

## **Dissecting Aneurysms at the A<sub>1</sub> Segment of the Anterior Cerebral Artery**

### **—Two Case Reports—**

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### **Abstract**

**Two rare cases of dissections which involve the anterior cerebral artery (ACA) are reported. A 58-year-old woman presented with a ruptured dissecting aneurysm manifesting as sudden onset of severe headache and consciousness disturbance followed by aphasia, right hemiparesis, paresis of the left lower extremity, and choreoathetotic movements of the upper arms and face. Computed tomography and angiography revealed subarachnoid hemorrhage due to a dissecting aneurysm at the left A<sub>1</sub> segment. The dissecting aneurysm was trapped surgically on the day of onset. Her neurological deficits disappeared within a month. A 39-year-old woman experienced continuous dull headache from the day before onset, and then suffered right hemiparesis. Magnetic resonance (MR) imaging revealed cerebral infarction at the left globus pallidus. Angiography and MR imaging revealed a dissecting aneurysm at the left A<sub>1</sub> segment and occlusion of the left Heubner's artery. She received conservative treatment and her neurological findings were improved. Dissections or dissecting aneurysms involving the ACA can be classified into three types: Extension of a dissection to the ACA from the internal carotid artery, dissection at the A<sub>1</sub> segment, and dissection at the A<sub>2</sub>-A<sub>4</sub> segments. These types of dissection have distinct uniform clinical features.**

Key words: dissection, dissecting aneurysm, A<sub>1</sub> segment, anterior cerebral artery

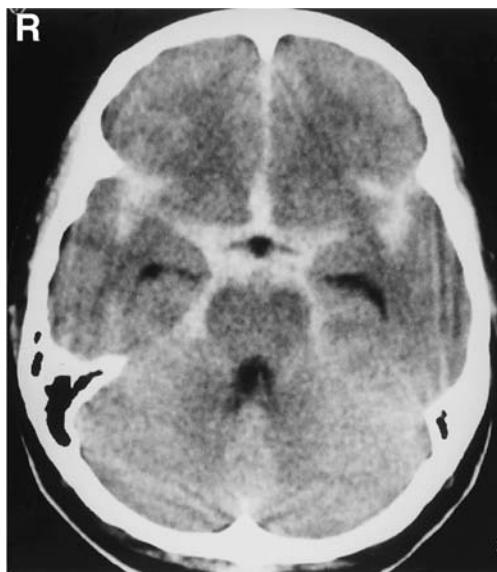
### **Introduction**

Dissections or dissecting aneurysms of intracranial arteries are rather rare<sup>35)</sup> and most frequently occur in the territories of the middle cerebral artery (MCA) or vertebrobasilar system.<sup>17,24)</sup> Dissections which involve the anterior cerebral artery (ACA) are extremely rare, with only five reported cases of dissections at the A<sub>1</sub> segment.<sup>6,10,19,23,31)</sup> Four dissections were identified by postmortem examination and one by angiography. We report two cases of dissecting aneurysms at the A<sub>1</sub> segment of the ACA which were identified by angiography and magnetic resonance (MR) imaging.

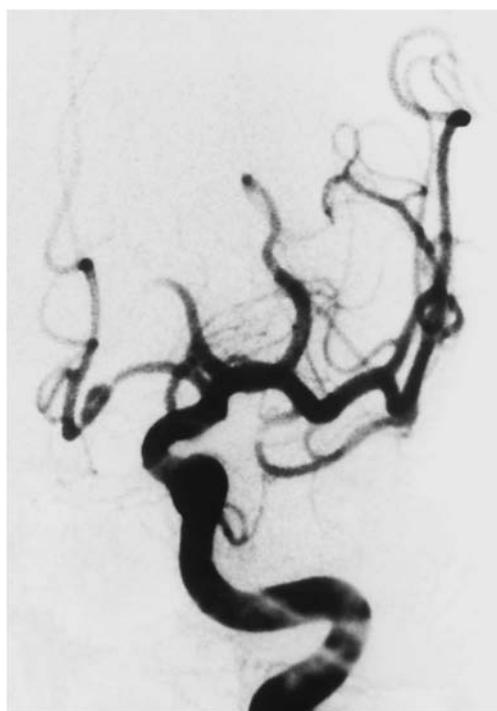
### **Case Reports**

**Case 1:** A 58-year-old previously healthy woman was admitted to the Tottori University Hospital on January 23, 1995. She suffered sudden onset of severe headache and lost consciousness. She was found by her family members, and was transported to our hospital by ambulance. On admission, she was confused and unable to speak due to motor aphasia, but responded to simple verbal orders. Choroathetotic involuntary movements involving her bilateral upper extremities and face were apparent. Complete paraplegia of the lower extremities as well as slight weakness of the right arm was observed. Laboratory findings were negative.

