Poor-Grade Ruptured Middle Cerebral Artery Aneurysm With Intracerebral Hematoma: Bleeding Characteristics and Management

Ken KAZUMATA, Hiroyasu KAMIYAMA*, Yuka YOKOYAMA, Katsuyuki ASAOKA, Shunsuke TERASAKA**, Kouji ITAMOTO, and Toshiya OSANAI

Department of Neurosurgery, Teine Keijinkai Hospital, Sapporo, Hokkaido; *Department of Neurosurgery, Asahikawa Red Cross Hospital, Asahikawa, Hokkaido; **Department of Neurosurgery, Hokkaido University School of Medicine, Sapporo, Hokkaido

Abstract

Poor-grade ruptured middle cerebral artery aneurysm is frequently associated with intraparenchymal hemorrhage, which is associated with high morbidity rates. We analyzed the clinical presentations and surgical strategies of 23 cases of ruptured middle cerebral artery aneurysm. Hematomas were divided into three types: temporal hematoma (7 patients), sylvian hematoma (10 patients), and frontal hematoma (6 patients). In 13 of 23 patients, preoperative brainstem symptoms suggested impending uncal herniation. Surgical procedures included external decompression in 11 patients, simple lateral temporal lobectomy in 5, and selective uncectomy in 9. Three patients died. Favorable outcome defined as upper half of severely disabled or better in the extended Glasgow Outcome Scale was achieved in 13 patients. Patients with frontal hematomas presented with both uncal herniation and brainstem signs preoperatively, but this subgroup showed unexpectedly good recovery. Patients with sylvian hematomas had relatively poor outcomes. The present series suggests that aggressive decompression and evacuation of hematoma in the acute stage may prevent significant postoperative brain swelling, and will not compromise the treatment of vasospasm.

Key words: subarachnoid hemorrhage, cerebral aneurysm, middle cerebral artery, sylvian hematoma, microsurgery

Introduction

Patients with poor-grade subarachnoid hemorrhage (SAH) from ruptured middle cerebral artery (MCA) aneurysm present with a diverse range of pathologies, such as concomitant intraparenchymal hematoma, asphyxia caused by spontaneous respiratory arrest, and increased intracranial pressure (ICP).1,3,5,6,17,19,28,34 Patients do not always achieve outcomes consistent with their initial neurological status, and 50–60% of patients affected by rupture of an intracranial aneurysm can become functional survivors.3,9–12,14,18,20,22,25,26 However, insufficient surgical intervention may result in loss of potentially salvageable function or life.1,3,9,12,15,20,27 Adequate treatment often requires hematoma evacuation and decompressive maneuvers in addition to clipping procedures.4,7,16,21,23,24,26

However, there are few detailed descriptions of procedures that are appropriate for the management of poor-grade SAH with ruptured MCA aneurysm. Previous studies have shown that age and initial Glasgow Coma Scale (GCS) can predict outcomes, but none have validated the exclusion criteria for aggressive treatment in patients with high-grade SAH.9,11,12,14,18–20,22,25,26 The present study describes the anatomical and clinical findings during surgical treatment of poor-grade ruptured MCA aneurysms, and tailored surgical strategies based on the distribution of hematoma. The three study objectives are to identify patients with the worst neurological status prior to treatment who were able to achieve useful daily life; to follow functional outcomes and living status in patients who underwent aggressive treatment; and to describe the surgical procedures and how these were employed in our clinical practice.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Side</th>
<th>Aneurysm size</th>
<th>Pupil abnormality</th>
<th>Decerebrate posture</th>
<th>Bleeding pattern</th>
<th>Preoperative decompression</th>
<th>DIND</th>
<th>Hydrocephalus</th>
<th>Decompression</th>
<th>Extended GOS</th>
<th>GOS mRS score</th>
<th>Hematoma and MCA Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54</td>
<td>F</td>
<td>rt</td>
<td>sylvian</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>GR</td>
<td>0</td>
<td>delayed ischemic neurological deficit, ETD, external decompression, LCSG, Glasgow Coma Scale, L.R. light reflex, LTL lateral temporal lobectomy, mRS modified Rankin scale.</td>
</tr>
<tr>
<td>2</td>
<td>78</td>
<td>F</td>
<td>lt</td>
<td>S</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>PVS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>3</td>
<td>78</td>
<td>F</td>
<td>rt</td>
<td>temporal</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>F</td>
<td>rt</td>
<td>temporal</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>5</td>
<td>54</td>
<td>F</td>
<td>lt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>6</td>
<td>80</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>7</td>
<td>66</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>8</td>
<td>99</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>9</td>
<td>53</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>10</td>
<td>77</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>11</td>
<td>81</td>
<td>F</td>
<td>rt</td>
<td>temporal</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>13</td>
<td>56</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>14</td>
<td>36</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>15</td>
<td>36</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>16</td>
<td>57</td>
<td>F</td>
<td>lt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>17</td>
<td>79</td>
<td>F</td>
<td>lt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>18</td>
<td>55</td>
<td>M</td>
<td>lt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>19</td>
<td>75</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>20</td>
<td>66</td>
<td>F</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>21</td>
<td>46</td>
<td>M</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>22</td>
<td>66</td>
<td>M</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
<tr>
<td>23</td>
<td>75</td>
<td>M</td>
<td>rt</td>
<td>S</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>RS</td>
<td>5</td>
<td>Hematoma and MCA Aneurysm</td>
</tr>
</tbody>
</table>

*S*: >10 mm; M: 10–25 mm; L: >25 mm. **Extended GOS extends the original 5 Glasgow Outcome Scale categories to 8 as follows: dead (D), vegetative state (VS), lower severe disability (lower SD), upper severe disability (upper SD), lower moderate disability (lower MD), upper moderate disability (upper MD), lower good recovery (lower GR), and upper good recovery (upper GR). DIND: delayed ischemic neurological deficit, ETD: external decompression, LCSG: Glasgow Coma Scale, L.R. light reflex, LTL lateral temporal lobectomy, mRS: modified Rankin scale.
Patients and Methods

The clinical, operative, and radiographic data for all cerebral aneurysms treated surgically by the first author were prospectively tabulated between April 2001 and March 2008. The study cohort consisted of 6 men and 17 women aged 36–86 years (mean 64.3 years) at surgery with poor-grade (World Federation of Neurosurgical Societies [WFNS] grade IV and V) SAH originating from ruptured MCA aneurysm. The individual characteristics are shown in Table 1. The clinical signs used to assign WFNS grade were evaluated immediately before surgery. Outcome scores were derived from an extended Glasgow Outcome Scale (GOS) and the modified Rankin scale (mRS) assessed at 1 year posthemorrhage. Outcome analysis was performed by the prognosis-based outcome assessment employed in the International Surgical Trial in Intracerebral Haemorrhage (STICH).13) Prognostic scores were calculated using the equation:

\[(10^* \text{ admission GCS}) - \text{age (yrs)} - (0.64^* \text{ volume [ml]})\]

For this study cohort, the prognostic score was below 27.672, representing a poor prognosis group. Favorable outcomes included good recovery (GR), moderate disability (MD), and the upper half of severely disabled (SD).9,13) Categorical comparisons were made for age, severity of midline shift, volume of hematoma, presence of dense subarachnoid clot in the basal cistern, incidence of angiographical vasospasm, and side of the lesion in groups with favorable and unfavorable outcomes using two-tailed t or \(\chi^2\) tests.

In general, patients with poor-grade SAH present with either massive intraparenchymal hematoma or dense subarachnoid clot.5,6,19) Hematoma associated with ruptured MCA aneurysms has been classified into two patterns, temporal intraparenchymal hematoma and intra-sylvian hematoma.15,16,19,22,34) In contrast, this study classified hematoma into three categories: temporal hematoma; sylvian hematoma; and frontal hematoma, representing hematoma extending to the base of the frontal lobe. Temporal hematoma was located in the temporal lobe (Fig. 1A). No clear definition has been accepted for differentiating sylvian hematoma from moderate amounts of clot in the sylvian fissure.15,16,19,24,27,34) We used the term sylvian hematoma to describe subarachnoid hematoma located in the sylvian fissure associated with a GCS score \(< 13\). Using this definition, sylvian hematoma invariably acted as a space-occupying lesion (Fig. 1B). We attempted to classify another subgroup of frontal hematoma (Fig. 1C), based on the following characteristic radiological and clinical findings. The aneurysm dome projects upward, and is buried in the lateral orbito-frontal gyrus or the insular cortex. The aneurysm ruptured into the external capsule, the frontal lobe, and then into the frontal horn.5) The third ventricle is widely dilated if the hematoma ruptured into the ventricular system.

A large craniotomy followed by a large question mark-shaped skin incision is required. A much larger craniotomy is performed in patients with sylvian hematoma if evacuation of hematoma may not be an option, to gain slackness at the time of aneurysm clipping. If massive temporal hematoma has caused brain herniation, a moderate-sized craniotomy and modest aspiration of hematoma prior to the approach may save time. If brain swelling does not subside after evacuation of the hematoma, a larger craniectomy can be added after applying the aneurysm clips before closing the wound (by leaving out the bone flap).

A modest amount of temporal hematoma can be evacuated through the cortical incision made on the superior temporal gyrus before dissecting the sylvian fissure. After applying the aneurysm clips, temporal hematoma is usually easy to evacuate. Evacuation of sylvian hematoma starts from the medial temporal lobe (planum temporale) adjacent to the aneurysm after applying the aneurysm clips (Fig. 2). Subpial hematoma usually extends along the long axis of the transverse temporal gyrus, and evacuation should be continued within the parenchyma until the bottom of the hematoma is observed. The posterior margin of large sylvian hematoma is located immediately anterior to the posterior isthmus, so the postero-inferior limit of the hematoma extending beyond the circular sulcus can be accessed without further opening of the distal part of the sylvian fissure.28,30) The evacuation process proceeds rostral to the superior limiting sulcus. The intra-sylvian clot is evacuated after removing the subpial hematoma. Clot usually contains pial arteries and veins, which

![Fig. 1 Computed tomography scans illustrating the classification of intracerebral hematoma associated with ruptured middle cerebral artery aneurysm: temporal hematoma (A), sylvian hematoma (B), and frontal hematoma (C).](image-url)
Fig. 2 A: Photographs of the autopsied brain from a patient who died of ruptured middle cerebral artery aneurysm. Dotted line indicates the pia mater surrounding the sylvian fissure. Arrows indicate the transverse temporal gyrus (Heschl’s gyrus) coursing along the longitudinal axis deep into the sylvian fissure. Cross-section reveals hematoma (arrow) in the sylvian fissure extending beyond the pia mater surrounding the sylvian fissure. Parenchymal hemorrhage is also observed in the transverse temporal gyrus (Heschl’s gyrus), insular cortex, and external capsule. Hematoma with a round shape at the posterior edge in the temporal stem of the sylvian fissure on computed tomography is also highly likely to be located in the intraparenchymal space beyond the sylvian point. B–G: Diagram (B) and photographs (C–G) illustrating the surgical procedures. (1) The head of the patient was rotated to the ipsilateral side of the hematoma and extended in a vertex-down direction so that the sylvian fissure and insula were oriented vertically in the surgical field and the transverse temporal gyrus was parallel to the perspective of the surgeon. Evacuation was performed through the subpial hematoma located along the long axis of Heschl’s gyrus (arrow). The bottom of the hematoma indicates the location immediately anterior to the posterior isthmus and caudal to the sylvian point (C, D). (2) The head of the patient was rotated to the contralateral side to the hematoma so that the sylvian fissure and insula were oriented vertically in the surgical field and the transverse temporal gyrus was parallel to the perspective of the surgeon. Evacuation of subpial hematoma located above superior limiting sulcus was completed (E, F). (3) Removing the subpial clot first facilitated intrasylvian clot evacuation by creating sufficient working space, and showed which vessels were to be sacrificed (G).

Fig. 3 Representative Case 7 with sylvian hematoma. Upper row: Preoperative computed tomography scans demonstrating large sylvian hematoma on admission. Lower row: Postoperative computed tomography scans documenting gross total evacuation of hematoma carried out with large hemicraniectomy.
Fig. 4 Representative Case 21 with frontal hematoma. Upper row: Preoperative computed tomography scans demonstrating frontal hematoma extending to the external capsule (arrow) with ventricle rupture (arrowhead) on admission. Lower row: Postoperative computed tomography scans documenting gross total evacuation of hematoma carried out with large hemicraniectomy.

which proved to be a useful landmark. This anatomical landmark is defined by the internal carotid terminal and inferior choroidal point, as described in detail previously (Fig. 5).32)

Results

Fifteen patients were admitted with WFNS grade V and 8 patients were admitted with WFNS grade IV. Thirteen patients exhibited either signs of uncal herniation or compression of the brainstem. Clinical assessment on admission revealed pupil abnormality in 13 patients (unilateral enlargement in 8, bilateral enlargement in 2, and bilateral miosis and absent light reflex in 3) and decerebrate posture in 4. Aneurysm size assessed by digital subtraction angiography revealed small aneurysm (<10 mm) in 19 patients, moderate (10–25 mm) in 1, large (>25 mm) in 1, and dissecting aneurysm in 2. Location of the hematoma on computed tomography (CT) was temporal in 7 patients, sylvian in 10, and frontal in 6. Four of the 6 frontal hematomas extended to the external capsule, perforating the anterior horn and filling the third ventricle. Clinical presentation was characterized by bilateral miosis and absence of light reflex, as well as decerebrate posture.

All patients except 2 underwent emergency craniotomy and clipping within 3 hours after admission. Two patients with poor initial GCS score (GCS 4) and extensive frontal hematoma were observed for 48 hours but neurological status remained unchanged. Craniotomy and clipping were then performed. Decompression maneuvers such as external
decompression were employed in 11 patients, selective unectomy in 9, and lateral temporal lobectomy in 5 as shown in Table 2. Ventriculostomy was successful in 10 patients. Fenestration of the lamina terminalis was performed and a cisternal drainage catheter was placed in all patients. Gross total evacuation of hematoma was performed in all patients. No postoperative parenchymal rebleeding was observed. The 10 patients with sylvian hematoma also received external decompression in 4, selective unectomy in 4, and lateral temporal lobectomy in 2. No significant delayed brain swelling was observed in 9 of these 10 patients. One patient suffered delayed brain swelling which required left temporal lobectomy and external decompression on day 4.

Five patients showed signs of delayed ischemic neurological deficit, and 7 patients had hydrocephalus confirmed by CT. Prognosis-based outcome analysis determined favorable outcomes in 13 of the 23 patients. Eleven of the 23 patients had GR and MD according to the GOS. Five patients remained in the persistent vegetative state, and 3 patients died. Similarly, overall outcomes showed 4 patients with mRS score of 0, 1 patient with mRS score of 1, 1 patient with mRS score of 2, 2 patients with mRS score of 3, 5 patients with mRS score of 4, 7 patients with mRS score of 5, and 3 patients with mRS score of 6. Therefore, 6 of the 23 patients had mRS score 0 to 2.

One-year follow-up survey of 20 patients showed 6 patients living with their family at home, 4 patients living in a nursing home, and 10 patients requiring hospital care.

The severity of midline shift confirmed by preoperative CT was significantly greater in patients with unfavorable outcome (15.0 ± 4.5 mm) than in patients with favorable outcome (4.5 ± 4.0 mm, p < 0.001). The volume of hematoma measured by preoperative CT was significantly larger in patients with unfavorable outcome (155 ± 25.8 cm$^3$) than in patients with favorable outcome (68.7 ± 48.9 cm$^3$, p < 0.001). There were no significant differences in preoperative thickness of subarachnoid clot in the basal cistern between patients with favorable outcome (4.5 ± 1.0 mm) than in patients with unfavorable outcome (4.0 ± 2.6 mm, p = 0.001). There were no significant differences in incidence of angiographic vasospasm between patients with favorable outcome (4 of 14) and patients with unfavorable outcome (2 of 9). Patients with favorable outcome tended to be younger (59.3 ± 12.5 yrs) than patients with unfavorable outcome (70.9 ± 14.4 yrs), although not significantly (p = 0.058). Patients with lesions on the right tended to achieve better outcome (8 of 11) than patients with lesion on the left (5 of 12), but not significantly ($\chi^2 = 2.25$).

### Discussion

The present series illustrates our experience with a potentially helpful surgical strategy. We emphasize the use of decompression and CSF flow diversion as adjuncts to clipping of aneurysms and hematoma evacuation in the treatment of poor-grade ruptured MCA aneurysm with intracerebral hematoma. The study population included only a small number of patients, and treatment was based on a review of literature and lessons learned from the experiences of fellow neurosurgeons.\(^{28,30,32}\) The limitations in this study include the small sample size, and lack of comparisons demonstrating the superiority of our treatment strategies. Previously, functional recovery and mortality rate were reported as 37–53% and 21–35%, respectively,\(^{15,16,22}\) whereas we achieved favorable functional recovery by the GOS of 48% and mortality rate of 13%. Therefore, our results may be characterized by efficacy in functional recovery and reduction of mortality rate. Regarding functional recovery, we have noticed that a large number of patients will inevitably become SD regardless of the success of the treatment strategy. Therefore, we used prognosis-based outcome analysis as employed in the STICH trial to clarify the role of decompression surgery. We specifically focused on patients rated as SD by the GOS, who potentially exhibit improvement in a wide range of neurological deficits after aggressive treatment.

Recovery occurs over a longer period of time beyond initial hospitalization.\(^{31}\) Therefore, this study aimed to explore subsequent quality of life for these patients. The level of consciousness improved to alert in 13 patients, whereas substantial impairment remained in 7 patients. Patients with temporal hematoma can be expected to recover well even if the initial status is devastating. In our series, one patient with temporal hematoma who presented with bilateral mydriasis prior to surgery was successfully treated and now lives at home. The clinical

---

**Table 2** Decompression methods employed in poor-grade ruptured middle cerebral artery aneurysms

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>ETD</th>
<th>Uncectomy</th>
<th>LTL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporal hematoma</td>
<td>7</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Sylvian hematoma</td>
<td>10</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Frontal hematoma</td>
<td>6</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

ETD: external decompression, LTL: lateral temporal lobectomy.
Fig. 6 Scatterplot indicating the relationship between preoperative Glasgow Coma Scale (GCS) and outcome. Open circles indicate patients with favorable outcomes. Closed circles indicate patients with unfavorable outcomes. Patients with large frontal hematoma tended to present with severe symptoms, but recovery was good. Patients with sylvian hematoma tended to have poor outcomes compared to other patients with comparable GCS score. Patients with temporal hematoma could recover from the entire range of initial statuses.

course in patients with sylvian hematoma may be characterized by poor outcomes because of the significantly delayed brain edema and high incidence of delayed ischemic neurological deficit. We regard the results as better than expected for patients with sylvian hematoma. Extensive evacuation and decompressive craniotomy successfully controlled the delayed brain swelling. However, outcomes included a significant number of patients with apallic state, suggesting substantial brain damage was more profound than with the other two types of hematoma with comparable initial GCS score (Fig. 6). Three of our 4 patients who presented with brainstem symptoms and extensive frontal base hematoma with ventricle perforation seemed to demonstrate marked recovery. Therefore, we emphasize that patients with frontal hematoma showing ventricle perforation can still be salvaged even if the initial status shows signs of brainstem dysfunction.

The surgical strategy can be summarized as follows.

Temporal lobe hematoma: Hemicraniectomy may not be necessary in most cases. Placement of a ventricle catheter is not practical due to midline shift. We instead employ a combination of fenestration of the lamina terminalis and placement of cisternal drainage to divert the circulation of CSF.

Sylvian hematoma: Effective clot removal is sometimes difficult because of fibrous adhesion of the clot to small vessels, particularly in the presence of increased ICP. This can induce injury to the adjacent brain and so sufficient decompression is difficult to perform if the clot extends deep into the distal sylvian fissure. The problem lies in limited exposure of the deep sylvian fissure. We propose use of the transverse temporal gyrus (Heschl’s gyrus) as an operative landmark. Removing the intraparenchymal hematoma first creates sufficient space to work with residual hematoma in the sylvian fissure. Medical interventions such as mannitol, barbiturate, hyperventilation, and hypothermia may also be considered as adjuncts to decrease ICP.15,16,24,27,34

Frontal hematoma: Blood from the aneurysm forms frontal hematoma and extends along the external capsule, then into the frontal horn to enlarge the third ventricle.5) Ventricular perforation might have decompression effects, so the patient may escape complete uncal herniation. Although deep coma and pupil abnormalities suggest irreversible brainstem damage, distended third ventricle may be important in this pathology. Therefore, despite poor neurological status, we should consider clipping the aneurysm and evacuating hematoma from the third ventricle.

Decompression maneuver: Planning craniectomy as part of the initial operation avoids the potential need to return to the operating room several days after the occurrence of SAH. This period coincides with the onset of vasospasm. Treatment of vasospasm and elevated ICP require conflicting strategies. Therefore, we consider that effective decompression maneuvers should be performed in the initial surgery, to avoid returning to the operating room at the period when vasospasm is expected. Fenestration of the lamina terminalis is always performed regardless of the type of hematoma, as the procedure is easy and relatively atraumatic.2) Although the incidence of hydrocephalus in the chronic phase is not expected to be reduced, combination with a cisternal drainage catheter may facilitate washout of degenerative products of the subarachnoid clot. Apart from the other decompression maneuvers described above, internal decompression is potentially invasive. In patients with sylvian hematoma on the right, we consider that removal of the temporal lobe should always be an option. In patients with sylvian hematoma on the left, modest internal decompression such as selective uncectomy may be an alternative to carry out further decompression. This strategy is derived from our earlier experience that only external decompression (craniectomy) does not effectively alleviate delayed brain swelling that occasionally induces midline shift, even though the bone flap has been left out.

Aggressive treatment should be always considered regardless of initial clinical status in patients with ruptured MCA aneurysm. Recovery can be unexpectedly good, particularly in patients with fron-
Hematoma and MCA Aneurysm

10) Hutter BO, