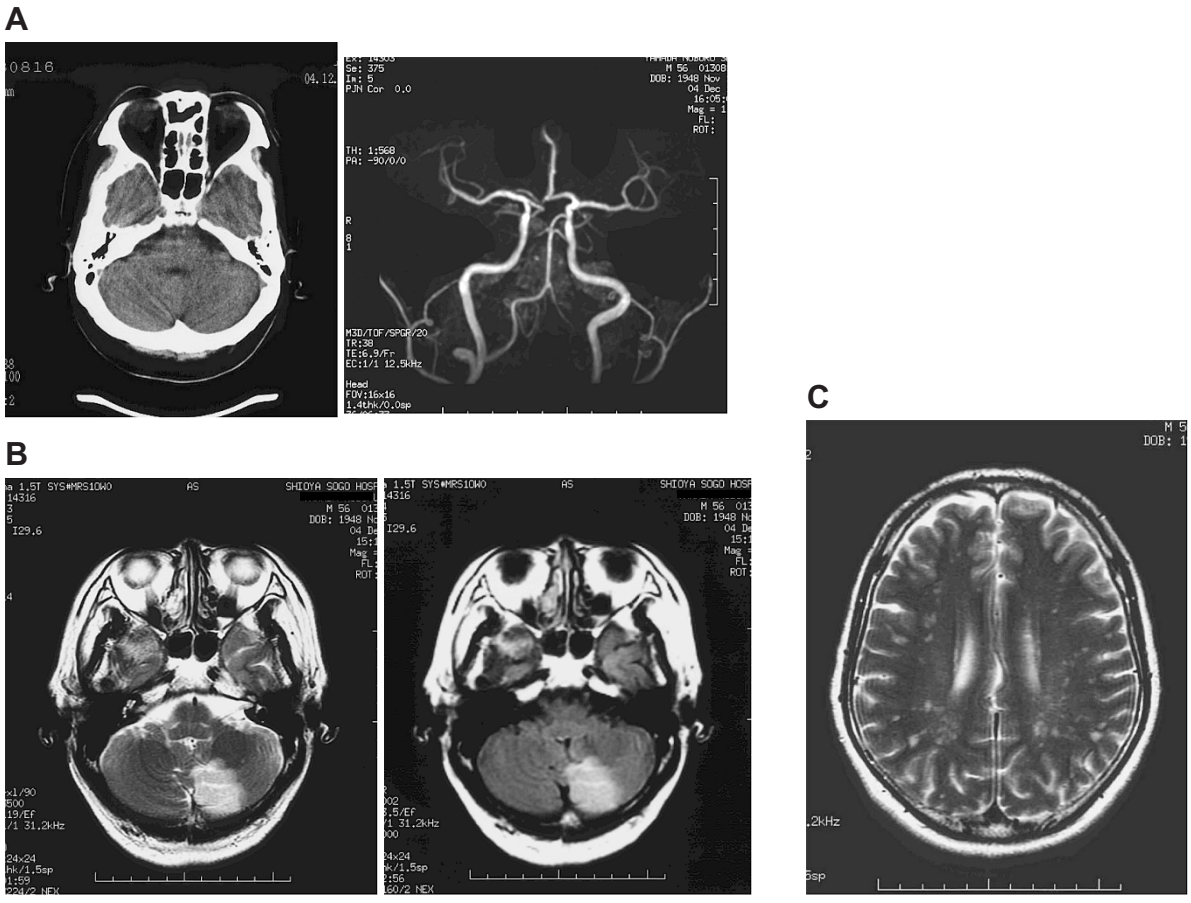

Fig. 1. *A: Brain CT (left panel) and MR angiogram (right panel) on admission, showing no abnormalities. B:* T_2*-weighted imaging (left panel) and FLAIR imaging (right panel) of MRI on December 20 demonstrated an area of high intensity in the left cerebellar hemisphere. C:* T_2*-weighted imaging of deep white matter lesion. There was no silent cerebral infarction or periventricular hyperintensity, but there were several hyper-intense punctuates in the deep white matter.*

drank alcohol equivalent to about 60 g/day of ethanol. In April 2004 he experienced a severe occipital headache upon abruptly rotating his neck backward while working on a fork-lift. In the first week of December 2004, he was very tired and suffered from insomnia. After waking up in the early morning on December 15, he bathed, drank coffee and smoked, and then was standing in front of the sink when he suddenly felt dizzy and fell down for a few minutes, although he did not lose consciousness. He had urinary incontinence. His family called an ambulance, but his dizziness had subsided when the ambulance arrived. On the way to the hospital, he complained of a severe headache, and vomited 3 times.

