A considerable amount of literature has been published on cerebral dissecting aneurysms. However, there has never been a precise study on the 3D structures of true lumens and pseudolumens in these aneurysms. We recently classified cerebral trunk aneurysms into four types based on the pathological state of the IEL and intima. Classic cerebral dissecting aneurysms correspond to Type 1 aneurysms. They possess a true lumen and a pseudolumen and exhibit acute clinical courses including SAH and cerebral infarction. Eight aneurysms were accompanied with subarachnoid hemorrhage (SAH) and one with infarction. Seven aneurysms were obtained at autopsy and two were obtained during surgery (trapping and bypass). All nine aneurysms were sectioned into serial axial slices measuring 5 to 10 µm in thickness. Taking each slice as an element, we reconstructed the 3D structure of the aneurysm.

The true lumen communicated with a pseudolumen through the disrupted portion of the internal elastic lamina (IEL) in all nine aneurysms. The ruptured portion was located just above the disrupted IEL. Two aneurysms had an exit back into the true lumen, but the other seven had no such exit.

Conclusions. The primary mechanism by which a cerebral dissecting aneurysm is created is by the sudden disruption of the IEL. The plane of dissection extends through the media. The majority of aneurysms have one entrance into the pseudolumen (entry-only type). This type is associated with an unstable clinical course. Some cerebral dissecting aneurysms have both an entrance and exit (entry–exit type). This type of aneurysm occasionally contains a constant flow of blood through the pseudolumen and is clinically more stable than entry-only aneurysms.

Key Words • dissecting aneurysm • aneurysm morphology

Clinical Material and Methods

Between 1985 and 1999, we treated 102 nontraumatic cerebral dissecting aneurysms (Type 1). Of the 102 aneurysms, 84 were accompanied with SAH and 18 with cerebral ischemia. Thirteen aneurysms arose from the anterior circulation and 89 aneurysms from the posterior circulation. Our basic treatment policy for dissecting aneurysms accompanied with SAH has been surgery to prevent rebleeding. Basically, we conducted proximal clipping or trapping for VA dissecting aneurysms and trapping and bypass or wrapping with a clip for dissecting aneurysms arising from arteries other than the VA.

Nine aneurysm specimens were excised from eight patients. Clinical summaries of these patients are listed in Table 1. Patients’ ages ranged from 34 to 67 years. The interval between the initial SAH and removal of the aneurysms in these patients varied from 20 hours to 35 days.

Of the nine aneurysms, seven arose from the VA, one from the ACA, and one from the SCA. In one patient there were dissecting aneurysms on the bilateral VAs. Eight aneurysms coincided with SAH and one with infarction. Two aneurysms were obtained during surgery (trapping and bypass).
Three-dimensional structure of cerebral dissecting aneurysms

**Results**

**Relationship Between Pseudolumen and True Lumen**

A substantial pathological finding common to all nine dissecting aneurysms was the communication between the true lumen and pseudolumen through a disrupted portion of the IEL (entry: Fig. 1). According to the reconstructed 3D structure of the aneurysm, the disruption of the IEL occurred longitudinally, not axially, along the direction of the artery. The media adjacent to the disrupted portion of IEL was also partially or wholly disrupted (Fig. 2) in all nine aneurysms; however, medial degeneration was basically not detected. The plane of dissection (pseudolumen) differed from site to site, even within the same aneurysm, extending longitudinally along the artery between the IEL and media, within the media, and between the media and adventitia. Thus, the plane of dissection extended throughout the media.

Walls adjacent to the rupture site were only composed of adventitia and/or fibrin derived from thrombus (Fig. 3). In the present series, however, the proliferation of collagen, which is a long-lasting repair process to reinforce the aneurysm wall, was not observed at the rupture site, even in the aneurysm that was removed 35 days after the initial onset of SAH (Fig. 1 upper).

Under high magnification, we found that the portion of IEL adjacent to the site of disruption was somewhat more damaged than the IEL in the healthy portion of artery (Fig. 2). In several cases, disrupted IEL had been repaired by intimal thickening without formation of a dissecting aneurysm (Fig. 4).

**One-Entry and Entry–Exit Aneurysms**

Of the nine dissecting aneurysms, seven had one entrance into the pseudolumen (entry-only type; Fig. 1) and two had both an entrance and an exit (entry–exit type: Fig. 5a). In all entry-only aneurysms, the entrance was larger than those found in entry–exit aneurysms. The rupture site was located just above the orifice of the entrance in all eight ruptured aneurysms (Figs. 1 and 5). In each entry-only dissecting aneurysm, the ruptured site corresponded to the portion of aneurysm that appeared to be the most dilated on angiograms.

In the two aneurysms that had both an entrance and exit, the pseudolumen was formed mainly within the media. In one of them, both the true lumen and pseudolumen were patent, and the pseudolumen was more dominant than the true lumen. In this aneurysm, dense collagen proliferation occurred on the inner surface of the pseudolumen (Fig. 5a). The rupture site of this aneurysm was located just above the entrance into the pseudolumen (Fig. 5a and b). In the other ischemic case, the pseudolumen was thrombosed.

