

# **Intracerebral Hemorrhage Caused by Ruptured Intracavernous Carotid Artery Aneurysm**

## **—Case Report—**

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### **Abstract**

**A 76-year-old woman presented with an enlarged right intracavernous carotid artery aneurysm first identified incidentally when she was hospitalized for thalamic hemorrhage. She was managed conservatively for 1 year, then suffered right total ophthalmoplegia associated with enlargement of the aneurysm. Two months later, she became comatose due to intracerebral hemorrhage in the right temporal lobe. Magnetic resonance imaging demonstrated further enlargement of the aneurysm. Emergency craniotomy found the lateral dural wall of the cavernous sinus was markedly expanded and torn by compression from the aneurysm. Rupture of the aneurysm into the intradural space through this dural defect was confirmed. The aneurysm was trapped after high-flow bypass, but the patient did not recover and died. Symptomatic enlarged intracavernous carotid artery aneurysm is potentially fatal and may indicate prompt surgical management.**

Key words: intracavernous carotid artery aneurysm, rupture, intracerebral hemorrhage

### **Introduction**

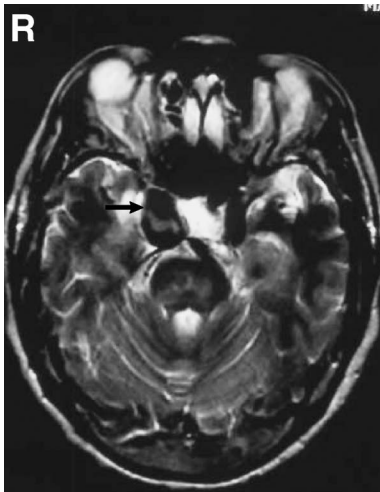
Intracavernous carotid artery (ICCA) aneurysms account for 3% to 11% of all intracranial aneurysms and for 14% of all internal carotid artery aneurysms.<sup>3,9-13)</sup> ICCA aneurysms may remain asymptomatic or manifest as symptoms caused by mass effects, such as symptoms of cranial nerves II, III, IV, V, or VI, depending on the direction of the aneurysm growth,<sup>2,9)</sup> or rupture, resulting in carotid-cavernous fistula but not in subarachnoid hemorrhage (SAH). Therefore, this type of aneurysm is believed to be benign in nature.<sup>6,7)</sup> However, rupture may result in subdural hemorrhage (SDH),<sup>1,2,4,14)</sup> SAH,<sup>8)</sup> or intracerebral hemorrhage (ICH),<sup>5)</sup> which are fatal in almost all cases.

We describe a case of ICCA aneurysm which enlarged progressively and finally caused fatal rupture into the temporal lobe.

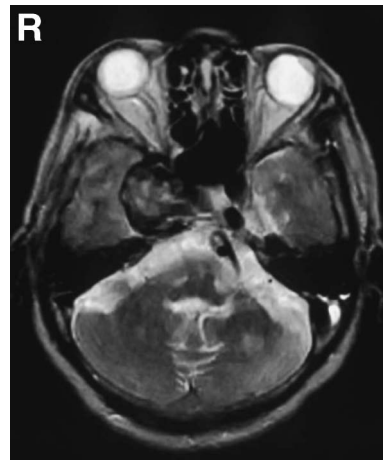
### **Case Report**

A 76-year-old woman was admitted to our hospital after sudden onset of dysarthria and nausea. Computed tomography (CT) disclosed left thalamic hemorrhage and a right cavernous sinus mass. Magnetic resonance (MR) imaging and MR angiography showed a right ICCA aneurysm of 22 mm in size (Fig. 1). Moreover, the left internal carotid artery and basilar artery were elongated. She had no symptoms related to the aneurysm, so was managed conservatively. At the 6-month follow up, MR imaging showed the aneurysm had not changed in size and the patient remained symptom free. Carotid angiography revealed an aneurysm of 28 mm in size originating from the C<sub>4</sub>-C<sub>5</sub> portion of the right internal carotid artery (Fig. 2).