On admission, his BP and pulse rate (PR) were 164/90 mmHg and 46–54/min, respectively. He was alert and there were no neurological abnormalities as assessed by a neurosurgeon. Brain CT did not show any bleeding or infarction (Fig. 1A), and MR angiography did not show any significant narrowing or occlusion of the cerebral arteries. His plasma cholesterol was 244 mg/dl, and his triglycerides were 381 mg/dl; otherwise, no abnormal findings were noted. An ECG showed sinus bradycardia (heart rate 46 beats/min), frequent premature supra-ventricular contractions and non-specific ST-T changes. Ambulatory ECG monitoring also showed frequent supraventricular premature contractions (2,130/day), but atrial fibrillation was not detected. During the first week of hospitalization, he received medication for insomnia, headache, and nausea. However, on December 20, infarction of the left cerebellar hemisphere was found by brain MRI (Fig. 1B) without the presentation of any symptoms. There was no silent cerebral infarct or periventricular hyperintensity, but there were several hyper-intense punctuates in the deep white matter on T_2-weighted images (Fig. 1C). Cerebral angiography revealed an angiographic string sign of the left vertebral artery, and we confirmed the diagnosis of vertebral artery dissection (Fig. 2). A T_1-weighted image on December 20 did not show a double lumen sign or a high intensity area indicating the presence of dissection (Fig. 3).

His discharge from the hospital was postponed for 1 week and he was treated conservatively again, but no disability or complications were seen. After leaving the hospital, he under-

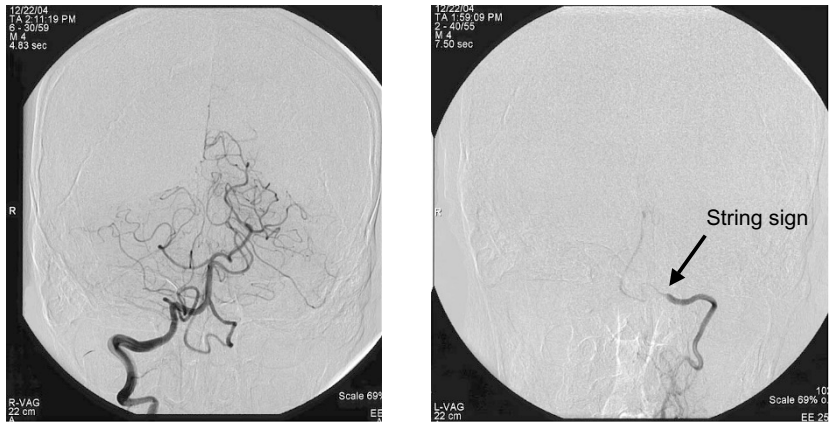

Fig. 2. *Right (right panel) and left vertebral angiogram (left panel) revealed the string sign of the left posterior inferior cerebellar artery.*

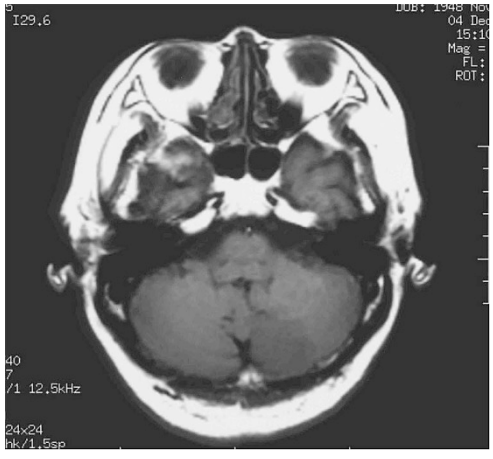

Fig. 3. *The* T_1*-weighted image on December 20 did not show the double lumen sign; however, a high intensity area indicated the presence of dissection.*

went ambulatory BP monitoring (ABPM). Although the mean 24-h BP was in the normal range (126/79 mmHg), nocturnal hypotension, morning BP surge, and daytime hypertension were noted (Fig. 4). Supine and standing BP and PR measured every 1 min are shown below. There was no significant orthostatic hypotension or hypertension. Cervical spondylosis was excluded by cervical X ray (Fig. 5).

Supine	Standing
152/83 mmHg, 54/min	130/85 mmHg, 72/min
137/79 mmHg, 54/min	136/89 mmHg, 74/min
134/80 mmHg, 59/min	134/93 mmHg, 73/min
132/76 mmHg, 53/min	135/90 mmHg, 72/min

Discussion

We encountered a case of cerebellar infarction due to the dissection of a vertebral artery. The onset of disease occurred in the early morning, and the patient showed an early morning BP surge upon ABPM performed after leaving the hospital. We speculated that a sudden increase of BP in the morning was the trigger of the dissection of the vertebral artery.

Pathogenesis of Dissection

The histopathological findings of vertebral artery dissection are similar to those observed in other arteries. In addition to the intradural segment of the vertebral artery having a thin media and adventitia with few elastic fibers, dissections of the intradural artery readily occur once a small segment of the intima is injured, and the injury extends along the artery (*4*). In addition to the weakness of the vessels, mechanical stress on the intima could initiate dissection.

In the present case, a severe headache upon rotation of the neck occurred in April 2004, and vulnerability of the vertebral artery might thus have existed prior to the cerebral infarction in December 2004. We speculate that an acute rise of BP in the morning could trigger the process of the vertebral artery dissection in a vessel with only a very small injury prior to the dissecting event.

As a differential diagnosis, Bow Hunter's syndrome (*5*), which is characterized by vertebral artery occlusion upon neck rotation, can be excluded, because the vertebral artery was occluded angiographically without rotation in the present case.

