Symptomatic Unruptured Cerebral Aneurysms: Features and Surgical Outcome

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Abstract

Development of less invasive imaging studies, such as magnetic resonance angiography, has increased the chances that unruptured cerebral aneurysms are found. The rupture risk of “symptomatic” aneurysms is higher than for “asymptomatic” aneurysms; so “symptomatic” aneurysms are more often surgically treated. Many reviews examine “asymptomatic” unruptured cerebral aneurysms, but few evaluate “symptomatic” aneurysms. The author has treated many patients with symptomatic unruptured cerebral aneurysms and found that improved cranial nerve signs can be expected if the surgical treatment is performed before the symptoms become irreversible; the critical period is approximately 3 months. It is important to suppress the pulsation of the aneurysms compressing the cranial nerves; both a clipping procedure and endovascular coiling are effective. Cranial nerve signs are more commonly the symptoms of unruptured cerebral aneurysms, but large to giant aneurysms can also be the causes of hemiparesis, hydrocephalus, epilepsy, or even cerebral infarction. This review summarizes the features and surgical outcome of symptomatic unruptured cerebral aneurysms.

Key words: cerebral aneurysm, symptomatic aneurysm, unruptured aneurysm, surgery, outcome

Introduction

Unruptured cerebral aneurysms can be divided into “asymptomatic” and “symptomatic.” The development of less invasive imaging methods, such as magnetic resonance angiography (MRA), has increased the chances that “asymptomatic” unruptured cerebral aneurysms are found and the management of these aneurysms remains one of the most controversial topics in neurosurgery. Patients with “symptomatic” unruptured cerebral aneurysms often show neurological symptoms such as cranial nerve palsy, epilepsy, and embolic cerebral ischemia. As the risk of rupture is higher than for “asymptomatic” aneurysms, surgical intervention is more commonly considered. We have reported our surgical cases of “symptomatic” cerebral aneurysms from the view point of visual dysfunction, and large and giant aneurysms located in the intracavernous, paraciloid and petrous portions of the internal carotid artery.

This review article will summarize our surgical experiences regarding “symptomatic” unruptured cerebral aneurysms and review the literature based on keywords such as headache, visual symptom, oculomotor, cavernous sinus-related and other cranial nerves, compression to surrounding brain, epilepsy, and cerebral ischemia. This review will focus on saccular aneurysms only and dissecting aneurysms will not be discussed.

Headache and Unruptured Cerebral Aneurysms

There are a number of reports regarding headache and unruptured cerebral aneurysms, although no subarachnoid hemorrhage was present in these patients. About half of the patients with unruptured cerebral aneurysms present with headache; two-thirds of these headaches are chronic and one-third are acute. The acute headaches are similar to those of subarachnoid hemorrhage but without stiff neck. This type of headache has been described as thunderclap headache and may be a warning of subsequent rupture. The mechanism for acute headache from unruptured cerebral aneurysms is most probably local thrombosis of aneurysms or local inflammation of the meninges. Headache associated with the dura of the cavernous sinus or other skull base regions often improves after surgical treatment. Coil embolization in 10 patients with large internal carotid artery aneurysms with headache or cranial nerve signs resulted in headache disappearing in all patients although
aneurysm volume was larger than that before surgery due to the coil embolization; the improved headache may be due to reduced transmitted pulsations.\(^{29}\) Recently, chronic headache was assessed after surgical treatment of unruptured cerebral aneurysms. Whether clipping or coiling was employed, surgical treatment resulted in relief of headache in the majority (90\%) of patients with preoperative headaches.\(^{56}\)

**Visual Dysfunction and Unruptured Cerebral Aneurysms**

Symptomatic unruptured cerebral aneurysms include many that compress the optic nerve or optic tract and show visual dysfunction. Commonly, they are large or giant aneurysms, because the symptom is directly due to compression of the visual pathway. We have summarized our experience with these aneurysms and reported their features and surgical outcomes along with the separate locations of the aneurysms.\(^{20}\) The most common location was the ophthalmic artery followed by the intracavernous and anterior communicating arteries. The types of visual field defect and the degrees of reduced visual acuity were highly variable, without typical clinical presentation. The interval between the onset of symptoms and surgical treatment was the only factor identified that affected the clinical outcome of the aneurysms presenting with visual dysfunction. All cases that showed improvement of visual function were surgically treated within 3 months of the onset of symptoms.\(^{20}\) Recovery of visual function can most often be expected when surgical treatment is performed expeditiously, before the visual dysfunction becomes irreversible. Intravascular surgery for patients with giant aneurysms showing visual dysfunction stressed the importance of early diagnosis and surgical intervention due to the poor outcome of patients without treatment.\(^{96}\)

