Ruptured Fusiform Aneurysm of the Proximal Anterior Cerebral Artery (A1 Segment)

—Case Report—

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Abstract

A 42-year-old man presented with a ruptured fusiform aneurysm of the proximal anterior cerebral artery (A1 segment) manifesting as sudden onset of severe headache. Brain computed tomography revealed subarachnoid hemorrhage in the basal cisterns, and left carotid angiography demonstrated a fusiform aneurysm of the left A1 segment. He underwent surgery via the left pterional approach. The left A1 segment exhibited a fusiform configuration. Adequate development of the anterior communicating artery was confirmed. Trapping of the aneurysm was performed. The aneurysm was associated with atherosclerotic changes. The postoperative course was uneventful, and the patient was discharged without neurological deficits 1 month after surgery. Fusiform aneurysm of the A1 segment is quite rare, and tends to bleed, so must be treated. The atherosclerotic origin indicates long-term follow up to identify subsequent lesions.

Key words: fusiform aneurysm, anterior cerebral artery, cerebral aneurysm, A1 segment

Introduction

Intracranial fusiform aneurysm accounts for about 1% of brain aneurysms, is caused by atherosclerosis, and usually occurs in the basilar artery, internal carotid artery (ICA), and middle cerebral artery (MCA). Fusiform aneurysm developing in the ICA or MCA sometimes extends to the anterior cerebral artery (ACA), but fusiform aneurysm restricted to the ACA is very rare, accounting for only about 1% of aneurysms arising from the ACA. The incidence of proximal ACA (A1 segment) aneurysms is reported to be 0.76–3.4%, often in cases of vascular anomalies complicated by aneurysms and cases of multiple aneurysms, and mostly of saccular type.

We encountered a case of fusiform aneurysm of the A1 segment which was treated by trapping.

Case Report

A 42-year-old man suffered sudden onset of headache and dizziness, and was transported to our hospital by ambulance on January 19, 2006. He had a past history of hypertension, gout, and hyperlipidemia. He had no history of syphilis, aortic stenosis, Marfan’s syndrome, or other conditions associated with cerebral aneurysm. His family history was unremarkable. He had been consuming 80 mg of alcohol and 30 cigarettes per day for 20 years.

Neurological examination found no abnormality. Brain computed tomography (CT) showed Fisher group 3 subarachnoid hemorrhage (SAH) (Fig. 1). The diagnosis was World Federation of Neurological Surgeons grade I SAH. Left internal carotid angiography revealed a fusiform dilatation of the left A1 segment. No vascular stenosis or thrombus formation were detected within the fusiform dilatation. Right internal carotid angiography disclosed the anterior communicating artery (ACoA) as well as...
sufficient collateral blood supply to the left distal ACA (Fig. 2). Right vertebral angiography revealed markedly tortuous arrangement and prolongation of the vertebral and basilar arteries. On the basis of these findings, the diagnosis was SAH due to rupture of the fusiform or dissecting aneurysm of the left A1 segment.

He underwent emergency surgery via a left pterional approach on the day of admission. The walls of the left ICA and left ACA were yellow and hard, indicating atherosclerotic change. The left A1 segment had irregular margins and included a fusiform dilatation which extended proximally to a point near the bifurcation of the recurrent artery of Heubner. The surroundings of the left A1 segment were occupied by fresh clot, suggesting that this aneurysm was responsible for the SAH. A well-developed ACoA was noted. The left A1 proximal group included six perforators, which all entered the anterior perforated substance. Another perforator branched from the aneurysm. This aneurysm wall exhibited circumferential thinning and was swollen like a blood blister, with blood flow within the vessel visible from outside. There was no intramural hematoma (Fig. 3A, B).

Wrapping was initially considered because of the presence of a perforator bifurcating from the aneurysm. However, the aneurysm wall was very thin, so only wrapping was unlikely to achieve adequate hemostasis. In addition, the ACoA was well-developed, the aneurysm wall exhibited circumferential thinning, and the A1 proximal group included multiple well-developed perforators. Therefore, the aneurysm was trapped, and the one perforator branching from the aneurysm was resected (Fig. 3C). After trapping, the blood flow through the left distal ACA and recurrent artery of Heubner was maintained. The rupture point covered with white thrombus was visible on the aneurysm wall closer to the optic chiasm (Fig. 3D).
The postoperative course was uneventful. Brain CT demonstrated no low density areas. Memory disturbance and reduction of activity were noted, probably due to obstruction of the perforator, and were alleviated by treatment with intravenous protirelin tartrate. He was discharged without neurological deficit on February 28, 2006.

Discussion

Fusiform aneurysm and dissecting aneurysm both present with a spindle-shaped appearance. Fusiform aneurysm is characterized by dilated and tortuous arteries associated with atherosclerosis.\(^{33}\) Progression of atherosclerosis can lead from dilated and tortuous arteries to the formation of fusiform aneurysm.\(^{11,14,16,23,24,29}\) Fusiform aneurysm develops from the parent artery irrespective of any bifurcation, forming a broad base, and both the parent artery and the aneurysm exhibit marked atherosclerosis.\(^{16,17,25,29}\) In the present case, left internal carotid angiography revealed fusiform dilatation of the left A1 segment indicating either fusiform or dissecting aneurysm. A nationwide study of non-traumatic intracranial dissecting aneurysm in Japan found that the diagnostic criteria are vascular narrowing (tapered narrowing, string sign), vascular occlusion, aneurysmal outpouching, intimal flap, retention of contrast media in false lumen, etc.\(^{34,35}\) The present case showed none of these criteria. Intraoperatively, no evidence of dissecting aneurysm was identified. On the basis of these findings, the morphological diagnosis was fusiform aneurysm. His family refused permission for histological examination of the aneurysm.

Only 11 cases of fusiform aneurysm confined to the A1 segment have been reported, including the present case (Table 1).\(^{15,16,23,27-29,36}\) Fusiform aneurysm is usually associated with neural compression or ischemia, and rarely with rupture.\(^{1,16,29}\) However, 10 of the 11 reported cases presented with SAH.\(^{15,16,23,27,28,36}\) Therefore, fusiform aneurysm in the A1 segment is very likely to cause SAH. Progressive expansion of fusiform aneurysm sometimes results in saccular or spherical shape, which is likely to result in hemorrhage and requires careful treatment.\(^{25,29}\)

Surgical treatment is required for fusiform aneurysm causing SAH or neurological symptoms. However, the management strategy of this type of aneurysm remains controversial.\(^ {26}\) Neck clipping is rarely useful. Angioplastic clipping must be performed in many cases, and involves great difficulties.\(^ {10}\) It depends on the relation between aneurysm and parent artery whether clipping is possible for fusiform aneurysm.\(^ {26}\) Ordinary clipping is difficult for fusiform aneurysm with fully circumferential enlargement of the parent artery like the present case, so trapping or wrapping must be performed.

Trapping was performed in two such cases.\(^ {36}\) Confirmation of the collateral blood supply and preservation of the perforating artery are both important. If trapping appears dangerous, additional anastomosis or only reinforcement of the aneurysm are advisable.\(^ {36}\) Particular care is needed during surgery, since fusiform aneurysm involves sclerosis of the parent artery.\(^ {17}\) Careless manipulation of the aneurysm is likely to cause vascular obstruction or irreversible damage to the aneurysm wall. Therefore, only reinforcement of the aneurysm should be performed if clipping is difficult.

The ideal procedure for the present case may be trapping of the segment after bifurcation of the perforator and subsequent wrapping of the aneurysm before the point of bifurcation. However, the present aneurysm wall was thin enough to make the blood flow within the vessel visible from outside. Wrapping of the cerebral aneurysm in 74 cases, and long-term angiographic follow-up survey in 34 of these cases found no aneurysm size or configuration change.\(^ {31}\) The wrapping procedure is safe and durable, so can prevent aneurysmal growth or hemorrhage. However, wrapping has a re-rupture rate of 12–18\%,\(^ {2,4,6,13,21,22,30}\) indicating insufficient safety and durability to prevent re-rupture and re-growth after treatment. Incomplete wrapping has resulted in many cases of re-rupture.\(^ {2,21,22}\) Re-growth of fusiform aneurysm may be caused by incomplete

### Table 1 Summary of 11 cases of fusiform aneurysm of the proximal anterior cerebral artery

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Site</th>
<th>Therapy</th>
<th>SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yasargil (1984)(^ {36})</td>
<td>64</td>
<td>F</td>
<td>Lt</td>
<td>trapping</td>
<td>+</td>
</tr>
<tr>
<td>Tamura et al. (1985)(^ {20})</td>
<td>54</td>
<td>F</td>
<td>Lt</td>
<td>trapping</td>
<td>+</td>
</tr>
<tr>
<td>Shigemori et al. (1987)(^ {23})</td>
<td>47</td>
<td>M</td>
<td>Lt</td>
<td>clipping</td>
<td>+</td>
</tr>
<tr>
<td>Suzuki et al. (1988)(^ {28})</td>
<td>62</td>
<td>F</td>
<td>Lt</td>
<td>ND</td>
<td>+</td>
</tr>
<tr>
<td>Oba et al. (1989)(^ {29})</td>
<td>49</td>
<td>M</td>
<td>Lt</td>
<td>ND</td>
<td>+</td>
</tr>
<tr>
<td>Suzuki et al. (1992)(^ {24})</td>
<td>49</td>
<td>M</td>
<td>Lt</td>
<td>trapping</td>
<td>+</td>
</tr>
<tr>
<td>Nomura et al. (2000)(^ {30})</td>
<td>68</td>
<td>M</td>
<td>lt</td>
<td>coiling</td>
<td>+</td>
</tr>
<tr>
<td>Present case</td>
<td>42</td>
<td>M</td>
<td>lt</td>
<td>trapping</td>
<td>+</td>
</tr>
</tbody>
</table>

ND: not described, SAH: subarachnoid hemorrhage.
wrapping. Similar factors, such as hypertension and arteriosclerosis, associated with severe atherosclerotic change in other intracranial vessels. Postnatal aggravating deficits. In the present case, we trapped the entire changes, personality disorders, and intellectual sequelae for the patient. If only the perforator from the A1 segment is injured, paralysis is usually absent and the psychiatric symptoms are emotional and intellectual deficits. In the present case, we trapped the entire aneurysm, including the perforator, with the first priority of adequate hemostasis, although we were aware of the risk of disturbing the perforator. We believe that selection of this method requires careful judgment.

Fusiform aneurysm of the A1 segment is rare, and the natural history remains unknown, but is likely to include bleeding. Therefore, this lesion should be isolated from the cerebral circulation with preservation of any perforator and blood flow through intact brain vessels. Fusiform aneurysm is often associated with severe athro sclerotic change in other intracranial vessels. Postnatal aggravating factors, such as hypertension and arteriosclerosis, are important in the induction of saccular aneurysms in rats and monkeys. Therefore, similar aneurysms are likely to develop at other sites. Long-term follow up is required after surgery for fusiform aneurysm.

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