Relation to Atherosclerosis

Atherosclerosis is uncommon in patients with a vertebral artery dissection. However, in this case, atherosclerosis may have been present in the vertebral artery, since the patient had out-of-office hypertension (home BP) (*6*), mild hyperlipidemia, and a smoking habit (*7*). Smoking itself is independently associated with silent cerebral infarct (*8*) and atherosclerotic changes (*9*) in Japanese. In middle-aged men,

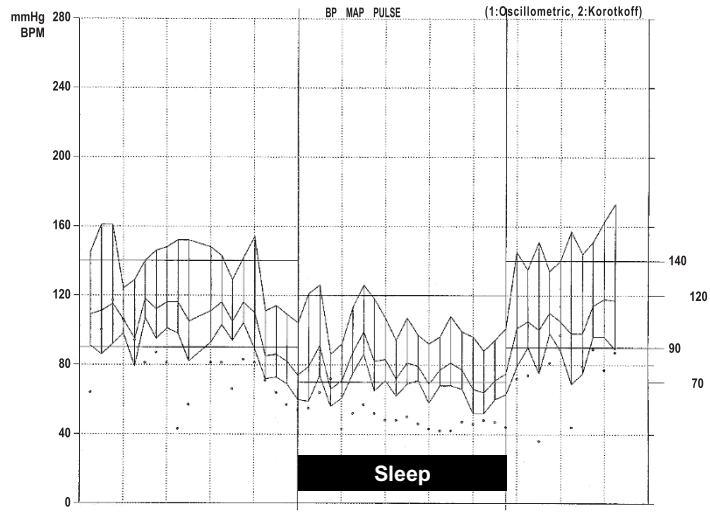

Fig. 4. *Ambulatory BP monitoring showed acute morning surge of BP just after awaking despite a normal range of 24-h average BP level (126/79 mmHg).*

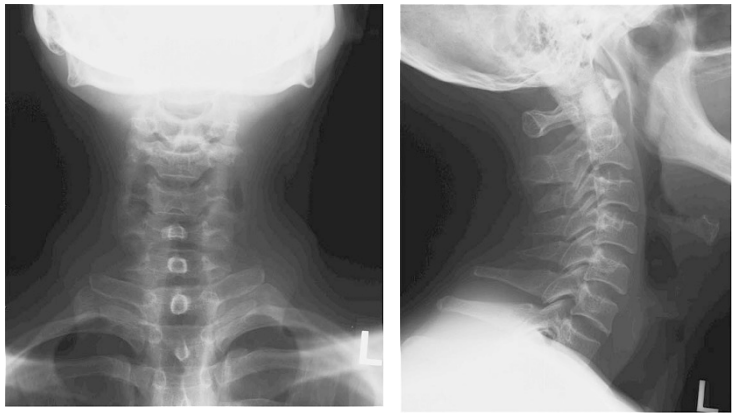

Fig. 5. *There were no findings of cervical spondylosis by cervical X ray.*

the total cholesterol level is associated with carotid atherosclerosis (*10*). Pulse wave velocity (PWV) is a highly reproducible measure (*11*) of arterial stiffness (*12*), atherosclerosis (*13*, *14*), coronary artery disease (*15*), and aortic calcification (*16*). In the present case, the brachial ankle PWV (baPWV) was in the normal range for the patient's age, ruling out severe atherosclerotic changes in the middle-sized arteries and aorta.

Relation to Morning BP Surge

Recently, early morning BP surge was suggested to be a strong risk factor for stroke (*17*) and left ventricular hypertrophy (*18*). The reported cutoff value of morning BP surge was 55 mmHg (*17*), and in the present case, the value was above this level. In this case, mental and physical stress and insomnia might have exacerbated the early morning BP surge. In addition to the intrinsic circadian BP rhythm, exposure to cold after bathing, and caffeine intake and smoking may have contributed to the sudden rise of BP in the morning. The patient's alcohol intake on the previous night might also have affected the nocturnal dipping of BP, and the subsequent morning rise of BP (*19*). Excessive alcohol intake increases the atherosclerotic cardiovascular risk associated with arterial stiffening in men even with normal BP (*20*). Abnormal circadian BP rhythm is highly reproducible, as reported previously (*21*). One limitation of this study was that there was no clear cause-effect relationship between morning rise of BP and arterial dissection.

There have been no reports in which ABPM was used to assess BP associated with vertebral artery dissection. The reported rate of masked hypertension, *i.e.*, normal BP in the office but higher BP in the ambulatory setting, is 10–15%, and masked hypertension may be associated with a risk of

cardiovascular events, as is sustained hypertension (*22*). Alternatively, higher home BP would better predict atherosclerosis (*23*). Therefore, it is possible that presence of masked hypertension which leads to the pathogenesis of vertebral artery dissection has been misdiagnosed in previous studies. A marked change in BP is also a risk for stroke (*24*).

In conclusion, we reported a case of vertebral artery dissection. The trigger for dissection was considered to be an acute rise of morning BP.

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