The initial symptom of ophthalmic artery aneurysms is commonly related to visual dysfunction due to the anatomical location of the aneurysm. In our experience, visual dysfunction was observed in 25\% of the cases.\(^{20}\) Analysis of 100 cases of ophthalmic artery aneurysms found 32\% showed visual dysfunction and most were unruptured and giant.\(^{28}\) In addition, early surgical intervention is desirable in cases of ophthalmic aneurysm with rapid deterioration of visual function to control the symptom and to prevent subsequent subarachnoid hemorrhage.\(^{12,20}\)

The most common symptoms of intracavernous aneurysms are related to the third, fourth, fifth, and sixth cranial nerves;\(^{63}\) visual dysfunction is not so common. We previously noted that visual dysfunction was found in 21\% of the cases with intracavernous giant aneurysms.\(^{20}\) Others report similar figures; two cases among 19,\(^ {74}\) one in ten cases,\(^ {71}\) and 5 cases among 26\(^ {96}\) demonstrated visual dysfunction associated with giant intracavernous aneurysms.

Anterior communicating artery (AcomA) aneurysms sometimes cause visual dysfunction if they become large or giant and directly compress the optic pathway.\(^ {20,71,74,96}\) The types of dysfunction are variable and there are no clear patterns. The main cause of visual dysfunction is thought to be related to the direct compression of the visual pathway, but the blood supply of the optic nerve or chiasm may also be compromised by occlusion or distortion of the ophthalmic artery or other small internal carotid (IC) arterial branches in the parasellar/suprasellar region.\(^ {20}\) The visual dysfunction caused by cerebral aneurysms tends to fluctuate over time, and possible explanations for this phenomenon include intraneurysmal thrombosis, intermittent dilation of the aneurysm, or alterations in intracranial pressure.\(^ {54}\)

Symptom fluctuation is characteristic of visual dysfunction resulting from cerebral aneurysm and may be different from that caused by brain tumors, which is typically steadily progressive.\(^ {54}\)

Direct penetration of the optic nerve by a cerebral aneurysm can also cause visual dysfunction. We reported a case of chiasmal penetration by an AcomA aneurysm,\(^ {20}\) and several others reported cases of optic nerve penetration by IC ophthalmic aneurysms.\(^ {11,30,49,53}\)

As mentioned above, the most common aneurysms showing visual dysfunction are located in the paraclinoid region, including IC ophthalmic aneurysms, and are usually large or giant.\(^ {20,24}\) The suction decompression technique is very useful for clipping these aneurysms.\(^ {9,24,91}\) The author has performed direct clipping of large and giant paraclinoid aneurysms using the suction decompression procedure in 24 cases (Table 1). The average age was 57 years old and patients were mostly female. Thirteen patients showed visual dysfunction preoperatively. Preoperative balloon test occlusion of the ipsilateral IC was positive in two patients. Seven of 13 cases showing preoperative visual dysfunction improved postoperatively and all cases were clipped within 3 months from onset. Three patients with postoperative deterioration of vision had only light perception preoperatively; thus, the deterioration did not affect the quality of life of these patients. As shown in Table 1, fenestrated clips, especially short and curved ones, were most commonly used for these aneurysms. For suction decompression, the average

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Table 1 Characteristics of 24 cases of large and giant paraclinoid internal carotid artery (IC) aneurysms treated with suction decompression

