De Novo Basilar Head Aneurysms
—Two Case Reports—

Ichiro KAWAHARA, Morito NAKAMOTO, Yoshitaka MATSUO, and Yoshiharu TOKUNAGA

Department of Neurosurgery, Nagasaki Prefecture Shimabara Hospital, Shimabara, Nagasaki

Abstract

We describe two rare cases of de novo basilar head aneurysms. The first patient presented with a ruptured basilar tip aneurysm, which developed de novo 5 years after the clipping of a left middle cerebral artery aneurysm. The second patient presented with a right basilar artery-superior cerebellar artery aneurysm, which developed de novo 3 years after the clipping of a right A1 aneurysm. The formation of de novo aneurysm in the posterior circulation has significant implications for screening. Patients must be carefully examined not only at the sites of the initial aneurysm, but also at separate sites, including the posterior circulation, to evaluate the possibility of de novo appearance of another aneurysm.

Key words: basilar head aneurysm, de novo aneurysm, follow up, posterior circulation, risk factor

Introduction

De novo aneurysms that develop at sites that were previously angiographically normal are well known, are either new aneurysms at sites separate from an initial aneurysm or aneurysms that were not detected by preoperative angiography,7 and are generally found several years after the diagnosis of initial aneurysms.10 The term “de novo” was first used to describe the development of an aneurysm of the middle cerebral artery (MCA) in a site that was previously angiographically normal.3 Subsequently, several cases of de novo aneurysms were reported. However, few de novo aneurysms have been found in the posterior circulation compared to many found in the anterior circulation.5,8–11,17 The natural history of these lesions remains poorly understood.

Here we describe two further cases of de novo basilar head aneurysms.

Case Reports

Case 1: A 48-year-old woman was admitted to our hospital with subarachnoid hemorrhage (SAH) (Hunt and Kosnik grade II, Fisher group III). The patient underwent complete clipping of a ruptured left MCA aneurysm (Fig. 1A). Cerebral angiography revealed no other aneurysm and no evidence of basilar tip aneurysm (Fig. 1B). The postoperative course was uneventful and the patient was discharged with no neurological deficit. The patient was a nonsmoker, with no family history of SAH or hypertension. Five years later, she suffered severe headache and nausea again during housekeeping activities. Computed tomography (CT) revealed recurrent SAH including intraventricular hemorrhage (Fisher group IV) (Fig. 1C). Neurological examination on admission was Hunt and Kosnik grade III. Cerebral angiography revealed a basilar tip aneurysm that had not been seen on the previous study (Fig. 1D, E). The patient underwent neck clipping via a right subtemporal approach. Postoperatively, slight oculomotor palsy appeared, but improved gradually. Follow-up CT revealed ventricular enlargement and a left ventriculoperitoneal

Fig. 1 Case 1. A: Left internal carotid angiogram showing successful neck clipping of a ruptured middle cerebral artery aneurysm. B: Right vertebral angiogram showing no abnormal findings. C: Computed tomography (CT) scan showing intraventricular hemorrhage 5 years later. D: Right vertebral angiogram showing a de novo basilar tip aneurysm projecting posteriorly 5 years later. E: Three-dimensional CT angiogram showing a de novo basilar tip aneurysm.
shunt was placed. The patient was discharged with no neurological deficit.

**Case 2:** A 49-year-old woman was admitted to our hospital for SAH (Hunt and Kosnik grade II, Fisher group II). The patient underwent complete clipping of a ruptured right A1 aneurysm (Fig. 2A). Cerebral angiography revealed no other aneurysm (Fig. 2B). The postoperative course was uneventful and the patient was discharged with no neurological deficit. Three years later, the patient suffered left frontal cerebral infarction (Fig. 2C). Cerebral angiography demonstrated a right basilar artery-superior cerebellar artery (BA-SCA) aneurysm which had not been seen on the previous study (Fig. 2D). The de novo aneurysm was followed up conservatively. Six years later, the patient suffered right cerebellar hemorrhage (Fig. 2E). The patient was a nonsmoker, with no family history of SAH or hypertension. Nine years later, she complained of general fatigue. Follow-up CT detected suspected regrowth of the de novo BA-SCA aneurysm, and confirmed by cerebral angiography (Fig. 2F). Neurological examination on admission was Hunt and Kosnik grade 0. The patient underwent endovascular surgery. The postoperative course was uneventful and she was discharged with no neurological deficit.

**Discussion**

Only 14 previous cases of de novo BA aneurysms have been reported (Table 1). We treated patients with de novo formation of a ruptured basilar tip aneurysm and an unruptured BA-SCA aneurysm. All but 2 of the 16 reported cases were female. The age at which the first SAH occurred ranged from 10–72 years (mean 42.8 years). The interval to the second SAH or the detection of de novo BA aneurysm ranged from 44 days to 20 years. Multiple aneurysms were present in 4 reported cases, and multiple de novo aneurysms were detected in 3 reported cases. Rupture of de novo aneurysms occurred in 11 of the 16 cases. De novo aneurysm in the posterior circulation appears to occur predominantly in young female patients. However, de novo aneurysms in the posterior circulation and in the anterior circulation following initial treatments have no clinical differences because no new risk factor is present.

