Early Development and Rupture of De Novo Aneurysm
—Case Report—

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Abstract

A 38-year-old non-smoker man presented with a ruptured aneurysm one month after clipping of a previous aneurysm. He was first admitted because of sudden onset of severe headache. Brain computed tomography showed subarachnoid hemorrhage. Angiography showed an aneurysm of the left anterior choroidal artery which was surgically clipped. Two weeks later, he was discharged without neurological deficits. One month after the initial hemorrhage, he was readmitted to the emergency room with stuporous mentality. Repeat angiography showed two aneurysms of the A2 portion of the left anterior cerebral artery which were not demonstrated by the initial angiography. The diagnosis was de novo aneurysms. The larger aneurysm was clipped and the other was coated. De novo aneurysm should be suspected if a patient with a previously clipped aneurysm complains of typical headache or any suggestive symptoms or signs of cranial nerve dysfunction, especially if known risk factors are present.

Key words: anterior choroidal artery aneurysm, de novo aneurysm, anterior cerebral artery aneurysm, multiple aneurysms

Introduction

The pathogenesis of intracranial saccular aneurysms involves two major factors: the presence of congenital developmental anomalies of the cerebral arteries brought about by the mechanical action of the blood stream and acquired arteriosclerosis. De novo aneurysm considered to be a special case of multiple aneurysms in which the lesions appear in series rather than in parallel. This de novo aneurysm develops at an interval of 3–20 years and is not detected by the previous angiography. The incidence and behavior of de novo aneurysms provide some insight into the poorly understood natural history of these lesions.

We describe a patient who developed two de novo aneurysms of the left A2 portion after clipping of a left anterior choroidal artery aneurysm at an interval of 30 days, and showed a clear evidence for the involvement of induced alterations of the arterial wall in the development of aneurysms in adults.

Case Presentation

A 38-year-old non-smoker man experienced sudden onset of severe headache at work. Brain computed tomography (CT) showed subarachnoid hemorrhage of Fisher grade III (Fig. 1 left). Angiography showed a 7 × 8 mm aneurysm of the left anterior choroidal artery (Fig. 1 right). The aneurysm was clipped on the second day after the ictus (Fig. 2 left). Two weeks later...
Fig. 2  left: Photograph during the first operation showing an anterior choroidal artery aneurysm with surrounding hematoma. The anterior choroidal artery aneurysm was clipped and the anterior cerebral artery and anterior communicating artery were dissected but there was no other aneurysm.  right: Photograph during the second operation showing a small aneurysm located in the left proximal A₂ portion and another larger aneurysm close by. The smaller aneurysm was coated and the larger aneurysm was clipped.

Fig. 3 Brain computed tomography scan one month after the initial hemorrhage showing intraventricular hemorrhage and subarachnoid hemorrhage.

Fig. 4  left: Left carotid angiogram revealing two aneurysms of the left A₂ portion. A small aneurysm (arrowhead) was located in the left proximal A₂ portion and another larger aneurysm (arrow) in the left A₂ portion.  right: Postoperative left carotid angiogram showing no vascular abnormality.

Later, he was discharged without neurological deficits.

One month after the initial hemorrhage, he was admitted to the emergency room with stuporous mentality. Brain CT demonstrated subarachnoid hemorrhage and intracerebral hematoma (Fig. 3). Angiography revealed two aneurysms of the left A₂ portion (Fig. 4 left). An emergency operation was performed and identified a small aneurysm located in the left proximal A₂ portion and another larger aneurysm close by. The smaller aneurysm was coated and the larger aneurysm was clipped (Fig. 2 right). Examination of a biopsy specimen of the aneurysmal wall excluded dissecting aneurysm. Postoperative angiography showed a clip on A₂ portion which had occluded the larger aneurysm (Fig. 4 right). He was discharged with mild right hemiparesis.