<table>
<thead>
<tr>
<th>Age</th>
<th>47–70 (average 57) yrs</th>
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<tbody>
<tr>
<td>Sex (male:female)</td>
<td>3:21</td>
</tr>
<tr>
<td>Size</td>
<td></td>
</tr>
<tr>
<td>large (15–24 mm)</td>
<td>12 cases</td>
</tr>
<tr>
<td>giant (≥ 25 mm)</td>
<td>12 cases</td>
</tr>
<tr>
<td>Onset symptoms</td>
<td></td>
</tr>
<tr>
<td>SAH</td>
<td>6 cases</td>
</tr>
<tr>
<td>visual disturbance</td>
<td>13 cases</td>
</tr>
<tr>
<td>incidental</td>
<td>5 cases</td>
</tr>
<tr>
<td>Time before clipping</td>
<td>1 day–6 mos</td>
</tr>
<tr>
<td>Positive preoperative BTO</td>
<td>2 cases</td>
</tr>
<tr>
<td>Postoperative visual function*</td>
<td></td>
</tr>
<tr>
<td>improved</td>
<td>7 cases **</td>
</tr>
<tr>
<td>unchanged</td>
<td>3 cases</td>
</tr>
<tr>
<td>deteriorated</td>
<td>3 cases</td>
</tr>
<tr>
<td>Types of clips used</td>
<td></td>
</tr>
<tr>
<td>fenestrated</td>
<td>18 cases</td>
</tr>
<tr>
<td>straight</td>
<td>6 cases</td>
</tr>
<tr>
<td>IC occlusion time</td>
<td>6–22 (average 16) min</td>
</tr>
<tr>
<td>Suctioned blood volume</td>
<td>25–300 (average 90) ml</td>
</tr>
<tr>
<td>Postoperative ischemic complication</td>
<td>1 case</td>
</tr>
<tr>
<td>Glasgow Outcome Scale</td>
<td></td>
</tr>
<tr>
<td>good recovery</td>
<td>22 cases</td>
</tr>
<tr>
<td>moderate disability</td>
<td>1 case</td>
</tr>
<tr>
<td>severe disability</td>
<td>1 case</td>
</tr>
</tbody>
</table>

*In 13 cases with preoperative disturbance. **All treated within 3 months from onset. BTO: balloon test occlusion, SAH: subarachnoid hemorrhage.

IC occlusion time was 16 minutes and the average suctioned blood volume was 90 ml. A representative case is shown in Figs. 1–3.

**Oculomotor Palsy and Unruptured Cerebral Aneurysms**

The most common aneurysms causing oculomotor palsy are internal carotid-posterior communicating (IC-PC) aneurysms, which are estimated to represent 90% of the cases. Intracavernous and bifurcation aneurysms (6%) and basilar tip aneurysms (3%) caused most of the other cases.

Preoperative oculomotor palsy was complete in 70% of the cases and partial in 30%, including pupillary-sparing forms (normal pupillary reactions but with extraocular muscle paresis and ptosis) in 5% to 15%. In the pupillary-sparing forms of oculomotor palsy, routine angiography is not recommended because the prevalence of aneurysm is low, but recent increases in the quality of MRA may justify checking the possibility of aneurysm even in such cases. It is important because oculomotor palsy may occur as a result of relatively small aneurysms in this location. Sixteen patients with small, unruptured cerebral aneurysms, 5.8 mm on average, presented with oculomotor palsy. Retrobulbar pain is also a prominent feature in the majority of aneurysmal oculomotor palsies. The same symptom was found in 11 of 16 cases and 7...
of 8 cases. Cadaveric and animal studies demonstrated that the oculomotor nerve contains sensory fibers that enter the brain stem and reach the spinal trigeminal nucleus, suggesting that retrobulbar pain observed in the cases with ipsilateral IC-PC aneurysms is related to these sensory fibers.

Whether or not oculomotor palsy improves after surgery depends on the period between the onset and surgery: the earlier the surgery, the higher the possibility of improvement. About 64% of cases treated within 2 weeks of onset had complete recovery from oculomotor palsy, in contrast to 30% of cases treated between 14 and 30 days and only 14% of those treated later than 30 days. The time between onset and surgical therapy is very important; surgical intervention was performed within 4.7 ± 3.3 days in cases with complete recovery, 24.2 ± 15.5 days in cases with partial recovery, and 41.0 ± 12.7 days in cases with no recovery.

Direct clipping is commonly chosen as the method of surgical intervention for IC-PC aneurysms; there are also reports of coil embolization for these aneurysms. Coil embolization was performed in 11 patients with unruptured IC-PC aneurysms and oculomotor palsy and detailed neuroophthalmological analyses carried out. Although complete resolution of the oculomotor palsy did not occur, residual partial oculomotor palsy did not cause diplopia with primary gaze and significant ptosis did not persist; so that oculomotor palsy improved comparably to the recovery observed after surgical clipping. Older age and the presence of microvascular risk factors, such as hypertension, smoking, and diabetes, seem to be detrimental to oculomotor palsy recovery.

