Revascularization for Anterior Cerebral Artery Dissecting Aneurysms
—Three Case Reports—

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Abstract
We describe three rare cases of dissecting aneurysms in the anterior cerebral artery (ACA) treated by surgical reconstruction, and reviewed 79 previously reported cases with the ACA dissecting aneurysm. We found that 35 (77.8%) of 45 patients with ischemic event and 15 (40.5%) of 37 patients with hemorrhagic event were treated conservatively, with 11.4% (4/35 cases) and 13.3% (2/15) risk of bleeding and rebleeding, respectively. Furthermore, half of these patients died. The other 32 patients were treated surgically, and their outcome was favorable, especially after surgical reconstruction. Simultaneous treatment of both hemorrhagic and ischemic events is essential. We recommend early treatment with revascularization for patients with ACA dissection that has hemorrhaged and for patients presenting with signs of clinical deterioration with ischemic event.

Key words: anterior cerebral artery, dissection, revascularization

Introduction
Intracranial dissecting aneurysms are being detected with increasing frequency and are recognized as a common cause of stroke. However, dissecting aneurysms in the anterior cerebral artery (ACA) are rare, and mostly documented as case reports. Therefore, the treatment strategy is difficult to establish on the basis of evidence. In addition, simultaneous treatment of both hemorrhagic and ischemic events is essential, but the treatment strategy remains controversial. However, surgical intervention should be considered in patients with hemorrhagic event or poor blood supply. Revascularization is one of the important methods to prevent further ischemic and/or hemorrhagic attacks in these patients.

We describe 3 cases of revascularization of dissecting aneurysm located in the ACA, and review 79 previously reported cases of nontraumatic dissecting aneurysm confined to the ACA.

Case Reports

Case 1: A 56-year-old woman experienced transient weakness in her left lower extremity while riding a bicycle and was consequently transferred to our institution. She was alert and responded to commands promptly. She had a past history of hypertension and was taking medication.

Blood, urine, and coagulation examinations found no abnormalities on admission. Computed tomography demonstrated high-density spots in the interhemispheric fissure (Fig. 1A). Angiography revealed stenosis and dilatation in the right A2 segment of the ACA (Fig. 1B). After hospitalization, the weakness deteriorated gradually within 12 hours. We interpreted this event as impending infarction, so performed emergency superficial temporal artery (STA)-ACA anastomosis on the right using the cortical branch of the ACA as the recipient artery. We could not perform an A3 side-to-side anastomosis because the left pericallosal artery did not run parallel. After the surgery, the weakness in her left lower extremity recovered immediately, and she was discharged without symptoms.

Case 2: A 44-year-old man suffered sudden headache and motor weakness in his right lower extremity. He was immediately admitted to our institution. On admission, he was alert and well oriented. He had no previous medical history. Computed tomography demonstrated high-density spots and subarachnoid hemorrhage in the interhemispheric fissure (Fig. 2A). Angiography revealed stenosis with dilatation in the left A2 segment (Fig. 2B). 99m-Technetium ethylene cystine dimmer single-photon emission computed tomography revealed extended bypass flow to the right ACA territory (Fig. 1C). Since serial angiography demonstrated improvement in the pearl and string sign in the right A2 segment (Fig. 1D), we did not perform endovascular proximal occlusion of the right A2 segment.

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Fig. 1 Representative findings in Case 1. A: Computed tomography scan demonstrating high-density spots in the interhemispheric fissure. B: Right carotid angiogram revealing stenosis with dilatation (arrowheads) in the right A2 segment. C: Postoperative right carotid angiogram revealing extended bypass flow to the right anterior cerebral artery territory. D, E: Serial right carotid angiograms demonstrating improvement in the pearl and string sign in the narrowed right A2 segment (D: 5 weeks after the onset, E: 10 weeks after the onset).

