

# **Postoperative Rupture of an Untreated Aneurysm on the 3rd Day After Subarachnoid Hemorrhage Surgery**

## **—Case Report—**

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### **Abstract**

A 58-year-old male presented with severe consciousness disturbance and left hemiparesis. Computed tomography (CT) revealed subarachnoid hemorrhage (SAH) and acute subdural hematoma caused by a ruptured right middle cerebral artery aneurysm. The aneurysm was clipped and the hematoma was evacuated. The patient had almost recovered without new neurological deficits on the next day. Arterial systolic blood pressure was postoperatively controlled within 120 to 150 mmHg. Continuous ventricular and cisternal drainage from the level 10 cm above the external auditory meatus was performed to drain bloody cerebrospinal fluid and prevent vasospasm. Three days after surgery, the patient suddenly lapsed into a coma. CT demonstrated diffuse SAH and bilateral intraventricular hemorrhage caused by rupture of an anterior communicating artery aneurysm. Neck clipping was performed immediately. Unfortunately, the patient died of primary damage due to SAH 3 days after the second surgery. In this case, cisternal drainage was probably important in the aneurysm rupture because of decreased intracranial pressure and change in the perianeurysm environment. Postoperative management of patients with residual untreated aneurysms must consider the possibility that cisternal drainage may result in higher transmural pressure, leading to rupture of the untreated aneurysms.

Key words: multiple aneurysms, unruptured aneurysm, ruptured aneurysm, subarachnoid hemorrhage, cisternal drainage

### **Introduction**

Multiple aneurysms are found in approximately 18–34% of intracranial aneurysm patients.<sup>3,7,8)</sup> All unruptured aneurysms may be impossible to treat during initial surgery in patients with subarachnoid hemorrhage (SAH) because the unruptured aneurysms may be contralateral to the operated side or distant from the ruptured aneurysm, or the patient may present with severe symptoms prior to surgery or be elderly.<sup>1)</sup> The annual incidence of hemorrhage from previously unruptured aneurysms ranges from 0.5% to 2.3% in Japan.<sup>5,12,15)</sup> Patients with unruptured aneurysms who have a prior history of SAH and multiple aneurysms are at higher risk of rupture,<sup>2,15)</sup> but such untreated aneurysms rarely cause clinical problems in the postoperative period.

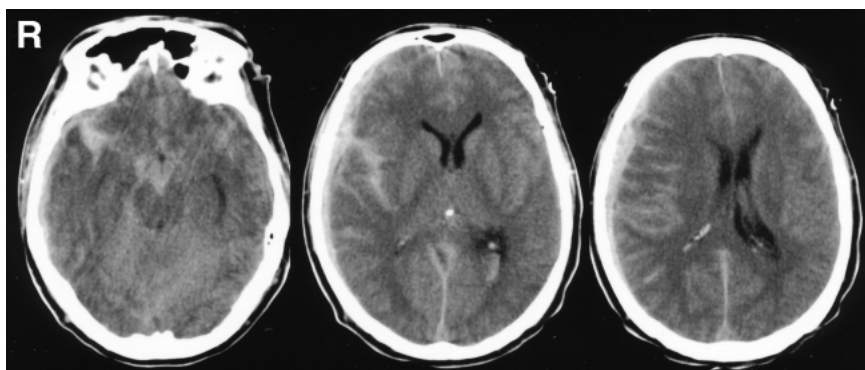
We treated a patient for SAH but an undetected

anterior communicating artery (ACoA) aneurysm ruptured 3 days after surgery, presumably caused by the postoperative management.

### **Case Report**

A 58-year-old man, in good health except for being a heavy smoker, lost consciousness at work for a few minutes and was transferred to our hospital after SAH was identified at a local hospital. On admission, he was in a deep coma with left hemiparesis. The right pupil was dilated more than the left. His eyes reacted sluggishly to light.

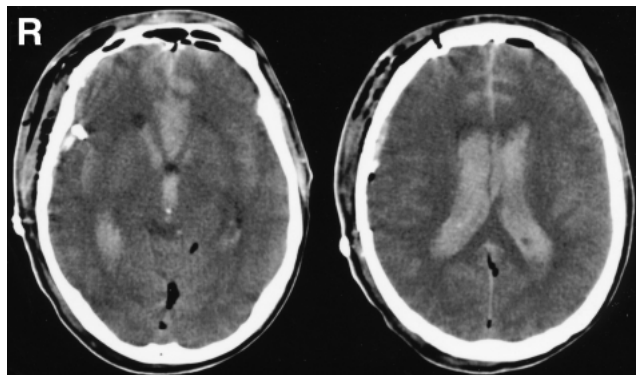
Computed tomography (CT) showed diffuse SAH predominantly in the right sylvian cistern and subdural hematoma (SDH) with mass effect that had caused midline shift (Fig. 1). Digital subtraction angiography (DSA) revealed a right middle cerebral artery (MCA) aneurysm (Fig. 2), which was responsible for both SAH and SDH. DSA was concluded after signs of cerebral herniation appeared, and the patient was rushed to emergency surgery. He



**Fig. 1** Computed tomography scans on admission showing diffuse subarachnoid hemorrhage predominantly in the right sylvian fissure and acute subdural hematoma with midline shift.



**Fig. 2** Right carotid angiogram on admission, left oblique view, showing a middle cerebral artery aneurysm (arrow).



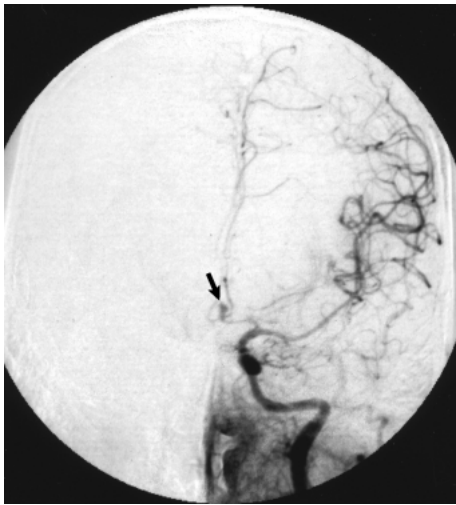
**Fig. 3** Computed tomography scans 3 days after surgery showing thick subarachnoid hemorrhage in the interhemispheric fissure and remarkably packed intraventricular hemorrhage.

underwent a right fronto-temporo-parietal large craniotomy and evacuation of the SDH, followed by clipping of the aneurysm through the right pterional approach. Drainage of cerebrospinal fluid (CSF) via a ventricular catheter during surgery relieved the pressure on the brain. Neck clipping of the aneurysm was achieved without complications. We confirmed that the MCA aneurysm had ruptured. A cisternal catheter was placed in the prepontine cistern to drain the bloody CSF after surgery.

The patient recovered consciousness, and the left hemiparesis disappeared on the 1st day after surgery (day 1). The bloody CSF drained spontaneously from drains placed 10 cm above the external auditory canal. Additionally, the patient was treated with intravenous administration of low molecular weight dextran to maintain colloidal pressure and fasudil hydrochloride (90 mg/day). We did not administer agents to decrease intracranial pressure (ICP). The ventricular catheter was removed on day 1, but the

cisternal drainage was continued. The volumes of CSF collected were: day 0, 50 ml from the ventricular side; day 1, 170 ml from the ventricular and 200 ml from the cisternal side; day 2, 210 ml from the cisternal side; day 3, 70 ml from the cisternal side until the subsequent emergency. Systolic and diastolic blood pressure averaged 150/80 mmHg.

The patient's clinical course was uneventful until day 3 when he suddenly lapsed into a deep coma. CT revealed thick diffuse SAH, especially in the interhemispheric fissure, and hematoma castings in the bilateral lateral ventricles (Fig. 3). The SAH pattern did not match the initial imaging of the MCA aneurysm. Left carotid angiography detected an ACoA aneurysm for the first time (Fig. 4). The ACoA aneurysm was clipped through the same right pterional approach. However, postoperative CT showed that a low density area had extended across the whole brain parenchyma. The patient died 3 days after the second operation (day 6).



**Fig. 4** Left carotid angiogram 3 days after surgery, anteroposterior view, showing an anterior communicating artery aneurysm (arrow) projecting posteriorly.

## Discussion

The risk factors for aneurysmal SAH related to hypertension, smoking, and drinking are relatively long-term factors which are also associated with the formation of aneurysms.<sup>2)</sup> Physical examinations and stress are immediate or short-term risk factors.<sup>10)</sup> These risk factors were mainly identified statistically, but essentially suggest the importance of increased blood pressure in aneurysm rupture. Circadian variations in aneurysm ruptures actually show a close correlation with the circadian fluctuations in the plasma concentrations of noradrenaline and blood pressure.<sup>10,13)</sup> Physical activity and Valsalva's maneuver can also easily affect blood pressure.<sup>10)</sup>

Morphological factors may also be significant in aneurysm rupture, such as aneurysm size, bleb formation, wall thickness, and large dome/neck ratio.<sup>2,9,14)</sup> Aneurysm rupture, especially in aneurysms with morphological weakness, is thought to be promoted by increased intraaneurysm pressure across the aneurysm wall due to an increase in blood pressure or a decrease in ICP. Intraaneurysm pressure sometimes resembles systemic blood pressure (SBP).<sup>10)</sup> These findings suggest that a rise in arterial pressure may result in aneurysm rupture due to increased hemodynamic stress. In our institution, SBP is observed not to be influenced until about 180 mmHg by reduced cerebral blood flow because of vasospasm, even though an unruptured aneurysm remains.

Hypertensive hypervolemic therapy was given to 40 patients with a total of 73 untreated aneurysms during a mean of 7.25 days who had suffered vasospasm after SAH.<sup>1)</sup> Despite aggressive induction of hypertension resulting in moderate SBP (180–200 mmHg) in 12 patients and severe SBP (> 200 mmHg) in nine patients, no bleeding occurred from the unruptured aneurysms, although only 6.8% were over 10 mm. In another study, 31 patients with 34 unruptured aneurysms, of which 29.4% were over 10 mm, received similar successful hypertensive treatment.<sup>11)</sup> Therefore, short-term postoperative hypertension is safe, even at the risk of adding hemodynamic stress to the aneurysm. However, one patient suffered fatal rupture of a giant carotid aneurysm while undergoing hypertensive therapy, although the details were not mentioned.<sup>4)</sup> In our patient, SBP was maintained within 120–150 mmHg. Therefore, we considered other factors except for hypertension as the cause of the aneurysm rupture. But, we did not continuously monitor SBP, so we do not know whether sudden SBP changes actually occurred.

Intravenous administration of mannitol or glycerol, ventricular drainage to control ICP, and cisternal or lumbar drainage for evacuation of bloody CSF to prevent vasospasm all reduce the ICP and may lead to relatively increased transmural pressure of the aneurysm. Ventricular drainage is closely associated with an increased incidence of aneurysm rebleeding, particularly in the presence of hydrocephalus.<sup>6)</sup> This study suggests that decreases in ICP caused by excessive drainage are associated with proportional increases in the risk of aneurysm rupture. Over 72% of ruptured aneurysms were in contact with perianeurysm structures such as the brain, dura, bone, and cranial nerves.<sup>9)</sup> These aneurysm environment, particularly unbalanced contact, is an additional important determinant in the risk of aneurysm ruptures.

At our hospital, surgical procedures for SAH include the placement of a ventricular drainage tube to relieve pressure on the brain, even if the patient does not present with acute hydrocephalus. A cisternal drainage tube is also placed in the carotid or prepontine cistern to drain bloody CSF after surgery. Both drainage tubes are opened 10 cm above the external auditory canal and allowed to drain spontaneously. The ventricular drainage catheter is removed as soon as it is no longer necessary for ICP control, usually the day after the operation, and cisternal drainage is continued for about 7 days, based on reduction of SAH on CT or improvement in the CSF from bloody to xanthochromic.

In the present case, an excessive amount of CSF,

about 370 ml, was drained only on day 1 from both ventricular and cisternal catheters; subsequent drainage was about 10 ml per hour. Whether the ICP of 10 cmH<sub>2</sub>O and the height of the cisternal drainage level contributed to the aneurysm rupture is not definitively known. Acute physical events such as postural changes and Valsalva's maneuvers are associated with reductions of ICP.<sup>10)</sup> A significant fall in ICP due to excessive drainage of CSF can occur particularly during drainage setting. Since rupture of such an aneurysm is unusual, we speculate that the rupture of the aneurysm was associated with a decrease in ICP and/or a change in the perianeurysm environment induced by CSF drainage. If this hypothesis is accurate, prophylactic management of vasospasm with evacuation of CSF may directly affect the transmural pressure in the aneurysm.

We did not recognize the unruptured aneurysm in our patient before surgery because angiography could not be completed. The possibility of unruptured aneurysms must be investigated as soon as possible after surgery. If preoperative magnetic resonance angiography or three-dimensional CT angiography had been performed, the ACoA aneurysm could have been clipped at the same procedure. Unruptured aneurysm at risk is difficult to predict. Therefore, postoperative management should consider the remaining aneurysms. Based on our experience, medical personnel should remain alert for sudden changes in the rate of CSF drainage and avoid the combination of hypertension and extensive drainage, especially in the presence of large or irregular-shaped aneurysms at high risk of rupture.

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