Although oculomotor palsy usually occurs with direct compression of the nerve by the aneurysm, a rare case of an unruptured IC-PC aneurysm increased in size and penetrated the oculomotor nerve, lead to the nerve palsy. As mentioned above, there are several reports of aneurysm penetration of optic nerve, but this is the only report showing oculomotor penetration.

### Giant Intracavernous Aneurysms

The natural history of unruptured intracavernous aneurysms is generally considered to be good and asymptomatic aneurysms or aneurysms not facing the subarachnoid space are often conservatively treated. However, if the size of the unruptured aneurysm increases to large or giant, many of these aneurysms cause visual dysfunction, eye movement disorder, or facial and retrobulbar pain, and sometimes extend into the subarachnoid space, inducing subarachnoid hemorrhage when ruptured, or massive nasal bleeding. We should consider surgical intervention for these types of aneurysms. Analysis of 57 cases reported the female dominance of these aneurysms (80%), with double vision (90%), retrobulbar pain (60%), headache (20%), and visual dysfunction (14%) as the main symptoms, and the cause of these symptoms was compression by giant aneurysms and ischemia. When the period between the onset of cranial nerve signs/symptoms and therapy becomes longer, the symptoms tend to be irreversible; so these giant intracavernous aneurysms should be treated surgically within a couple of months from onset.

There are a number of case reports that are interesting from the view point of their symptomatic aspects: a patient with a petrous carotid artery extending into the cavernous sinus who exhibited cavernous sinus syndrome and a patient with Horner’s syndrome and the aneurysm located in the posterior cavernous sinus.

Various types of surgical treatments for intracavernous giant aneurysms have been evaluated historically. Although ligation of the internal carotid or common carotid artery at the neck of the patients were the main choices previously, there are many recent options as well, including direct ligation or intravascular ligation combined with extracranial-intracranial (EC-IC) bypass. When selecting surgical treatment for these giant aneurysms, the general concept is that the method should be selected based on the result of the balloon test occlusion (BTO) of the ipsilateral IC; we have been using this concept for our treatment algorithm (Fig. 4). BTO of the ipsilateral IC is performed for 30 minutes (20 minutes in another report), and if the patient shows any neurological deficit, high flow bypass and IC ligation or trapping is chosen. In patients without neurological deficit, single photon emission computed tomography.

![Giant Intracavernous Aneurysms](image-url)
emission computed tomography (SPECT) is performed along with the BTO; if the SPECT shows asymmetry, high flow bypass plus IC ligation or trapping is chosen for younger patients and low flow bypass (superficial temporary artery-middle cerebral artery [STA-MCA] anastomosis) is chosen for older patients. When the SPECT shows no asymmetry, either low flow bypass plus IC ligation or trapping or IC ligation only is chosen. Patients whose cortical cerebral blood flow (CBF) decreased to below 30 ml/100 g/min were considered a moderate risk and underwent STA-MCA bypass followed by carotid occlusion; patients with CBF levels greater than 30 ml/100 g/min were considered a low risk and underwent only carotid occlusion. Residual flow of less than 70–75% indicated high flow bypass, and between 70–75% and 90% indicated STA-MCA bypass.

We performed surgical therapies for 27 patients with giant intracavernous aneurysms based on the above treatment algorithm. A representative case is shown in Fig. 5. Abducens nerve palsy was the most commonly observed preoperative disturbance, occurring in 20 cases; oculomotor nerve disturbances were observed in 12 cases, optic nerve in 6 cases, trigeminal nerve in 6 cases, and trochlear nerve in 5 cases. These cranial nerve signs improved in most of the cases in which surgical treatment was performed within three months after onset of the symptom. The period of three months seems to be significant, because performing surgical therapy is recommended within this time. Ipsilateral ischemic complication occurred in only one of 26 cases and similarly good results were reported by others; thus, it is important to determine the surgical treatment based on the results of a preoperative BTO and to perform the surgery within three months of onset to obtain good surgical outcomes in patients with giant intracavernous aneurysms.

Historically, common carotid (CC) or IC ligations were most commonly performed to treat giant intracavernous aneurysms. About 49% of cases treated with IC ligation and 28% of those treated with CC ligation showed postoperative ischemic complications, whereas such complications occurred in 40% of IC ligations and 14% of CC ligations. CC ligations can be performed more safely than IC ligations, but IC ligations promote thrombosis of the aneurysm more rapidly than do CC ligations. Delayed ischemic complications after CC or IC ligations have been reported, and the chances of performing CC or IC ligation without bypass surgery have been decreasing in recent years. A long-term follow-up study of patients with intracavernous aneurysms treated by proximal occlusion found that 2 of 11 cases developed newly formed aneurysms; similar reports suggest that long-term follow-ups of the effects of IC ligation are necessary.