Fig. 2 Representative findings in Case 2. A: Computed tomography scan demonstrating subarachnoid hemorrhage in the interhemispheric fissure and cerebral sulcus. B: Left carotid angiogram revealing stenosis with dilatation (arrowheads) in the left A2 segment. C: Intraoperative photograph showing the diseased segment extended widely from the A1-A2 junction to the A5 segment, and diffuse thrombus in the pseudo-lumen (arrowheads) through the vessel wall. D: Postoperative left carotid angiogram demonstrating extended bypass flow to the anterior cerebral artery territory.

ealed decreased cerebral blood flow in the left ACA territory. The diagnosis was dissecting aneurysm with cerebral ischemia and subarachnoid hemorrhage. We performed STA-ACA anastomosis on the left using the frontal branch of the STA as a free tandem graft to extend the true lumen of the recipient artery, and ligated the left proximal A2 segment. The diseased segment extended from the A1-A2 junction to A5, and a diffuse thrombus in the pseudo-lumen was observed through the vessel wall (Fig. 2C). Postoperative angiography demonstrated extended bypass flow to the ACA territory (Fig. 2D). After the surgery, the motor weakness in his right lower extremity recovered gradually, and he could walk after discharge.

Case 3: A 66-year-old woman suffered sudden onset of severe headache while taking a bath. She was brought to our institution in an ambulance. No neurological deficit was recognized on admission. She had no previous medical history. Blood, urine, and coagulation examinations found no abnormalities on admission. Computed tomography demonstrated subarachnoid hemorrhage in the interhemispheric fissure and cerebral sulcus (Fig. 3A).

Fig. 3 Representative findings in Case 3. A: Computed tomography scan demonstrating subarachnoid hemorrhage in the interhemispheric fissure and cerebral sulcus. B, C: Right carotid angiograms revealing a fusiform dilatation (arrow) in the right A4 segment. D: Photomicrograph showing thickening of the tunica intima (arrowheads). Hematoxylin and eosin stain, original magnification ×40. E: Photomicrograph showing disruption of the internal elastic lamina and media (arrowheads). Elastica Van Gieson stain, original magnification ×40.
Angiography revealed a fusiform dilatation in the right A4 segment (Fig. 3B, C). The diagnosis was subarachnoid hemorrhage due to right A4 dissecting aneurysm rupture. Consequently, we performed aneurysm resection with in situ end-to-end anastomosis. Histological examination showed thickening of the tunica intima and disruption of the internal elastic lamina and media (Fig. 3D, E). Her postoperative course was uneventful, and she was discharged without neurological deficit.

**Discussion**

Dissecting aneurysm involving the anterior intracranial circulation accounts for up to 19.1% of all intracranial dissecting aneurysms, and 37% of these dissecting aneurysms are located on the ACA.29) A total of 82 cases of nontraumatic dissecting aneurysms have been described including the present 3 cases (Table 1).1–33,35,36,38–49) Among these patients, 45 presented with infarction, 26 suffered hemorrhagic events, and 11 experienced both ischemia and hemorrhage. The mean age of the patients was 50.3 years (range, 22–72 years), and men (n = 46) were affected to a greater extent than women (n = 36).

The etiology of intracranial dissecting aneurysm includes Guillain-Barré syndrome (GBS), fibromuscular dysplasia (FMD), moyamoya disease, polycystic nodosa (PN), migraine, and vascular wall abnormality caused by systemic hypertension,5,6,18,19,21,33,34,37,49) In our review, 6 cases were associated with these possible causative factors (FMD in 2,18,19) migraine in 2,6,18) and GBS,33) PN,6) polycystic kidney,42) past history of subarachnoid hemorrhage,19) and resection of arteriovenous malformation1) in one each, and 33 cases had a medical history of hypertension,5,6,7,9,13–15,17,19–21,23,26,28,30,35,39,41–45,48) (Table 1). The other medical associations were asthma,23) hyperlipidemia,23) atrial fibrillation,23) and diabetes mellitus.39)

The locations of ACA dissection are summarized in Table 2. The ACA dissections with ischemic event originated from the A1 and A2 segments.4,5,10–12,15–17,23–28,30,31,33,36,39,41–45) Most cases (88.6%) were from the A2 segment,4,5,11,12,15–17,23–27,30,31,33,36,39,40,41,43–45) The ACA dissections with hemorrhagic event originated from all segments of the ACA.1–3,6–8,10,19–20,22,23,26,28,30,40,42,46,48) However, over 50% of the cases were from the A1 segment.6,8,10,19,20,23,28,32,40,42,46) The ACA dissections with both ischemic and hemorrhagic events tended to originate from more peripheral segments compared with the ACA dissections with hemorrhagic event.9,21,24,25,28,35,49) Most cases (90.9%) were from the A2 segment.9,21,25,28,35,49) The location of ACA dissection was not described in two cases.3,33)