**Illustrative Case**

**Case 2**

This 52-year-old man was admitted to the hospital on an emergency basis for treatment of SAH. He had a history of uncontrolled hypertension. On admission, the patient was alert and displayed no neurological deficit (World Federation of Neurosurgical Societies Grade I). Four-vessel arteriography was performed and revealed a fusiform aneurysm with a smooth contour arising from the left A1 segment of the ACA (Fig. 5c). One day post-SAH, the patient underwent a right frontal craniotomy. Via an interhemispheric approach, the pink fusiform aneurysm was trapped and resected. The left ACA distal to the aneurysm was anastomosed to the right ACA (Fig. 5c). The patient’s postoperative course was excellent and he was able to return to work.

**Discussion**

**Pathological Mechanism of Cerebral Dissecting Aneurysm**

The IEL is the most important layer of the cerebral arte-

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**TABLE 1**

Clinical characteristics of eight patients with nine dissecting aneurysms*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location</th>
<th>Present-</th>
<th>Excision</th>
<th>Aneurysm Type of Specimen†</th>
<th>Aneurysm</th>
<th>Pathological Type of Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34, F</td>
<td>lt</td>
<td>SCA</td>
<td>SAH</td>
<td>surgery on Day 23</td>
<td>NA</td>
<td>trapping &amp; STA–SCA bypass (Day 23)</td>
<td>entry only</td>
</tr>
<tr>
<td>2</td>
<td>52, M</td>
<td>lt</td>
<td>A1</td>
<td>SAH</td>
<td>surgery on Day 1</td>
<td>NA</td>
<td>trapping &amp; A2–A1 bypass (Day 1)</td>
<td>entry-exit</td>
</tr>
<tr>
<td>3</td>
<td>67, M</td>
<td>lt</td>
<td>VA</td>
<td>SAH</td>
<td>autopsy on Day 12</td>
<td>NA</td>
<td>proximal clipping (Day 1)</td>
<td>entry only</td>
</tr>
<tr>
<td>4</td>
<td>56, M</td>
<td>lt</td>
<td>VA</td>
<td>SAH</td>
<td>autopsy on Day 14</td>
<td>Day 2</td>
<td>NA</td>
<td>entry only</td>
</tr>
<tr>
<td>5</td>
<td>43, F</td>
<td>lt</td>
<td>VA</td>
<td>SAH</td>
<td>autopsy on Day 14</td>
<td>Day 7</td>
<td>NA</td>
<td>entry only</td>
</tr>
<tr>
<td>6</td>
<td>50, M</td>
<td>rt</td>
<td>VA</td>
<td>SAH</td>
<td>autopsy on Day 8</td>
<td>NA</td>
<td>proximal clipping (Day 1)</td>
<td>entry only</td>
</tr>
<tr>
<td>7</td>
<td>49, M</td>
<td>rt</td>
<td>VA</td>
<td>SAH</td>
<td>autopsy on Day 35</td>
<td>Day 14</td>
<td>NA</td>
<td>entry only</td>
</tr>
<tr>
<td>8</td>
<td>41, M</td>
<td>rt</td>
<td>VA</td>
<td>infarction</td>
<td>surgery on Day 15</td>
<td>Day 0</td>
<td>NA</td>
<td>entry-exit</td>
</tr>
</tbody>
</table>

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* NA = not applicable.
† Day post-SAH.

and bypass) and seven were obtained at autopsy. In the six patients in whom the aneurysms were obtained at autopsy, the causes of death included respiratory failure after proximal clipping in two patients and initial brain damage with or without rebleeding in four patients in whom surgery could not be performed because of poor clinical status.

In each case, the entire aneurysm, as well as healthy portions of arteries lying on both sides, were sectioned into serial axial slices measuring 5 to 10 μm in thickness. Each slice was stained with hematoxylin and eosin and by the elastica van Gieson method. Taking each slice as an element, we reconstructed the schematic 3D image of each aneurysm.
rial wall for the protection of the artery against hemodynamic stress. In one experimental study, this layer alone withstood a blood pressure of 600 mm Hg without the support of other layers.\(^8\) It is reasonable to assume that cerebral aneurysm formation occurs at the portion of locus minoris resistantiae provided by the weakness of the IEL.\(^2,22\) Since the first report of Turnbull\(^2,24\) in 1915, hypotheses on the mechanism of nontraumatic cerebral dissecting aneurysms have included syphilitic arteritis,\(^2,24\) rupture of the vasa vasorum,\(^2,26\) medial degeneration,\(^2,25,26\) and congenital defect of the media and/or the IEL.\(^2,26\) In the present series, no medial degeneration, inflammation, or rupture of the vasa vasorum was detected adjacent to the disrupted portion of the IEL. More recently, investigators have asserted that there is a relationship between a defect in the IEL, either congenital or acquired, and the genesis of cerebral dissecting aneurysms.\(^6,7,18\) There are several reports of traumatic cerebral dissecting aneurysms.\(^2,4,21\) The pathological findings of traumatic cerebral dissecting aneurysms are similar to those of nontraumatic cerebral dissecting aneurysm, including disruption of the IEL.\(^2,4,21\)