Computed tomography (CT) revealed diffuse subarachnoid hemorrhage (SAH), especially in the anterior part of the interhemispheric fissure which was classified as Fisher's group 3 (Fig. 1). Left carotid angiography disclosed the so-called string



**Fig. 1** Case 1. Computed tomography scan on admission showing diffuse subarachnoid hemorrhage, especially in the anterior part of interhemispheric fissure which suggests a ruptured anterior communicating artery aneurysm.



**Fig. 2** Case 1. Left carotid angiogram (oblique view) on admission showing the so-called string and pearl sign, and double contour appearance in the aneurysmal dilatation at the A<sub>1</sub> segment of the anterior cerebral artery.



**Fig. 3** Case 1. Right carotid angiogram (oblique view) with digital compression of the left common carotid artery on admission showing a dissecting aneurysm at the left A<sub>1</sub> segment via collateral flow through the anterior communicating artery.

and pearl sign at the A<sub>1</sub> segment of the ACA. The aneurysmal dilatation had a double contour appearance, which suggested the presence of true and false lumina in a dissecting aneurysm (Fig. 2). Right carotid angiography with digital compression of the left common carotid artery demonstrated the same aneurysmal dilatation at the left A<sub>1</sub> segment via the collateral flow through the anterior communicating artery (AcomA) (Fig. 3). Angiography detected no other abnormal findings. The diagnosis was SAH caused by ruptured dissecting aneurysm at the A<sub>1</sub> segment of the left ACA. After cerebral angiography, her neurological abnormalities such as motor aphasia, involuntary movement, and left leg paresis disappeared but she was still slightly confused. Her preoperative World Federation of Neurosurgical Societies grade was III, based on the Glasgow Coma Scale score of 14 and the presence of right hemiparesis.

Trapping of the dissecting aneurysm was performed via the left pterional approach. After the sylvian fissure was opened, the left internal carotid artery (ICA) was identified and the A<sub>1</sub> segment of the

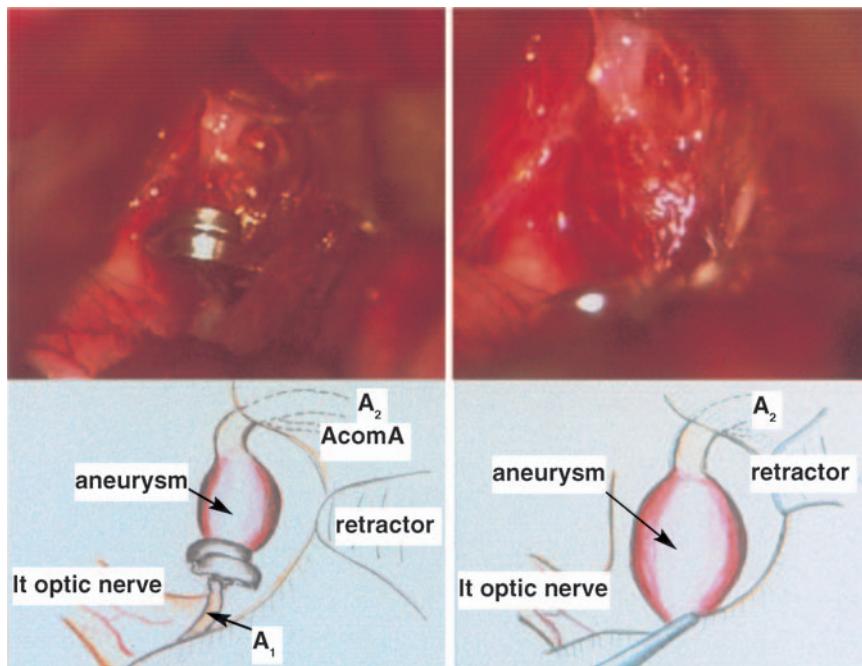


Fig. 4 Case 1. Operating microscope photographs showing a reddish and fusiform dilatation at the A<sub>1</sub> segment (right), and the collapsed aneurysmal dilatation after a straight clip was applied to the A<sub>1</sub> segment just proximal to the dissecting aneurysm (left).

