

Anterior Cerebral Artery Dissection Presenting Subarachnoid Hemorrhage and Cerebral Infarction

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Abstract

A 35-year-old man presented with simultaneous occurrence of subarachnoid hemorrhage (SAH) and cerebral infarction (CI) caused by anterior cerebral artery (ACA) dissection. He complained of sudden onset of left frontal headache and his symptoms progressed to consciousness disturbance and right hemiparesis. Computed tomography and magnetic resonance imaging demonstrated SAH localized in the left interhemispheric fissure and CI in the territory of the left ACA. Right carotid angiography demonstrated a long double lumen sign at the left A2 to A4 segment of the left ACA, leading to a diagnosis of the combined type of CI and SAH caused by ACA dissection. Although many surgeons have previously tried to perform endovascular treatment, we selected only medication in this case, and his neurological findings gradually improved. Only 9 cases including the present case presented with simultaneous occurrence of SAH and CI caused by ACA dissection. Many of these patients showed stenosis with dilatation of ACA on carotid angiography. The prognoses of these patients were good. However, many SAH patients with dissecting aneurysm had poor prognoses. To improve the strategy for managing ACA dissection, we need to accumulate a greater number of such cases in the future. We also recommend that angiography should be performed in the patients with ACA dissection.

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Key words: anterior cerebral artery, dissection, simultaneous, cerebral infarction, subarachnoid hemorrhage

Introduction

Infarction in the anterior cerebral artery (ACA) territory was first described in the early 20th century based on autopsy results¹. Recent studies using computed tomography (CT) and magnetic resonance

imaging (MRI) have investigated the etiologies and clinical features. Cerebral infarcts localized in the ACA territory are reported to comprise 0.5–3% of all ischemic strokes^{2,3}. Spontaneous dissecting aneurysms of the internal carotid circulation occur frequently less than those of the vertebrobasilar circulation, with more than 90% of dissecting

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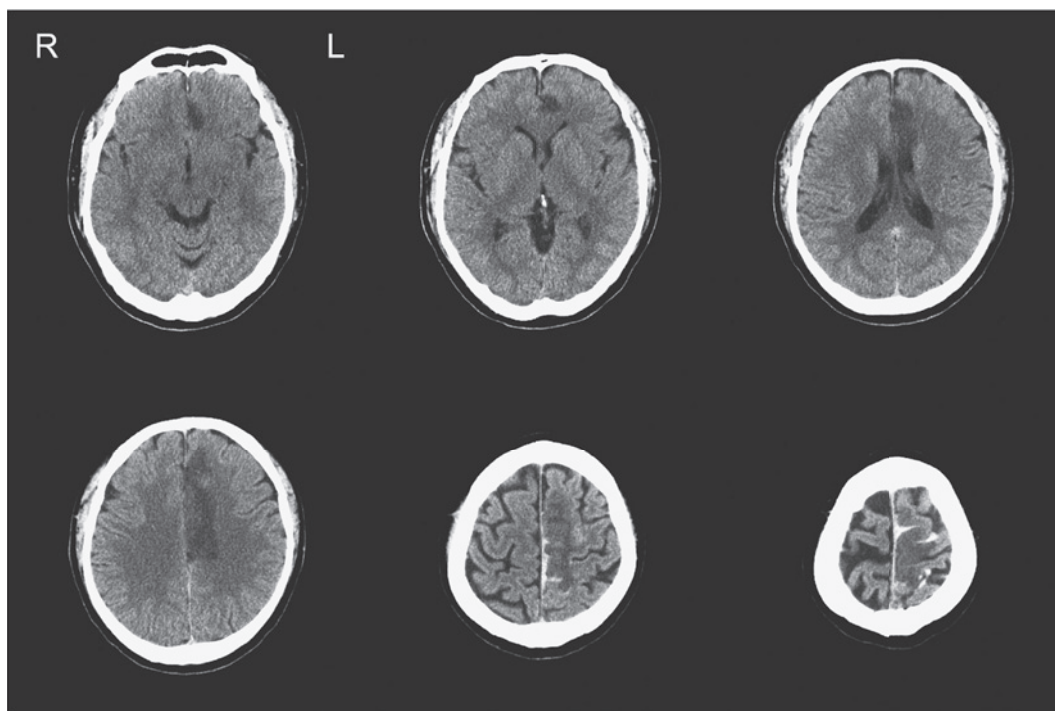


Fig. 1 Computed tomography on admission.