Discussion

The first case of de novo aneurysm occurred in a
### Table 1 Previous cases of de novo aneurysms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Author (Year)</th>
<th>Site</th>
<th>Initial disease or condition</th>
<th>De novo aneurysm</th>
<th>Interval (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Graf and Hamby (1964)</td>
<td>rt MCA aneurysm</td>
<td>It MCA aneurysm</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Gurdjian et al. (1965)</td>
<td>41 carotid ligations</td>
<td>1 case of mirror aneurysm on the opposite carotid siphon</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Du Boulay (1965)</td>
<td>carotid ligation</td>
<td>mirror aneurysm of the PcomA</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Somach and Shenkin (1966)</td>
<td>PcomA</td>
<td>opposite parasellar aneurysm</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Heiskanen and Marttila (1970)</td>
<td>It PcomA aneurysm</td>
<td>AcomA aneurysm</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Winn et al. (1977)</td>
<td>rt PcomA aneurysm, carotid ligation</td>
<td>It PcomA aneurysm</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Klemme (1977)</td>
<td>rt PcomA aneurysm, carotid ligation</td>
<td>It PcomA aneurysm</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Nukui et al. (1982)</td>
<td>rt PcomA aneurysm, carotid ligation</td>
<td>It PcomA aneurysm</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Hyun et al. (1993)</td>
<td>rt ACA aneurysm</td>
<td>It frontopolar artery aneurysm</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Larson et al. (1995)</td>
<td>rt PcomA aneurysm</td>
<td>rt MCA aneurysm</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td></td>
<td>intracavernous ICA aneurysm</td>
<td>AcomA aneurysm</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td></td>
<td>A₁-A₂ junction aneurysm</td>
<td>enlargement and rupture of A₁-A₂ junction aneurysm</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>


The patient who died of a ruptured aneurysm of the left middle cerebral artery. Three years before, the patient had undergone clipping surgery for a right middle cerebral artery aneurysm. Because the subsequent aneurysm was not visualized in the initial angiogram, the authors introduced the term de novo for this type of aneurysm. Thereafter, at least nine reports of de novo aneurysms have been published (Table 1). In our case, the two A₂ portion aneurysms developed one month after the clipping of a left anterior choroidal artery aneurysm. Previous cases appeared on the opposite internal carotid artery after carotid ligation for aneurysms of the internal carotid or posterior communicating artery with two exceptions.

The coexistence of aneurysms with sellar neoplasms has an incidence of 6%, or 11 aneurysms among 150 pituitary adenomas and 33 cranioophangiomas. The annual incidence of de novo aneurysm is calculated to be 100 per 100,000 patients with known aneurysm.

De novo aneurysms commonly occur in the 3rd and 4th decades of life at an interval of 3–20 years. In our case, the de novo aneurysms developed at a short interval of 30 days. We have treated 2000 patients with aneurysms during the last 20 years and experienced three cases of de novo aneurysms. The other two patients developed de novo aneurysm after clipping of the aneurysms at intervals of 8–10 years.

Three theories for the pathogenesis of de novo aneurysms have been proposed. First, defects in the media are known to be especially large in the contralateral side or mirror locations to an established lesion. Carotid ligation or other intracranial aneurysm surgery may cause changes in the balance and periodicity of pulsatile collateral flow to allow aneurysmal development at fairly predictable sites, which would otherwise remain as a “preaneurysmal” lesion. Second, de novo aneurysm may also result from the same inherent defect that caused the development of the preceding aneurysm. Medial defects, acquired injury of the internal elastic lamella, atherosclerosis, hypertension, and variations in the circle of Willis are all likely to predispose to further aneurysm formation. Third, the false negative rates of 1.8% to 3.8% of cerebral angiography in defecting aneurysms should be considered.

Smoking, oral contraceptive medication, and female sex hormones in general are all important contributors to the degenerative changes of vessels. The risk of subarachnoid hemorrhage in cigarette smokers is 3.7 to 5.7 times that in age-matched non-smokers. There are several common characteristics: most patients were young women and had multiple aneurysms; the junctional dilatations were located on the proximal portion of posterior communicating artery; and the junctional dilatation transformed...
into true aneurysm at an interval of 6 to 9 years.\textsuperscript{8)} However, our patient was a non-smoker.

Angiography should be considered if typical headache or any suggestive symptoms or signs of cranial nerve dysfunction develop or if special risk factors are present. The clinician should also suspect another aneurysm if a patient who was previously treated for aneurysms complains of any symptoms that suggest another lesion, even if the postoperative arteriography was normal. The present case demonstrated two cerebral aneurysms of the left A2 portion at a very short interval of 30 days after clipping of a left anterior choroidal artery aneurysm. Therefore, de novo aneurysms may arise at any time.

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References


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