left ACA was exposed from the bifurcation of the ICA to its distal portion. A reddish fusiform dilatation was seen 12 mm distal to the bifurcation (Fig. 4 right). The wall was so thin that the blood flow could be seen. A perforating branch was observed arising from the A<sub>1</sub> segment proximal to the dissecting aneurysm. A straight clip with a 7 mm blade was applied to the A<sub>1</sub> segment just proximal to the dissection, causing the aneurysmal dilatation to collapse (Fig. 4 left). The distal part of the A<sub>1</sub> segment, the AcomA, and the A<sub>2</sub> segment of the left ACA were exposed and identified, and a straight clip with a 7 mm blade was applied to the ACA adjacent to the distal end of the dissecting aneurysm.

The postoperative course was uneventful. The right hemiparesis improved gradually, and the patient could walk 2 weeks after the surgery. CT demonstrated no low-density areas during the course, but single photon emission CT using technetium-99m hexamethylpropylene amine oxime showed a low perfusion area at the medial and inferior part of the left frontal lobe. Cerebral angiography obtained 3 weeks after the surgery showed the dissecting aneurysm had disappeared (Fig. 5), and the left A<sub>2</sub>-A<sub>4</sub> segments of ACA were visualized via collateral flow through the AcomA. The left A<sub>2</sub> segment was narrowed compared to normal, probably due to delayed vasospasm. She was discharged one month after admission without neurological deficit. She has since resumed her normal activities.

**Case 2:** A 39-year-old woman was admitted to



Fig. 5 Case 1. Left carotid angiogram (anteroposterior view) 3 weeks after the onset showing the dissecting aneurysm has disappeared, and the left A<sub>1</sub> segment terminates with the perforating branch.

Teraoka Memorial Hospital on July 28, 1997. She had suffered from dull frontal headache continuous-

ly for a day. Subsequently, she had spoken words without meaning. She manifested right hemiparesis one day later and then was transported to the hospital by ambulance. On admission, she was confused slightly, but responded to simple verbal orders. She had neither weakness nor numbness in her limbs except for right facial nerve paresis. Laboratory findings were negative, and lumbar puncture showed clear cerebrospinal fluid.

MR imaging revealed a cerebral infarction at the territory of left Heubner's artery (Fig. 6). Left carotid angiography disclosed a fusiform aneurysm at the A<sub>1</sub> segment of the ACA (Fig. 7). There was no double contour appearance at the aneurysmal dilatation, nor any other abnormal findings. Axial MR imaging revealed double contour appearance in the aneurysmal dilatation, which suggested the presence of true and false lumina in the fusiform aneurysm (Fig. 8). The diagnosis was cerebral infarction caused by dissection at the A<sub>1</sub> segment of the left ACA and obliteration of the left Heubner's artery.

Her neurological deficits improved gradually after conservative treatment. Follow-up MR imaging demonstrated regression of the dissection. Repeat cerebral angiography obtained 4 weeks after admission showed reduction of the dissection and no progression to the distal portion (Fig. 9). Subsequently, she was discharged without neurological deficit and has resumed her normal activities.

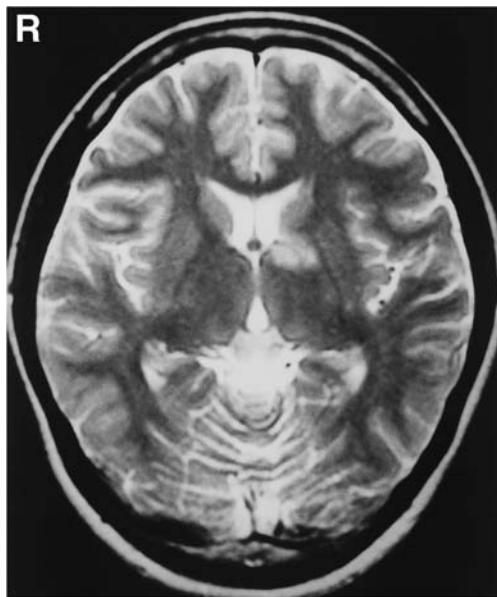


Fig. 6 Case 2. T<sub>2</sub>-weighted magnetic resonance image the day after onset revealing cerebral infarction in the territory of the left Heubner's artery.



Fig. 7 Case 2. Left carotid angiogram (anteroposterior view) disclosing a fusiform aneurysm at the A<sub>1</sub> segment of the anterior cerebral artery. There was no double contour appearance in the aneurysmal dilatation.



Fig. 8 Case 2. Axial T<sub>1</sub>-weighted magnetic resonance image 4 days after onset revealing double contour appearance in the aneurysmal dilatation, which suggested the presence of true and false lumina in the fusiform aneurysm.



**Fig. 9 Case 2. Repeat cerebral angiogram 4 weeks after onset showing reduction of the dissection and no progression to the distal portion.**

## Discussion

Only 32 cases of dissections which involve the ACA have been reported (Table 1), including only five cases of dissections at the A<sub>1</sub> segment.<sup>6,10,19,23,31</sup> Dissections or dissecting aneurysms involving the ACA can be divided into three types based on the affected site, the age of patients, the onset, progression, and outcome of the disease. Type I dissection usually originates at the ICA and extends to the ACA as well as to the MCA. Type II dissection often occurs at the A<sub>1</sub> segment of the ACA. Type III dissection mainly involves distal ACA, namely the A<sub>2</sub>, A<sub>3</sub>, and A<sub>4</sub> segments.

Most type I dissections seem to occur in young adults. All 10 patients were between 14–41 years old<sup>3,5,7,13,15,18,25,26,30</sup> except for a 75-year-old woman.<sup>1)</sup> The mean age was 24 years. The etiology of type I was speculated to be trauma in two cases, congenital fragility of the vascular wall in two, atherosclerosis in one, and unknown in five. Nine of the 10 patients had cerebral infarctions and died within 8 days of the onset. However, the 75-year-old woman<sup>1)</sup> manifested SAH and survived with mild right

hemiparesis and aphasia after surgical clamping of the affected ICA. The high mortality associated with type I dissections may be due to the sharp reduction in collateral flow to the territory of the affected ICA via the AcomA in the presence of the dissections of the A<sub>1</sub> segment of ACA. Occlusion at the earlier stage may prevent farther extension of dissection and/or possibly preserve more collateral flow through the AcomA, so we suggest that intravascular occlusion of the affected ICA may be the best treatment.

The five cases of type II dissections mostly occurred in young females except for one pediatric case.<sup>6,10,19,23,31</sup> Type II dissections often caused SAH. A 26-year-old female suffered SAH but misdiagnosis of ruptured AcomA aneurysm was made. After she died on day 12, autopsy showed a ruptured dissecting aneurysm at the A<sub>1</sub> segment of the ACA and an unruptured AcomA aneurysm. A 5-year-old boy manifested headache, right hemiparesis, and aphasia 3 days after minor head injury. Left carotid angiography demonstrated no filling of the left ACA. He died after receiving decompressive craniotomy. Postmortem examination revealed dissecting subintimal hematoma of the left ACA (A<sub>1</sub>, A<sub>2</sub>) and massive infarction of left frontal lobe. The poor outcome may have been due to the dissection of the A<sub>1</sub> segment of the ACA which extended to the A<sub>2</sub> segment, as a result of poor collateral flow through the AcomA. A 22-year-old pregnant female died of massive abdominal hemorrhage, and a 16-year-old female died of intracerebral hematoma (ICH) due to moyamoya disease. Autopsy of both cases disclosed unruptured dissections at the A<sub>1</sub> segments of ACA. A 48-year-old female experienced repeated bouts of severe headaches. She eventually lost consciousness one month after cerebral infarction at the territory of the left ACA. Surgery identified a dissecting aneurysm at the left A<sub>1</sub> and A<sub>2</sub> portions of the ACA. She recovered after the surgery. Our cases are considered to be type II dissection based on the affected site and the progression of the disease. Case I was the first to be identified angiographically and treated with trapping surgery.