The mainstay of surgical treatment for intracavernous giant aneurysms in recent years has been IC li-
gation combined with either STA-MCA anastomosis or high flow bypass, due to the development of microsurgical techniques, and because preoperative flow reserve can be analyzed more precisely by SPECT in the presence of BTO. BTO or SPECT with BTO is performed preoperatively and low risk patients with adequate cerebral blood flow reserve commonly receive STA-MCA anastomosis, and high risk patients with less cerebral blood flow reserve commonly receive high flow bypass. Younger patients more commonly receive high flow bypass. Either the saphenous vein or the radial artery is used for high flow bypass, and long-term patency is reported for both.

**Other Cranial Nerve Signs in Unruptured Cerebral Aneurysms**

Unruptured aneurysms causing visual dysfunction, oculomotor palsy and cavernous sinus syndrome have been summarized so far. Unruptured aneurysms causing other cranial nerve signs or interesting case reports from the viewpoint of aneurysm location and its symptom will be discussed here.

Trochlear nerve palsy may be caused by unruptured aneurysms. One patient had a large, partially thrombosed aneurysm at the P2/P3 segment of the right posterior cerebral artery; in addition to ipsilateral trochlear nerve palsy, the patient showed left homonymous hemianopsia and left hemisensory disturbance. Both the trochlear nerve palsy and homonymous hemianopsia were attributed to direct compression by the aneurysm, whereas the sensory disturbance may have been caused by embolization of an aneurysmal thrombus into the right thalamic perforators. Coil embolization induced gradual improvement in these neurological deficits. Trochlear nerve palsy was induced by peripheral superior cerebellar artery aneurysms located in the perimesencephalic cistern in two patients, and the latter patient showed improvement after surgical clipping. As mentioned previously, intracavernous aneurysms usually cause cavernous syndrome, including trochlear nerve palsy. Pure trochlear nerve palsy from an intracavernous aneurysm was only reported in two cases; in one, the symptom improved during the natural course.

There are several reports of unruptured cerebral aneurysms causing trigeminal neuralgia. A large IC-PC aneurysm directly compressed the root entry zone of the trigeminal and facial nerves; these symptoms disappeared 3 months after clipping. An anterior inferior cerebellar artery (AICA) aneurysm compressed the trigeminal nerve and caused typical trigeminal neuralgia in the V2 and V3 regions. This aneurysm caused subarachnoid hemorrhage and clipping resolved the trigeminal neuralgia.

Although the majority of unruptured cerebral aneurysms causing abducens palsy are intracavernous aneurysms, a case of unruptured vertebral artery aneurysm caused ipsilateral abducens palsy. Intraoperative observation confirmed the presence of a 1.3-cm diameter aneurysm compressing the abducens nerve.

Several groups reported unruptured aneurysms that caused hemifacial spasm. “Symptomatic” hemifacial spasm caused by brain tumors, cerebral arteriovenous malformations (AVMs) or cerebral aneurysms comprise 0.4% to 2.2% of all hemifacial spasm patients. Patients with vertebral artery-posterior inferior cerebellar artery (VA-PICA) aneurysms that showed ipsilateral hemifacial spasm, which disappeared after clipping the aneurysms.

Reports of cerebral aneurysms associated with the onset of hearing disturbance also exist. Although there are reports of distal AICA aneurysms leading to hearing disturbance, most cases are associated with subarachnoid hemorrhage, and cases of unruptured aneurysms including such symptoms were rare. One case of an IC petrous segment aneurysm was associated with the onset of hearing disturbance. Cerebral aneurysms in this location can cause several types of cranial nerve signs depending on the direction of the enlargement; we reported a rare case causing cavernous sinus syndrome. A review of reports of large IC petrous segment aneurysms found that the most common initial symptom was hearing disturbance (42%), followed by trigeminal dysfunction (19%), abducens palsy (17%), and facial palsy (14%).

**Unruptured Aneurysm Showing Signs of Compression to Surrounding Brain Regions Other Than Cranial Nerves**

If an unruptured cerebral aneurysm increases in size to compress the surrounding brain regions, causing a mass effect, it sometimes induces rapid deterioration of neurological symptoms. One of the causes of this rapid deterioration is acute intraluminal thrombosis, which is related to the progressive apposition of intramural and/or intraluminal thrombotic material as a result of small hemorrhages within the wall or intraluminal turbulent flow. Two giant middle cerebral artery (MCA) aneurysms causing massive surrounding edema and acute intraluminal thrombosis leading to aphasia and hemiparesis were reported in 1984. Although sur-
gical resection of the aneurysm was performed in both cases, the outcomes were not good. Three similar cases (1 AcomA aneurysm, 2 MCA aneurysms) showing acute thrombosis with surrounding edema were all treated by partial excision of the intraluminal thrombus, bypass and IC occlusion, and conservative medication with heparin. All three patients showed full neurological recovery after treatment. The outcomes of surgical treatments of 202 unruptured cerebral aneurysms included 36 cases with mass effects from aneurysms that ranged from 11 mm to 25 mm in 7 cases and more than 25 mm in 29 cases. Detailed descriptions of the "mass effects" were not given, but cranial nerve signs may have been involved in these cases.

An unruptured cerebral aneurysm located at the terminal section of the left IC caused right hemisensory disturbance. The aneurysm was 20 mm in diameter and the bulbo-fundus projected superiorly into the left thalamus; symptoms disappeared after clipping. Local irritation of the thalamic nuclei was considered to be the cause of the hemisensory disturbance.

From the viewpoint of endovascular surgery, the efficacy of endosaccular aneurysm occlusion was assessed in 26 unruptured aneurysms causing mass effect. Among these, 6 of the aneurysms caused compression symptoms other than cranial nerve signs: AcomA aneurysms caused dementia, basilar tip and IC tip aneurysms caused hemiparesis, and PICA aneurysms caused ataxia and hemiparesis. All aneurysms were giant except one, and the surgical results were good; the symptoms disappeared in 3 and improved in 3. A series of 73 basilar tip aneurysms were treated by endovascular surgery using coils. Eight were unruptured aneurysms with mass effect; detailed analyses of the symptoms were not described.

Unruptured cerebral aneurysms rarely cause hydrocephalus; and most of such cases are large or giant basilar tip aneurysms that are commonly difficult to clip. To alleviate hydrocephalus, a ventriculo-peritoneal (V-P) shunt is often performed, but fatal rupture of the aneurysm occurred due to reduced intracranial pressure after the V-P shunt. Endovascular coil embolization was performed for a large basilar tip aneurysm that was causing obstructive hydrocephalus. Dementia and gait disturbance improved immediately and the ventricular size was reduced, which was attributed to reduced intraaneurysmal pressure induced by the coiling, and this patient did not require a V-P shunt.

Some IC aneurysms extending toward the sella turcica cause pituitary dysfunction. A 2-cm diameter paraclinoid aneurysm caused headache, left temporal hemianopsia, and galactorrhea. In addition to reduced reactions of thyroid-stimulating hormone and luteinizing hormone, marked hyperprolactinemia (more than 300 μg/l, which is compatible with that of prolactinoma) was observed. The increased prolactin was attributed to hypothalamic or pituitary stalk compression by the aneurysm, resulting in interference with the delivery of prolactin-inhibiting factor to the pituitary, and to a putative hypothalamic prolactin-stimulating factor. The prolactin level was normalized after the aneurysm was clipped. The authors recommended that if there are any clinical suspicions of a carotid aneurysm in a patient with hyperprolactinemia, that an angio-gram or MRI should be performed.

Secondary chorea may result from focal basal ganglion lesions, including those due to giant cerebral aneurysms. Parent artery occlusion was performed for the treatment of a giant MCA aneurysm using coils and balloons. The preoperative symptom was chorea due to the aneurysm compressing the basal ganglia, thalamus, and midbrain; the symptom was alleviated postoperatively.

**Unruptured Cerebral Aneurysms Causing Epilepsy**

Sometimes epilepsy is the initial symptom of unruptured cerebral aneurysms. This can be due to several mechanisms: compression or ischemia of the cerebral tissue surrounding the aneurysm, calcification of the aneurysmal wall, or gliosis in the surrounding brain due to subclinical, repeated microhemorrhages. In one series, 10 of 202 cases (5%) showed epilepsy as an initial symptom caused by aneurysms between 11 and 25 mm in 4 cases and more than 25 mm in 6 cases. Two cases of such aneurysms caused seizures; one MCA aneurysm and one IC-PC aneurysm. Based on intraoperative observations, the epilepsy was attributed to gliosis around the aneurysm via subclinical hemorrhages in the MCA case and local compression of the temporal lobe by the aneurysm in the IC-PC case. Complex partial seizures due to temporal lobe compression and injury occur more commonly in patients with large or giant MCA aneurysms, possibly due to the lower threshold for epilepsy in the injured mesial temporal lobe. A case of complex partial seizures was associated with an unruptured thrombosed basilar tip aneurysm. An electroencephalogram demonstrated that the focus of the epilepsy was located in the left temporal lobe; the cause of epilepsy was considered to be microinfection in the temporal lobe due to the migration of an aneurysmal embolus through the posterior cerebral artery.

Epilepsy often disappears after clipping of the...
Unruptured Cerebral Aneurysms Causing Cerebral Infarction

Unruptured aneurysms can be the cause of embolisms at the periphery resulting from intraluminal thrombi that, due to delayed or turbulent flow inside, migrate and occlude the peripheral artery, which occurs in 3.0% to 6.3% of unruptured aneurysms.\(^7,5,7,7,100\) Analysis of 269 cases of unruptured aneurysms found 9 cases (3.3%) of infarction due to migrated thrombus from the aneurysm\(^7,5\); four were MCA aneurysms, three were IC aneurysms, and one each of posterior cerebral artery aneurysm and vertebral artery aneurysm. The sizes ranged from 5 to 45 mm (average 12.5 mm). Six of the nine patients were treated surgically (two experienced infarctions related to the intraoperative manipulation) and two were treated conservatively with aspirin. There were no new ischemic events observed during the follow-up period (average 38 months), regardless of the therapeutic options. Review of 41 previously reported cases found no superiority of surgical treatment over conservative therapy for preventing the recurrence of ischemic events. The risk of recurrence was low regardless of which treatment was chosen. However, surgical treatments may be effective in preventing such recurrences.\(^42,81\) If surgical treatment is considered for these aneurysms, not only clipping, but also thrombectomy, trapping, or bypass should be considered depending on the circumstances of each case, and meticulous care should be taken to avoid the migration of intraluminal thrombi into the periphery.

Thrombus in a giant unruptured aneurysm may extend over the orifice of the aneurysm and occlude the surrounding cerebral arteries. Such a case involved the onset of left hemiparesis, progressive consciousness disturbance, and finally death.\(^13\) A thrombosed AcomA aneurysm and cerebral infarction in the bilateral anterior cerebral artery region and left MCA region were observed by MRI. Autopsy revealed a packed thrombus in the aneurysm and in the lumen of the right A1 and bilateral A2 segments of the anterior cerebral artery. The orifice of the aneurysm was small (1.5 mm) compared to the giant size of the aneurysm; so low flow, turbulent flow, and low shear force might have caused accumulation of platelets, endothelial injury, and promoted thrombosis formation. Two cases of spontaneous IC occlusion due to giant IC aneurysms were reported.\(^82\) IC stretching and compression by the giant aneurysms, which increased in size, might have caused the thrombosis of the parent artery.

A left giant MCA aneurysm was associated with the onset of ischemic symptoms. A stretched and collapsed branch of the MCA attached to the dome of the aneurysm expanded after clipping of the aneurysm and the symptom improved.\(^65\) The ischemic event might have been due to flow disturbances in the stretched branch of the MCA. A similar mechanism was also reported. The blood flow in the stretched lenticulostriate artery attached to the dome of A1 aneurysm recovered after clipping, and the patient showed improved right sensory disturbance.\(^73\)

Summary

This review summarizes our own experiences\(^18–25\) and reviewed the literature regarding symptomatic unruptured cerebral aneurysms and their surgical outcomes. Surgical therapy is more commonly recommended for these aneurysms, because the rupture ratio is higher than for asymptomatic aneurysms.\(^100\) The symptoms observed are mostly due to direct compression of the nerve or brain by the aneurysms, but sometimes are related to aneurysm-induced changes in blood flow or ischemia. If we expect to improve symptoms after surgery, it is important to keep the period from onset to therapy short and to perform surgery before existing symptoms become irreversible.

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