It is a computed tomography on admission at 9 hours after onset. High density areas were observed in the left interhemispheric fissure, and a low density area is observed in the territory of the left anterior cerebral artery.

aneurysms occurring in the vertebrobasilar circulation⁴. ACA dissection is reported to comprise 36.7% of intracranial dissections⁵. Spontaneous intracranial dissection often develops in young healthy people and is associated with severe symptom. We report a case of simultaneous cerebral infarction and subarachnoid hemorrhage (SAH) caused by dissection of the ACA. It was a rare case, but was an important case considering the management of younger stroke.

Case Report

A 35-year-old male patient complained of sudden onset of left frontal headache during work. The headache worsened gradually, and he was admitted to a hospital. After seven hours, however, his symptoms progressed to consciousness disturbance and right hemiparesis. Then he was referred to our hospital. He had a habit of smoking 30 cigarettes a day, and a history of hypertension without medication. He did not have a history of head injury.

On admission, he demonstrated aphasia, right

facial palsy, hemiparesis and sensory disturbance. The National Institute of Health Stroke Scale (NIHSS) was 20. Systolic blood pressure was 148 mmHg and diastolic blood pressure was 100 mmHg. General laboratory data were normal. Initial computed tomography at 9 hours after onset demonstrated SAH localized in the left interhemispheric fissure, and cerebral infarction in the territory of the left ACA (**Fig. 1**). In MRI fluid attenuation inversion recovery (FLAIR) image also showed high intensity signals, and T2*-weighted image showed low intensity signals in the left interhemispheric fissure (**Fig. 2**). FLAIR, T2-weighted and diffusion-weighted image (DWI) MRI showed cerebral infarct in the territory of the left ACA (**Fig. 2**). Left carotid angiography (CAG) demonstrated a long double lumen sign at the left A2 to A4 segment one day after onset (**Fig. 3**). We diagnosed the combined type of cerebral infarction and SAH caused by ACA dissection.

We administered nicardipine, glycerol and edaravone for the treatment of cerebral infarction⁶⁻⁸. The patient's neurological findings gradually

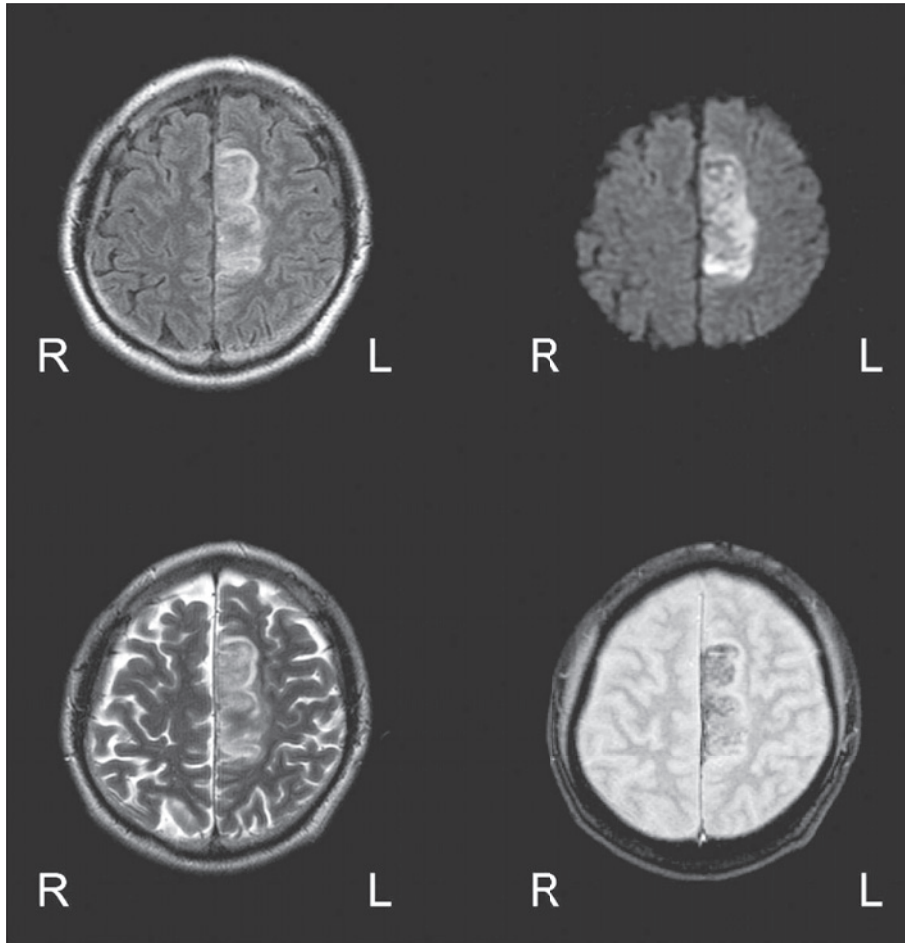


Fig. 2 Magnetic resonance imaging.