One year later, she developed right total ophthalmoplegia. MR imaging demonstrated enlargement of the aneurysm to 30 mm (Fig. 3). However, the patient and her family refused any further studies. Two months later, she gradually became drowsy over 1 week and she was hospitalized. On admission, her Glasgow Coma Scale score was 9.



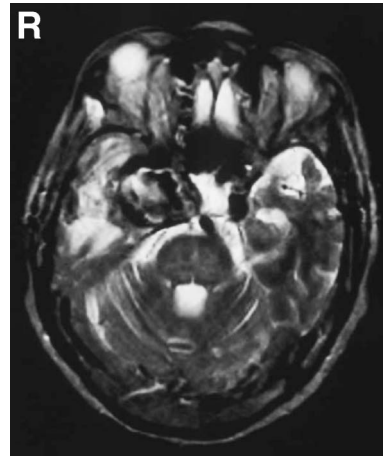
**Fig. 1** Axial T<sub>2</sub>-weighted magnetic resonance image showing a hypointense aneurysm in the right cavernous sinus (arrow).



**Fig. 3** Axial T<sub>2</sub>-weighted magnetic resonance image showing the enlarged aneurysm.



**Fig. 2** Right common carotid angiogram, anteroposterior view, showing the same aneurysm.

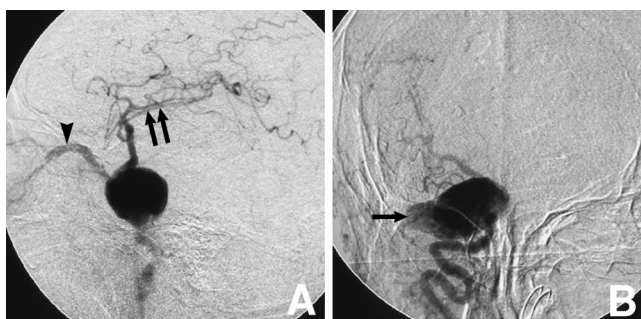


**Fig. 4** Axial T<sub>2</sub>-weighted magnetic resonance image demonstrating further enlargement of the aneurysm and severe edema.

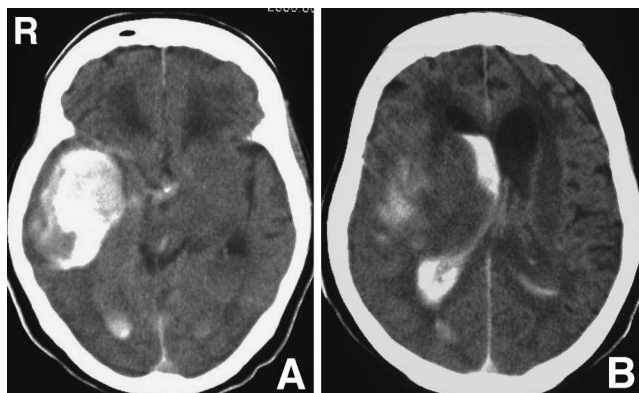
MR imaging demonstrated further enlargement of the aneurysm to 40 mm, especially laterally, which had resulted in severe edema in the temporal lobe (Fig. 4). She became comatose on transfer for angiography. Angiography showed that the aneurysm had extended laterally into the middle fossa and was associated with carotid-cavernous fistula. The middle cerebral arteries were markedly displaced and the blood flow was sluggish (Fig. 5). CT immediately after angiography demonstrated a large hematoma and extravasation of the contrast medium into the temporal lobe and ventricles, suggesting transdural rupture of the aneurysm (Fig. 6).

Emergent surgery was performed. The bilateral

lateral ventricles were drained and a high-flow bypass with radial artery graft was interposed between the external carotid artery and the M<sub>2</sub> portion of the middle cerebral artery. Finally, the aneurysm was trapped at the cervical internal carotid artery and the ophthalmic segment of the carotid artery. Removal of the hematoma in the temporal lobe showed that the lateral dural wall of the cavernous sinus had protruded into the middle fossa, and demonstrated a ovale shaped dural defect sized 15 × 20 mm around the protrusion. Thin walled aneurysm dome appeared through the defect and the rupture point in the wall was confirmed. Postoperative angiography demonstrated that the aneurysm had disappeared and the arterial graft was patent.



