Significance of Acute Cerebral Swelling in Patients with Sylvian Hematoma due to Ruptured Middle Cerebral Artery Aneurysm, and its Management

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Abstract

A retrospective study of 75 patients treated surgically for ruptured middle cerebral artery (MCA) aneurysm within 48 hours evaluated clinical grade at admission, secondary development and management of cerebral swelling associated with space-occupying hematoma, cerebral infarction caused by vasospasm, development of hydrocephalus, and clinical outcome. Clinical grade at admission was significantly better in patients without than in those with hematoma (p < 0.01). Twenty-seven patients with sylvian hematoma caused by ruptured MCA aneurysm often developed ipsilateral cerebral swelling in the early period after subarachnoid hemorrhage. Seventeen of these patients developed serious cerebral swelling and received barbiturate therapy. Nine of these 17 patients had good outcome, but six patients died of cerebral swelling. The incidence of hydrocephalus was significantly higher in patients with than in those without hematoma (p < 0.01). The incidence of infarction was more pronounced in patients with sylvian hematoma. Clinical outcome was significantly better in patients without than in those with sylvian hematoma (p < 0.01). Development of cerebral swelling in patients with sylvian hematoma due to ruptured MCA aneurysm has a significant effect on outcome, and improvements in management are required.

Key words: middle cerebral artery aneurysm, sylvian hematoma, cerebral swelling, barbiturate therapy

Introduction

Intracerebral hemorrhage is a less frequent manifestation of aneurysmal rupture than subarachnoid hemorrhage (SAH), and indicates a poor prognosis after aneurysmal rupture, as the mortality ranges between 33% and 58%. Hematoma with a mass effect causes the development of vasogenic edema, which also negatively influences the clinical outcome. Vasogenic edema, subsequent to blood-brain barrier (BBB) disruption, is the most common form of cerebral edema, and is associated with many clinical problems including brain tumors, hemorrhage, and infarction. The secondary development of cerebral edema (swelling) in patients with ruptured aneurysm is apparently a clinical problem, but the significance has not been assessed.

We describe our experience in the management of cerebral swelling in patients with sylvian hematoma caused by ruptured middle cerebral artery (MCA) aneurysm, and evaluate the development of cerebral infarction caused by vasospasm, hydrocephalus, and the clinical outcome.

Subjects and Methods

This study included 75 of the 92 patients with ruptured MCA aneurysm admitted to the Department of Neurosurgery of Prefectural Gifu Hospital between January 1, 1986 and March 31, 1997. All patients were treated by aneurysmal clipping within 48 hours after the onset of SAH. Seventeen patients were excluded, eight who were not treated surgically because of rapid deterioration caused by rebleeding or moribund condition, and nine who were treat-
ed by delayed surgery. These were 21 males and 54 females, aged 27–84 years (mean 56 years). Clinical status was graded at admission according to the Hunt and Kosnik scale.11

All patients were treated using a unilateral pterional approach. After obliteration of the aneurysm, Liliequist’s membrane was opened, and a cisternal drain15 was inserted between the optic nerve and the internal carotid artery into the interpeduncular cistern. The closed drain was connected to the drainage circuit, which was located about 5 to 10 cm higher than the forehead. Patients with sylvian hematoma (significantly thick sylvian SAH, more than 5 ml) with or without extension into the brain were classified as the SH group, those with intraparenchymal hematoma other than sylvian hematoma were classified as the ICH group, and those without hematoma were classified as the NH group.

Acute cerebral swelling in the SH group was divided into three categories according to the computed tomography (CT) findings: mild cerebral swelling indicating focal cerebral edema surrounding the sylvian hematoma without obliteration of the basal cistern, moderate cerebral swelling indicating more advanced cerebral edema with significant midline shift and partially obliteration of the basal cistern, and severe cerebral swelling indicating critically advanced cerebral edema with complete obliteration of the basal cistern. Barbiturate therapy was administered to the patients who developed serious cerebral swelling. In these patients, controlled ventilation was maintained and arterial and central venous pressure and electroencephalography were recorded. The circuit for continuous cisternal drainage15 was located about 5 cm higher than the forehead. Barbiturate therapy was begun with 200 to 250 mg intravenous infusion of thiopental, followed by continuous intravenous infusion of pentobarbital at 100 to 200 mg per hour. Mechanical hyperventilation was combined with administration of osmotic diuretics, phenytoin, and/or steroids. Repeat CT was used to monitor the intracranial condition. Transcranial Doppler ultrasound was utilized to monitor the blood flow velocity from the arteries at the base of the brain. Single photon emission computed tomography was also used as an option.