Although only seven cases of type II dissection including ours have been reported, some dissections or dissecting aneurysms at A<sub>1</sub> segments may be misdiagnosed as such aneurysms. For example, 38 of 4295 aneurysms (0.88%) were aneurysms at the A<sub>1</sub> segment of ACA and nine (0.2%) of these were fusiform aneurysms or saccular aneurysms which did not arise from a bifurcation.<sup>27)</sup> Two of 1000 aneurysms (0.2%) were fusiform aneurysms at the A<sub>1</sub> segment.<sup>33)</sup> All of these cases may harbor type II dissection. Alternatively, type II dissection may be clas-

**Table 1 Table 1 Classification of dissecting aneurysms involving the anterior cerebral artery**

Type*	Author (Year)	Age/ Sex	Manifestation	Symptoms and signs	Etiology	Outcome**
I	Dratz and Woodholl (1947) <sup>5)</sup>	21/F	infarction	rt hemiparesis, semicoma	trauma	dead
	Anderson and Schechter (1959) <sup>3)</sup>	41/M	infarction	rt hemiparesis, aphasia, rt hemianopia	arteriosclerosis	dead
	Wolman (1959) <sup>30)</sup>	16/M	infarction	headache, nasal bleeding, lt hemiplegia, aphasia	congenital defect of the vessel wall	dead
	Scott et al. (1960) <sup>25)</sup>	29/F	infarction	lt hemiparesis, lt facial nerve palsy, aphasia	trauma (surgical complication)	dead
	Nedwich et al. (1963) <sup>18)</sup>	30/F	infarction	lt hemiparesis	unknown	dead
	Grosman et al. (1980) <sup>7)</sup>	23/M	infarction	dysphagia, rt hemiparesis	unknown	dead
	Adams et al. (1982) <sup>1)</sup>	75/F	SAH	headache	unknown	moderately disabled
	Steiner et al. (1986) <sup>26)</sup>	23/F	infarction	headache, lt hemiparesis, semicoma	congenital weakness of elastic lamina	dead
	Linden et al. (1987) <sup>15)</sup>	23/F	infarction	lt hemiplegia, coma	unknown	dead
	Kitani et al. (1987) <sup>13)</sup>	14/M	infarction	headache, lt hemiplegia	unknown	dead
II	Gherardi and Lee (1967) <sup>6)</sup>	26/F	SAH	headache, coma	unknown	dead
	Nelson (1968) <sup>19)</sup>	5/M	infarction	headache, rt hemiparesis, aphasia	trauma	dead
	Pilz (1977) <sup>23)</sup>	22/F	incidental		Guillain-Barré syndrome	dead
	Yamashita et al. (1983) <sup>31)</sup>	16/F	incidental		moyamoya disease + trauma	dead
	Honda et al. (1997) <sup>10)</sup>	48/F	infarction → SAH	headache, rt hemiparesis	unknown	alive
III	Present Case 1	58/F	SAH	headache, paraplegia, involuntary movement of face and arms	unknown	good recovery
	Present Case 2	39/F	infarction	headache, rt hemiparesis	unknown	good recovery
	Nakazawa et al. (1984) <sup>17)</sup>	43/M	infarction	headache, vertigo, involuntary movement of legs, convulsion, rt hemiparesis	unknown	good recovery
	Amagasa et al. (1988) <sup>2)</sup>	44/M	infarction	aphasia, rt hemiparesis	trauma	good recovery
	Sasaki et al. (1991) <sup>24)</sup>	57/M	infarction	headache, weakness of lt leg	unknown	moderately disabled
	Terai and Matsubara (1991) <sup>29)</sup>	51/M	infarction	headache, rt hemiparesis	unknown	good recovery
	Guridi et al. (1993) <sup>8)</sup>	72/F	SAH + ICH	headache, neck pain, loss of consciousness, rt hemiparesis	arteriosclerosis	moderately disabled
	Nomura et al. (1993) <sup>20)</sup>	37/M	infarction	headache, weakness of lt leg	trauma	good recovery
	Yasukawa et al. (1993) <sup>34)</sup>	62/M	infarction + SAH	rt hemiparesis, dysarthria	unknown	good recovery
	Kidooka et al. (1993) <sup>12)</sup>	45/M	infarction	headache, rt hemiparesis	unknown	good recovery
		45/M	infarction	headache, involuntary movement of legs, rt hemiparesis	unknown	good recovery
	Ishikawa et al. (1993) <sup>11)</sup>	42/M	infarction	headache, weakness of lt leg	unknown	good recovery
	Yano et al. (1995) <sup>32)</sup>	27/M	SAH + ICH	headache, rt hemiparesis	unknown	good recovery
	Nagata and Sato (1996) <sup>16)</sup>	43/M	infarction	headache, weakness of rt leg	unknown	good recovery
	Hayashi et al. (1996) <sup>9)</sup>	36/M	ICH	loss of consciousness, weakness of lt leg	unknown	good recovery

Contd.

**Table 1, contd.**

Type*	Author (Year)	Age/ Sex	Manifestation	Symptoms and signs	Etiology	Outcome**
III	Araki et al. (1996) <sup>4)</sup>	52/M	infarction	headache, weakness of lt leg	unknown	good recovery
	Okuno et al. (1996) <sup>22)</sup>	50/M	hemorrhagic infarction	headache, urinary incontinence	unknown	good recovery
	Koyama et al. (1996) <sup>14)</sup>	45/M	infarction	dizziness, weakness of lt leg	unknown	good recovery
		44/M	infarction	rt hemiparesis, dysarthria	unknown	good recovery

\*Our classification was assigned as follows: type I, extension of dissection to anterior cerebral artery from internal carotid artery; type II, dissection at A<sub>1</sub> segment of anterior cerebral artery; type III, dissection at A<sub>2</sub>-A<sub>4</sub> segments of anterior cerebral artery. \*\*According to Glasgow Outcome Scale. ICH: intracerebral hematoma, SAH: subarachnoid hemorrhage.

sified as SAH with unknown etiology, because dissections or dissecting aneurysms are often difficult to identify angiographically. MR imaging may be useful to demonstrate dissection or dissecting aneurysm.

The 17 cases of type III dissections were mainly found in middle-aged males, aged between 27-72 years (mean age 47 years).<sup>2,4,8,9,11,12,14,16,17,20,22,24,29,32,34)</sup> The patients were Japanese except for one.<sup>8)</sup> The etiology was presumed to be trauma in two cases, arteriosclerosis in one, and unknown in the others. Twelve of the 17 type III dissections caused cerebral infarctions, one SAH and infarction, two SAH and ICH, one hemorrhagic infarction, and one ICH. Fifteen patients had hemiparesis or weakness of the lower extremities. Nine of 12 patients who suffered only ischemic attacks were treated conservatively and two were operated with wrapping surgery of dissecting aneurysms at the chronic stage, whereas one was treated with trapping with A<sub>3</sub>-A<sub>3</sub> anastomosis. Trapping and resection of the dissection was performed in one patient with SAH, trapping with bypass surgery in one, and conservative treatment in one. All 15 patients showed complete improvement of neurological abnormalities with exceptions of moderate hemiparesis in two patients. However, one patient died suddenly of massive and recent gastric bleeding caused by acute erosive gastritis 6 weeks after the onset.<sup>8)</sup>

Headache is a common symptom even in patients with dissections of the ACA causing ischemia without SAH. All 16 patients who showed cerebral ischemia and were able to speak on admission complained of headache at the onset. Our first patient had aphasia, right hemiparesis, paresis of the left lower extremity, and choreoathetotic movements of the upper extremities and face after recovery of consciousness. Thus, hemiparesis and/or paresis of the lower extremities is also a characteristic of type II

dissection of the ACA. Two cases of type III dissection showed transient involuntary movements at the onset.<sup>12,17)</sup> Involvement of the extra-pyramidal system which is perfused by the ACA might cause such involuntary movements.

We selected trapping surgery for the first patient with SAH, because angiography indicated collateral flow through the AcomA, based on reports that trapping surgery is a good treatment for a fusiform aneurysm at the A<sub>1</sub> segment,<sup>21,33)</sup> when collateral flow through the AcomA was sufficient. Treatment for a dissecting aneurysm at the A<sub>1</sub> segment is likely to be similar to treatment for fusiform aneurysm.<sup>28)</sup>

Our two cases of dissecting aneurysms at the A<sub>1</sub> segment of the ACA were identified by angiography and MR imaging, and one was treated by trapping surgery. We classified dissections or dissecting aneurysms involving the ACA into three types based on the affected site. Type I dissection occurs in relatively younger patients and causes fatal brain infarctions. Type II dissection tends to occur in females and causes SAH or cerebral infarction, although there were only seven cases of this type. The outcome seemed to depend on the sufficiency of collateral flow through the AcomA and the amount of subarachnoid clot in the case of SAH. Type III dissection occurs in middle-aged males and causes transient ischemic attacks and/or rather mild SAH.

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