Treatment of dissecting aneurysm of the ACA remains controversial. The outcomes are shown in Table 3. The outcome was relatively good in 45 patients with ischemic event (good outcome in 35 cases, moderate disability in 7, and death in 3). The outcome of conservative therapy was acceptable in 35 patients with ischemic event who were treated with or without antiplatelet therapy (good outcome in 26 cases, moderate disability in 6, and death in 3). However, antiplatelet therapy carries a serious risk. Two patients died of subarachnoid hemorrhage during antiplatelet therapy for ischemia.39,44) Furthermore, 2 patients who were not treated with antiplatelet therapy developed subarachnoid hemorrhage during the chronic stage.5,11) ACA dissection with ischemic event should be

**Table 1 Summary of cases of anterior cerebral artery dissecction**

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Sex (male:female)</th>
<th>Age (years)</th>
<th>Sign</th>
<th>Past medical history</th>
<th>Location is not described in 2 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>82</td>
<td>46:36</td>
<td>22–72 range</td>
<td>45</td>
<td>43</td>
<td>2 cases.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mean 50.3</td>
<td></td>
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</tbody>
</table>

**Table 2 Location of anterior cerebral artery dissections**

<table>
<thead>
<tr>
<th>A1</th>
<th>A2</th>
<th>Distal</th>
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</thead>
<tbody>
<tr>
<td>With ischemia (n = 44)</td>
<td>5 (11.4%)</td>
<td>39 (88.6%)</td>
</tr>
<tr>
<td>With hemorrhage (n = 25)</td>
<td>14 (56.0%)</td>
<td>6 (24.0%)</td>
</tr>
<tr>
<td>With ischemia and hemorrhage (n = 11)</td>
<td>0 (0.0%)</td>
<td>10 (90.9%)</td>
</tr>
</tbody>
</table>

Location is not described in 2 cases.

**Table 3 Outcome of anterior cerebral artery dissections**

<table>
<thead>
<tr>
<th>GR</th>
<th>MD</th>
<th>SD + VS</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>With ischemia (n = 45)</td>
<td>35 (77.8%)</td>
<td>7 (15.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>conservative therapy (n = 35)</td>
<td>26 (74.3%)</td>
<td>6 (17.1%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>surgery (n = 10)</td>
<td>9 (90.0%)</td>
<td>1 (10.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>With hemorrhage (n = 37)</td>
<td>23 (62.2%)</td>
<td>5 (13.5%)</td>
<td>7 (18.9%)</td>
</tr>
<tr>
<td>conservative therapy (n = 15)</td>
<td>10 (66.7%)</td>
<td>2 (13.3%)</td>
<td>1 (6.7%)</td>
</tr>
<tr>
<td>surgery (n = 22)</td>
<td>13 (59.1%)</td>
<td>3 (13.0%)</td>
<td>6 (27.3%)</td>
</tr>
</tbody>
</table>

treated conservatively because of the good outcome. However, our review found that four (11.4%) of the conservatively treated patients experienced bleeding and 2 died of the bleeding episode. Therefore, surgical intervention should be considered if the dissection progresses with clinical or angiographic deterioration. Several surgical techniques have been attempted, such as wrapping, and trapping with or without bypass. Complications related to these surgeries have not been reported. The present Case 1 illustrates ACA dissection treated with STA-ACA anastomosis.

The outcome was relatively poor in 37 patients with bleeding (good outcome in 23 cases, moderate disability in 5, severe disability in 4, vegetative state in 3, and death in 2). Fifteen patients were treated conservatively, of whom 1 died and the other was severely disabled because of rebleeding. Twenty-two patients were treated surgically, wrapping, trapping with or without bypass, clipping, and endovascular surgery. Two patients who were treated surgically experienced rebleeding; 1 patient had undergone clipping of the aneurysmal bulge and wrapping may be insufficient treatment for ACA dissection with hemorrhagic event. Therefore, we recommend early treatment with revascularization for patients with dissection that has hemorrhaged. The present Case 2 shows the extensive spread of a diseased segment in the ACA. All our 3 cases of surgical reconstruction of dissecting aneurysm located in the ACA exhibited good outcomes.

We recommend early treatment with revascularization for patients with dissection that has hemorrhaged and for patients presenting with signs of clinical deterioration with ischemic event. However, conservative management is adequate for dissecting aneurysms manifesting as ischemic symptoms unless the aneurysm progresses or fails to regress over time.

References

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