Outcome was assessed at discharge according to the Glasgow Outcome Scale (GOS)12: good (GOS 1 or 2), poor (GOS 3 or 4), and death. Clinical grade on admission, development of acute cerebral swelling, cerebral infarction caused by vasospasm, hydrocephalus after SAH, and outcome were compared between the three groups. Statistical analysis of the data used the chi-square test or Fisher exact test, and significance was assessed as a p value of less than 0.05.

**Results**

**I. Clinical grade on admission**

Assessment of the clinical grade of Hunt and Kosnik13 at admission (Table 1) found that the NH group had 43 patients in good clinical condition (grades 1–3) and three patients in poor clinical condition (grade 4). The SH group contained 14 patients in good clinical condition and 13 patients in poor clinical condition. The two patients in the ICH group were in poor condition. The difference in clinical grade between the patients with (the SH and ICH groups) and without (the NH group) hematoma was significant (p < 0.01).

**II. Effect of barbiturate therapy on acute cerebral swelling**

The development of acute cerebral swelling in patients who received intravenous barbiturates and clinical outcome are summarized in Table 2. Patients with sylvian hematoma often developed ipsilateral cerebral swelling seen on CT scans on days 1 to 7 (average day 4) after SAH (day 0). Moderate or severe cerebral swelling was seen on days 1 to 6 (average day 3). Greater volume of sylvian hematoma was associated with increased development and severity of cerebral swelling (p < 0.05). Only two of eight patients with sylvian hematoma of less than 10 ml developed moderate or severe cerebral swelling. In contrast, 15 of 19 patients with sylvian hematoma of more than 10 ml developed moderate or severe cerebral swelling.

All five patients with moderate and 12 patients with severe cerebral swelling received barbiturate therapy. Nine of these 17 patients achieved a good outcome. All five patients with moderate cerebral swelling had a good outcome, but only four of 12

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**Table 1 Clinical grade at admission in patients with sylvian or intraparenchymal hematoma, and no hematoma**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of cases</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>NH</td>
<td>46</td>
<td>1</td>
</tr>
<tr>
<td>ICH</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>SH</td>
<td>27</td>
<td>0</td>
</tr>
</tbody>
</table>

The difference between the patients with (the sylvian hematoma [SH] and intracerebral hematoma [ICH] groups) and without (the no hematoma [NH] group) hematoma is significant (p<0.01).
patients with severe cerebral swelling had a good outcome and one remained severely disabled. Barbiturate therapy was stopped 6 to 17 days (mean 13 days) after the initiation of the therapy in the survivors. Overall, six patients died of the development of cerebral swelling, three died of extensive cerebral infarction caused by vasospasm, and one recovered consciousness but died of acute hepatitis.

III. Effect of hematoma evacuation on the development of cerebral swelling

Fourteen of 17 patients who underwent aggressive removal of sylvian hematoma developed serious cerebral edema. Seven of 10 patients who did not undergo removal of sylvian hematoma did not develop significant cerebral edema, but six of these seven had small sylvian hematoma of less than 10 ml. These seven patients with small sylvian hematoma were treated surgically to exclude the ruptured aneurysm, and evacuation of the hematoma was not the operative purpose.

IV. Cerebral infarction caused by vasospasm

Cortical or subcortical infarction due to vasospasm was seen on CT scans in eight of 46 patients in the NH group, and eight of 24 patients in the SH group, excluding patients who died in the early period after surgery. The incidence of cerebral infarction was greater in the SH group, but the difference was not significant (p = 0.23). Neither patient in the ICH group developed infarction.

V. Hydrocephalus after SAH

Normal pressure hydrocephalus in patients who survived more than 1 month after SAH occurred in seven of 45 patients in the NH group, and in 10 of 18 patients in the SH group. Both patients in the ICH group developed hydrocephalus. The difference in the development of hydrocephalus between the patients with (the SH and ICH groups) and without (the NH group) hematoma was significant (p < 0.01).

VI. Overall outcome

Forty-one of 46 patients in the NH group and 14 of 27 patients in the SH group had good outcomes. Ten patients in the SH group died. The difference in outcome between the NH and SH groups was significant (p < 0.01). Three patients died of complications such as hepatic failure and pneumonia. Both patients in the ICH group developed cerebral swelling and responded to barbiturate therapy, but one patient died of acute hepatitis (Table 3).

Illustrative Case

A 64-year-old male suffered sudden onset of severe headache followed by loss of consciousness, and was transferred to our clinic. On admission, he was somnolent with Hunt and Kosnik grade 3. CT revealed SAH associated with left sylvian hematoma (15 ml) (Fig. 1A). Angiography revealed a ruptured aneurysm of the left MCA. Early surgery was performed and postoperatively he became alert. Postoperative CT showed reduced brain bulk and no cerebral swelling (Fig. 1B). However, he suffered insidious onset of unconsciousness with anisocoria and right hemiplegia showing decerebration on day 3. CT revealed critical development of left cerebral swelling (Fig. 1C). Barbiturates were administered...
for 9 days. On day 35, a ventriculoperitoneal shunt was placed to treat hydrocephalus. He gradually became alert. CT showed resolution without sequela (Fig. 1D). He was discharged with no neurological deficit 2 months after admission.

**Discussion**

The Fisher classification of subarachnoid blood visualized by CT classes SAH with thick sylvian (insular) accumulation or sylvian hematoma into group 3, and SAH with intraparenchymal or intraventricular accumulation as group 4. The incidence of a hematoma after aneurysmal rupture is 4–34%, and ruptured MCA aneurysms cause hematoma more frequently than aneurysms in other locations. Hematoma from a ruptured MCA aneurysm may cause progressive intracranial hypertension or cause devastating mass effect of SAH, but the result of emergent surgery in deeply comatose patients is quite poor. This effect of space-occupying hematoma following ruptured aneurysm is well recognized, but the serious effect of secondary cerebral swelling has not been fully investigated.

Our experience showed that patients with sylvian hematoma caused by ruptured MCA aneurysm often develop ipsilateral cerebral swelling. Serious cerebral swelling had occurred by day 3 on average after SAH, and patients with such cerebral swelling had a worse outcome. Cerebral edema can be generally classified into vasogenic edema subsequent to BBB disruption, and cytotoxic edema resulting from interference with cellular metabolism. Vasogenic edema is the most common form, and is characterized by increased permeability of cerebral capillary endothelial cells after BBB disruption. It is associated with clinical problems causing neuroimaging changes, including brain tumors, abscess, hemorrhage, infarction, and contusion. In the present study, CT detected moderate or severe cerebral swelling in the early period after SAH. Sudden formation of sylvian hematoma may damage the surrounding brain, and BBB disruption followed by increased permeability of endothelial cells and the tight junction may occur, resulting in formation of vasogenic cerebral edema. In addition, surgical manipulation such as mechanical traumatization, electrocautery, desiccation, and retractor compression might affect the BBB integrity, and result in the evolution of postoperative vasogenic edema.

Since barbiturate therapy has been considered one of useful modalities in the management of critical intracranial pressure (ICP) increases, barbiturate therapy is being used with increasing frequency in the management of a widening spectrum of neurological disorders. The effect of barbiturates on ICP is related to direct vasoconstriction of the cerebral vasculature or reduction of cellular metabolism and consequent decrease in cerebral blood flow (CBF), resulting in a decrease in intracranial volume and ICP. Reduced cerebral edema formation may occur because barbiturates commonly lower mean systolic blood pressure with a consequent reduction in the hydraulic pressure across the BBB. Stabilization of the lysosomal membrane or decreased CBF following barbiturate administration may also reduce cerebral edema formation. Therefore, barbiturate therapy is considered to be a useful modality in the management of critical ICP increases following the occurrence of cerebral edema. In the present study, all patients who developed moderate cerebral swelling had good outcome,
although only five of 12 patients with severe cerebral swelling survived after barbiturate therapy. Therefore, more aggressive management might be necessary to some cases. Decompressive surgery was performed in a series of 25 patients with intracerebral hemorrhage due to ruptured aneurysm. Eighteen patients had ruptured MCA aneurysm. Eight of the 25 patients underwent emergent internal decompression (lobectomy) and 15 patients underwent emergent decompressive craniotomy. Twelve patients survived, but 13 patients died. Further studies may be necessary for the evaluation of such surgical management in cases with sylvian hematoma.

The development of cerebral swelling in patients with sylvian hematoma due to ruptured MCA aneurysm has a significant effect on the outcome. Barbiturate therapy is useful for the management of patients with serious cerebral swelling, but an improved protocol is required. Other methods of management should be investigated for these patients.

References


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Cerebral Swelling Associated with Sylvian Hematoma

Commentary

This study confirms the finding of Fisher and others that thick subarachnoid hemorrhage is associated with a poorer outcome than more minor hemorrhages. In this retrospective study of 75 patients treated over an 11 year, 2 month period for ruptured middle cerebral artery aneurysms, those 27 patients with sylvian hematomas had poorer clinical grades, higher incidence of brain swelling, higher incidence of hydrocephalus, and poorer outcomes than the 46 patients without sylvian hematomas. The sylvian hematoma group also had a higher incidence of cerebral infarction from vasospasm but the difference was not statistically significant.

The authors propose that barbiturate therapy improves the outcome for patients with sylvian fissure hematomas that develop cerebral edema. A protocol of barbiturate administration was provided for all five patients with moderate and all twelve patients with severe cerebral edema. All five with moderate edema had a good outcome, whereas only four of twelve with severe edema had a good outcome. The precise role of barbiturates in the management of cerebral edema from sylvian subarachnoid hemorrhage cannot be determined from such a retrospective study but its use seems appropriate from an intuitive standpoint.

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Niikawa et al. present a ten year experience of acute cerebral swelling in 75 patients with sylvian hematoma due to ruptured middle cerebral artery aneurysms. The Fisher classification in its grade 3 put any cisternal clot \( \geq 3 \times 5 \) mm and made a relation with the development of vasospasm (ref. 4 of this article). In this series the most important factor for the development of cerebral swelling was the presence of blood in the sylvian cistern and the volume of the hematoma was accurately measured. The incidence of cerebral infarction was greater in the sylvian hematoma group but was not significant. The outcome was dependent on the cerebral swelling severity. The barbiturate therapy was effective in cases without severe cerebral swelling, in this particular group the outcome was more often poor. The therapy proposed here is well documented and is widely used. This kind of complication after subarachnoid hemorrhage is frequent in the middle cerebral artery aneurysms, but the dynamic process after the rupture of an aneurysm put together several mechanisms in the first hours and days, with the secondary development of vasospasm and cerebral swelling: the differentiation between them is not always easy, but it is important because of its therapeutical implications. This paper confirms that the amount of the sylvian hematoma is an important clinical clue to indicate an aggressive therapy in the patients with ruptured middle cerebral artery aneurysms, but in its severe forms the current therapies are not effective enough and need to be improved.

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Based on broad experience in managing ruptured MCA aneurysms, the authors demonstrated that patients with sylvian hematoma often develop severe ipsilateral cerebral swelling in the early period and exhibit a high incidence of cerebral infarction due to vasospasm. They concluded that an improved management protocol is required due to the limited efficacy of barbiturate therapy.

The authors showed that 17 patients out of 27 with sylvian hematoma developed acute brain swelling and hence underwent barbiturate therapy. The incidence seems slightly higher than has been generally recognized. The authors attributed the swelling to vasogenic edema caused by the developing hematoma and surgical manipulations. I consider that the sylvian veins compressed by the packed hematoma may have also contributed to this swelling.

While the discussion of this paper covers a wide range of issues, limited information concerning the efficacy of removing sylvian hematoma to prevent swelling was included. Although most patients (14/17) undergoing aggressive removal were reported to develop serious edema, I personally believe that removing the hematoma in the early period or at least adequate reduction in its mass effect is essential. Whether surgical removal can be traumatic and consequently induce additional swelling or not depends on multiple factors including the modes of removal (e.g. microsurgical removal with some irrigation system, combined use of intra- and/or postoperative fibrinolysis), surgeon’s skills and the extent of removal. Future analysis on the results of coil embolization followed by fibrinolytic therapy would indirectly reveal the causal relationship between surgical manipulation and the swelling. In my opinion, every effort should be made to remove sylvian hematoma of significant size effectively.

About half of the patients with sylvian hematoma (13/27) were shown to be in clinical grade 4 on admission. Some may have been in bad condition irrespec-
tive of the swelling. The presence of these patients, who exhibited the initial SAH attack, may have overestimated the significance of the swelling.

The authors should answer these questions by further accumulating and analyzing surgical cases.

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Acute cerebral swelling is occasionally experienced when massive sylvian hematoma forms after SAH from ruptured middle cerebral artery aneurysm. The authors investigated this phenomenon and analyzed the clinical courses of their 75 patients retrospectively. Patients with sylvian hematoma were in higher grade of Hunt and Kosnik at admission, developed ipsilateral cerebral swelling more often, and developed subsequent normal pressure hydrocephalus significantly more often than those without hematoma. These factors resulted in significantly worse overall outcome of patients with sylvian hematoma. The authors introduced barbiturate therapy for acute cerebral swelling and good effect was obtained to some extent of the outcome of patients with mild or moderate cerebral swelling. However, in spite of their efforts, the clinical outcome of patients with severe cerebral swelling still remained poor. As the authors mentioned in this paper, further improvements in the management of such cases are necessary.

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