In the left interhemispheric fissure, we detected high intensity signals on fluid attenuation inversion recovery (FLAIR) image (upper left) and low intensity signals on T2*-weighted image (lower right). FLAIR, T2-weighted (lower left) and diffusion-weighted images (upper right) showed a high intensity area in the territory of left anterior cerebral artery.

improved on day 5. We could not find any new lesion on MRI, and right CAG did not show enlargement of ACA dissection on day 10. Then the patient started walk rehabilitation. NIHSS had improved to 10 on day 14 and 2 at 3 months after the onset. Modified Rankin Scale was 1 at 3 months.

Discussion

Shimoyama et al. indicated that spontaneous intracranial arterial dissection (SICAD) was found in 64.3% of patients with ACA infarction. SICAD is relatively infrequent among patients with middle cerebral artery (MCA) or posterior cerebral artery infarction⁹. There are two reasons to explain this difference. First, embolism in the ACA may be

unlikely because the diameter of the A1 segment is approximately half that of the M1 segment and cerebral blood flow is lower in the ACA than in the MCA³. Second, atherosclerotic changes are frequently found in the MCA¹⁰. According to the Spontaneous Cervicocephalic Arterial Dissections Study (SCADS), 'Double lumen' demonstrated on DSA is defined as the major criterion of cervicocephalic arterial dissection¹¹. Our patient demonstrated a long 'double lumen' in the CAG, and we could diagnose the patient as having arterial dissection. Regarding pathological features, a subintimal dissection tends to result in stenosis of the arterial lumen, leading to cerebral infarctions, whereas a subadventitial dissection may cause aneurysmal dilatation of the artery, resulting in

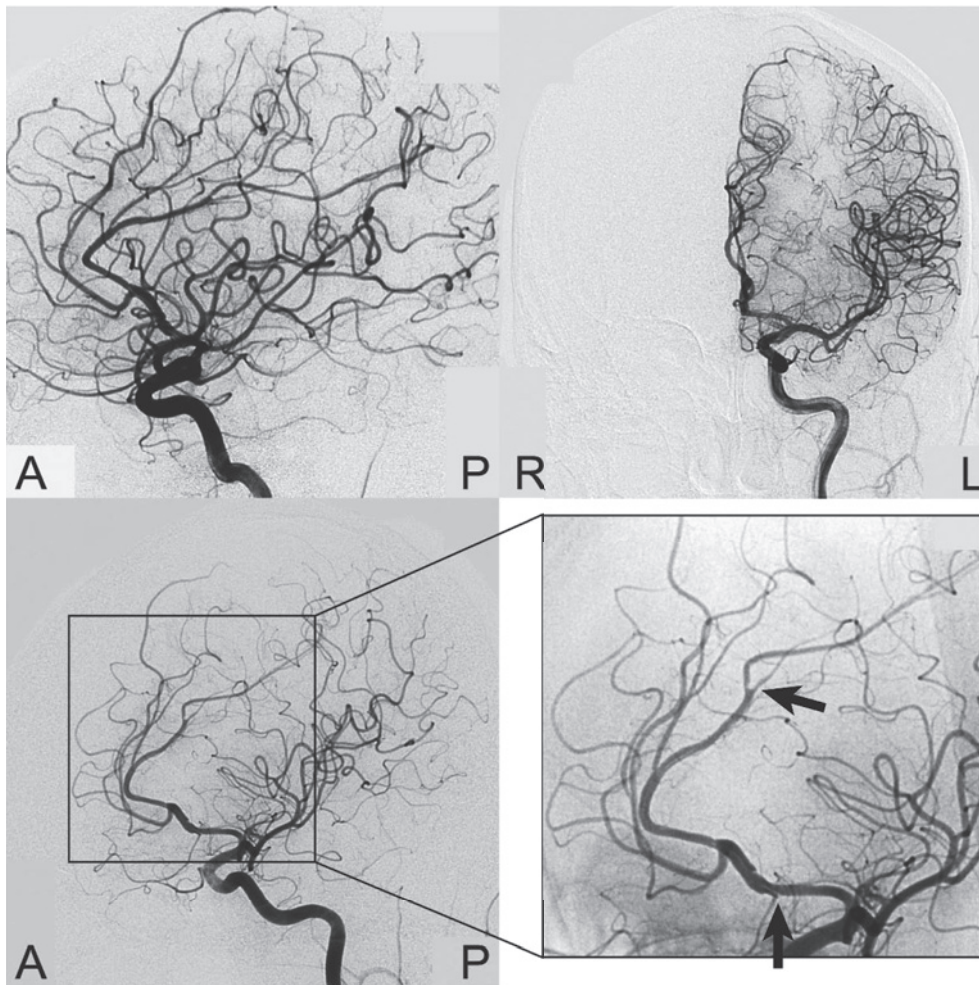


Fig. 3 Right carotid angiography showing lateral view (upper left), anteroposterior view (upper right), left anterior oblique (LAO) 30° (lower left) and magnified image of LAO (lower right).