Generally, the incidence and pathogenesis of de novo aneurysms are poorly understood. Known risk factors for

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**Table 1** Reported cases of de novo basilar artery (BA) aneurysms

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age (yrs)/Sex</th>
<th>Location of initial aneurysms</th>
<th>Location of de novo aneurysms</th>
<th>Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miller et al. (1985)[10]</td>
<td>49/F</td>
<td>lt ICA</td>
<td>BA-SCA, lt PCoA</td>
<td>11 yrs</td>
</tr>
<tr>
<td></td>
<td>41/F</td>
<td>rt MCA</td>
<td>BA tip, ACoA</td>
<td>5 yrs</td>
</tr>
<tr>
<td></td>
<td>48/F</td>
<td>rt ICA-PCoA</td>
<td>BA tip</td>
<td>20 yrs</td>
</tr>
<tr>
<td>Koeleveld et al. (1991)[8]</td>
<td>13/F</td>
<td>lt ICA tip, lt ICA-AChA</td>
<td>BA tip, lt MCA, lt ACA, rt ICA tip</td>
<td>13 yrs</td>
</tr>
<tr>
<td>Sugiura et al. (1997)[14]</td>
<td>65/F</td>
<td>ACoA</td>
<td>BA tip</td>
<td>13 yrs</td>
</tr>
<tr>
<td>Johnston et al. (1998)[4]</td>
<td>10/F</td>
<td>ICA</td>
<td>BA trunk</td>
<td>8 yrs</td>
</tr>
<tr>
<td>Lee and Brophy (2003)[9]</td>
<td>19/M</td>
<td>lt ICA, rt MCA</td>
<td>BA tip</td>
<td>6 yrs</td>
</tr>
<tr>
<td>Kang et al. (2005)[8]</td>
<td>30/F</td>
<td>rt ICA</td>
<td>BA trunk</td>
<td>7 mos</td>
</tr>
<tr>
<td>Fujimoto et al. (2005)[2]</td>
<td>72/F</td>
<td>rt MCA, lt ICA-PCoA, lt ICA cave</td>
<td>BA top</td>
<td>5 yrs</td>
</tr>
<tr>
<td>Shimokawara et al. (2007)[12]</td>
<td>58/F</td>
<td>ACoA, lt ICA-PCoA</td>
<td>lt BA-SCA</td>
<td>7 yrs</td>
</tr>
<tr>
<td>Kim et al. (2007)[7]</td>
<td>69/F</td>
<td>rt MCA</td>
<td>BA tip</td>
<td>1.2 yrs</td>
</tr>
<tr>
<td></td>
<td>41/F</td>
<td>lt ICA-PCoA</td>
<td>BA tip</td>
<td>16.6 yrs</td>
</tr>
<tr>
<td></td>
<td>36/F</td>
<td>rt M1</td>
<td>BA tip</td>
<td>9.8 yrs</td>
</tr>
<tr>
<td>Schebesch et al. (2008)[11]</td>
<td>371/M</td>
<td>ACoA</td>
<td>BA tip</td>
<td>44 days</td>
</tr>
<tr>
<td>Present Case 1</td>
<td>48/F</td>
<td>lt MCA</td>
<td>BA tip</td>
<td>5 yrs</td>
</tr>
<tr>
<td>Present Case 2</td>
<td>49/F</td>
<td>rt A1</td>
<td>rt BA-SCA</td>
<td>3 yrs</td>
</tr>
</tbody>
</table>

the formation of new aneurysms include hypertension, young age, female, smoking, Marfan syndrome, and moyamoya disease.\textsuperscript{2,7,10,11} In addition, abnormalities in the structure or function of cerebral blood vessels, arterial damage, and inflammation may be related to de novo aneurysm formation.\textsuperscript{4,13} Hemodynamic stress may play a major role in the pathogenesis because de novo aneurysm formation often occurs after ligation or trapping of major vessels, and after surgical removal of arteriovenous malformations.\textsuperscript{1,6,13} Massive vasospasm may also induce hemodynamic changes that ultimately result in the development of de novo aneurysm.\textsuperscript{11} However, no cause can be identified in most cases, as in the present patients. The majority of de novo aneurysms previously reported may represent growth of pre-existing lesions such as “tiny,” “blister,” or “infundibula” lesions that had been missed at the initial angiography.\textsuperscript{10} De novo aneurysms may be a special case of multiple aneurysms in which the lesions appear in series with time lags rather than simultaneously.

Once a cerebral aneurysm has been eliminated completely, the patient is thought to be cured. However, we sometimes encounter de novo aneurysms after initial treatment. The incidence of de novo formation and rupture of aneurysms is 60/100,000/yr, which is 6 times the incidence of SAH in the general population.\textsuperscript{10} Actually, the incidence of the de novo formation of aneurysms may be higher.\textsuperscript{10,15} Therefore, patients with known risk factors for de novo aneurysms should be strictly followed up to evaluate the possibility of de novo aneurysm, especially patients with multiple aneurysms and young patients, even after successful treatment of the initial aneurysms. In addition, the present and previously reported cases suggest that the status of the posterior circulation must be carefully examined, especially in patients with recurrent SAH. The interval for follow-up angiography is not clear, since the rate of growth of aneurysms is not predictable. In addition, magnetic resonance angiography and three-dimensional CT are better modalities, but may miss tiny aneurysms.

References


Address reprint requests to: Ichiro Kawahara, M.D., Department of Neurosurgery, Nagasaki Prefecture Shimabara Hospital, 7895 Shimokawashiri-machi, Shimabara, Nagasaki 855-0861, Japan.

E-mail: i-kwhr@umin.ac.jp