**Fig. 1.** Case 7. **Upper:** Photomicrograph showing serial axial slices of a typical entry-only dissecting aneurysm treated conservatively and removed 35 days post-SAH. The wall adjacent to the rupture site (long arrow) is only composed of fibrin and thin collagen. The other arrows indicate disrupted ends of the IEL. Elastica van Gieson, original magnification × 40. **Lower:** Right VA arteriogram (left) revealing a fusiform aneurysm with an irregular contour. Photograph (center) of the aneurysm, which was obtained at autopsy on Day 35 post-SAH. Illustration (right) of the reconstructed entry-only dissecting aneurysm. Wavy line represents the IEL; gray area represents the media.

**Fig. 2.** Photomicrograph demonstrating the disrupted IEL. The IEL adjacent to the disrupted end is damaged and the media is also disrupted (arrow). Elastica van Gieson, original magnification × 400.
Based on the findings of the present study, we strongly support the hypothesis that the substantial pathological mechanism by which a cerebral dissecting aneurysm is created is the sudden widespread disruption of the IEL, as has been asserted by several authors. In our series, the plane of dissection differed from site to site within the aneurysm. We assume that the plane of dissection extends through the loose portion of the media and may be determined by the extent of disruption of the media. In every case, disruption of media was observed adjacent to the disrupted IEL. It is assumed that if the media is wholly disrupted, the dissecting plane is formed between the media and adventitia, whereas, if disruption of the media is minimal, the dissecting plane is formed between the IEL and the media. In fact, according to our survey of previous literature, dissecting aneurysms accompanied with infarction have less medial damage than those with SAH.

Repair of Dissecting Aneurysms

The rebleeding rate of cerebral dissecting aneurysms accompanied with SAH is remarkably high. In the present study, 48 (57.1%) of 84 patients who experienced SAH suffered rebleeding. This rate gradually decreased from 1 week after SAH; however, in 10% of cases rebleeding still occurred after Day 30 post-SAH. Long-lasting tissue repair processes generally include collagen proliferation. Collagen fiber is the structural protein that repairs and reinforces injured tissue.

In an experimental study, a thin neointima began to appear 1 week after a stretch injury is sustained by arteries. It is presumed to take at least several weeks to produce enough collagen fiber in the neointima to support a long-lasting repair process. In the present study, our impression was that dissecting aneurysms still carried a risk for rebleeding even as late as Day 35 post-SAH.

Patients with cerebral dissecting aneurysms frequently suffer from nuchal–occipital headaches before SAH occurs. In Case 8, a VA dissecting aneurysm was detected on a magnetic resonance angiogram obtained in a patient who was experiencing a sudden nuchal headache 4 days before onset of SAH. Similarly, headaches that occur before onset of SAH may announce the moment of generation of dissecting aneurysms.

Sudden disruptions of the IEL may occur occasionally and these disruptions are irreversible. The majority of
disrupted IELs may be covered with local intimal thickening and may not develop into dissecting aneurysms.

Differences in Clinical Course Between Entry-Only and Entry–Exit Aneurysms

Entry–exit cerebral dissecting aneurysms are assumed to occur less frequently than entry-only ones. However, thus far it has been impossible to determine the exact ratio, because earlier reports on cerebral dissecting aneurysms have only demonstrated the presence of entry–exit aneurysms in one patient with an ischemic middle cerebral artery dissecting aneurysm.15

In Case 2 of the present series, the age of the entry–exit aneurysm was assumed to be more than several weeks at the time of bleeding, because dense collagen proliferation was observed on the surface of the pseudolumen, which had formed within the media. However, we assume that
Three-dimensional structure of cerebral dissecting aneurysms

the age of the majority of entry-only aneurysms at the time of bleeding ranged from several hours to 1 week, at the longest, because there was no evidence of long-term repair mechanisms such as the appearance of neointima or collagen proliferation when the aneurysms were removed.

We previously reported the case of a patient in whom there was an assumed entry–exit ischemic cerebral arterial dissection extending from the internal carotid artery to the M1 segment of the middle cerebral artery. In that case, both the true lumen and the pseudolumen remained persistently patent and were not associated with subsequent symptoms.

Similarly, in the present series we found that in some entry–exit dissecting aneurysms there is a constant stable flow through the pseudolumen for long periods with few clinical symptoms.

However, in the entry-only dissecting aneurysms, the structure of the pseudolumen is a cul-de-sac, having no exit point and making it impossible to buffer hemodynamic pressure. Therefore, we know that blood flow in the pseudolumen is unstable and rebleeding can easily occur during the acute stage. At the end of the pseudolumen, thrombosis can easily occur. This thrombosis in the margin of the pseudolumen is usually detected on angiography as stenosis. In contrast, the margin of the entry–exit dissecting aneurysm tends to be smooth if the pseudolumen is patent.

References

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