Long double lumen sign was observed in the A2 to A4 segment of the left anterior cerebral artery (from **the lower arrow** to **the upper arrow**). In the A2 segment, dilatation of the artery was also found.

hemorrhagic events. Seven of 9 cases of ACA dissection with simultaneous subarachnoid hemorrhage and infarction showed stenosis with dilatation (**Table 1**). In these cases, we estimated to occur the both mechanisms. Many of the patients with ACA dissection accompanied by simultaneous SAH and infarction showed nonperimesencephalic SAH. Generally, a good outcome of nonaneurysmal nonperimesencephalic SAH has been reported¹². Therefore, we consider that all cases of ACA dissection with SAH and infarction showed a relative good outcome comparison with dissection with SAH, because it is not easy to demonstrated perimesencephalic lesion by ACA dissection.

Sato et al. indicated that patients with stroke in

the ACA territory caused by dissection were younger and less commonly had a history of heart disease or previous stroke than patients without dissection¹³. Anterior cerebral artery dissection in 9 patients who simultaneously developed SAH and cerebral infarction including the present case showed similar characteristics, such as good outcome and middle age (**Table 1**)¹⁴⁻¹⁸. In the case of Inoue et al., a 48-year-old man was complained of sudden onset of headache and left hemiparesis. MRI and CT detected dissection from A1 to A2, and demonstrated SAH and CI in the ACA territory. The patient initially treated by edaravone, but digital subtraction angiography (DSA) confirmed the enlarged fusiform dilatation of the right A2 with

Table 1 Cases of simultaneous subarachnoid hemorrhage and infarction caused by dissection of the anterior cerebral artery

Authors	Age/Sex	Site of Lesion	Angiographic findings	outcome
Yasukawa et al. (1993) ¹⁴	62/M	Lt. A2	stenosis with dilatation	good recovery
Kato et al. (2000) ¹⁵	40/F	Lt. A2	stenosis with dilatation	good recovery
Miyahara et al. (2001) ¹⁶	50/M	Rt. A2-A3	stenosis with dilatation	good recovery
Ohkuma et al. (2003) ¹⁷	40/M	A2	stenosis without dilatation	good recovery
Ohkuma et al. (2003) ¹⁷	64/F	A2	stenosis with dilatation	good recovery
Ohkuma et al. (2003) ¹⁷	49/F	A2	stenosis with dilatation	good recovery
Ohkuma et al. (2003) ¹⁷	49/M	A3	stenosis without dilatation	good recovery
Inoue et al. (2010) ¹⁸	49/M	ACoA-rt. A2	stenosis with dilatation	good recovery
Present case	35/M	Lt. A2-A3	stenosis with dilatation	good recovery

ACoA=anterior communicating artery.

distal narrowing on day 14. So endovascular trapping was performed. The patient remained neurologically stable after operation and got an outpatient with mild monoparesis of the left lower extremity eight month after the onset.

Because the prognoses are different, it is very important to distinguish ischemic or hemorrhagic disease early. The optimal treatment for ACA dissection remains controversial. The onset of ischemia is generally indicated for conservative therapy, whereas the onset of bleeding almost always requires surgical treatment. In this case, because vasodilatation was limited, we selected conservative treatment by administration of edaravone without surgery and achieved a good outcome. A Japanese multi-center study showed that a novel free radical scavenger, edaravone, was efficacious in patients with acute ischemic stroke^{19,20}. In vitro and in vivo studies showed that edaravone inhibited brain edema after ischemia^{21,22}, delayed neuronal death²³, and vascular endothelial cell injury²⁴. Specifically, because edaravone in the membrane can promote the delivery of fat soluble vitamin E inside the membrane and water soluble vitamin C outside the membrane and effectively prevent damage to membrane lipids, which are susceptible to radical injury, we suggest that edaravone is not complicating factor for arterial dissection.

Recently, we could easily diagnose such cases as ACA dissection with advanced technology such as DSA and MRI. Because treatment differs based on pathophysiology of the stroke type and many SAH

patients with dissecting aneurysm had poor prognoses, prompt and accurate diagnosis is important²⁵. To improve the strategy for managing ACA dissection, we need to accumulate a greater number of ACA dissection cases in the future. We also recommend that angiography be performed in patients with ACA dissection.

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