**Fig. 5** Right common carotid angiograms, (A) lateral view showing the severely stretched middle cerebral arteries (arrows) and the superior ophthalmic vein in the arterial phase (arrowhead), and (B) anteroposterior view showing the laterally protruding aneurysm dome (arrow) in the middle fossa.



**Fig. 6** Axial computed tomography scans showing (A) a large hematoma with extravasation of the contrast medium in the right temporal lobe, and (B) the hematoma in the lateral ventricle.

However, the patient did not recover and died 7 days after surgery. No autopsy was performed.

### Discussion

The natural course of ICCA aneurysms most frequently involves painful or painless ophthalmoplegia and trigeminal neuralgia, which follow a variable course if treated conservatively. The symptoms improved in 4 of 10 cases of initially symptomatic ICCA aneurysms, remained unchanged in 2, and worsened in 4.<sup>10</sup> Ophthalmoplegia remained stable in 27 of 59 cases, worsened in 29, and one developed SAH.<sup>7</sup> Symptoms remained unchanged in three of four cases, aggravated in one, and SAH occurred in one.<sup>15</sup> One of the five cases improved, two remained

unchanged, one worsened, and one died of ICH.<sup>13</sup> All symptomatic ICCA aneurysms were of the giant type.<sup>7,15</sup>

The relationship between the changes in symptoms and in aneurysm size is not clear. Although the symptoms may improve or deteriorate without changes in aneurysm size, enlargement of the aneurysm tends to be associated with deterioration of the symptoms. Such variations in clinical manifestations may suggest that the cranial nerves are subtly affected by direct mechanical compression and by tissue ischemia.<sup>9,10</sup> Transdural rupture of ICCA aneurysm is very rare, and can cause SDH,<sup>1,2,4,14</sup> ICH,<sup>5</sup> and SAH.<sup>8</sup> All these aneurysms were either large or giant, as in our patient. The outer layer of the lateral wall of the cavernous sinus consists of a meningeal dural layer which is tightly attached to the endosteal dural layer at the upper edge of the maxillary nerve. Each division of the trigeminal nerve is fixed at the superior orbital fissure, foramen rotundum, and foramen ovale. If the ICCA aneurysm is large, the lateral wall of the cavernous sinus is stretched and becomes thinner. Further enlargement of the aneurysm may tear the already weakened portion of the wall. Therefore, an ICCA aneurysm could rupture transdurally, if the aneurysm is large enough to stretch the cavernous sinus wall.

Previous cases suggest that an ICCA aneurysm exceeding 20 mm in size can rupture transdurally.<sup>1,2,4,5,8,14</sup> Giant ICCA aneurysms rarely rupture into the cavernous sinus, possibly because the cavernous sinus space is obliterated by the aneurysm. In our case, angiography identified a carotid-cavernous fistula, suggesting that the sudden drop in the aneurysm wall pressure after rupture allowed some space to reappear in the cavernous sinus, resulting in the formation of the carotid-cavernous fistula. Three of the four patients with SDH and one with SAH presented with ophthalmoplegia associated with pain or numbness in the first branch area of the trigeminal nerve.<sup>2,4,8,14</sup> These symptoms progressed rapidly during the 1 to 5 days before rupture. This time lag may reflect the difference between the period of rapid aneurysm growth and the period of aneurysm rupture followed by dural tearing. Our patient had suffered total ophthalmoplegia with aneurysm enlargement 2 months before rupture. Since MR imaging showed aneurysm enlargement but no intracranial hemorrhage, this may have been a good time for surgical intervention. She became drowsy about 5 days before rupture, when MR imaging showed further enlargement of the aneurysm causing edema in the adjacent temporal lobe, but without any signs of SDH

or ICH. We speculate that the growing aneurysm had protruded into the subdural space at this time. Immediate surgical treatment might have been indicated.

We conclude that large or giant ICCA aneurysms associated with rapidly progressive symptoms need some form of emergent surgical management such as ligation or trapping to prevent potentially fatal rupture into the intradural space.

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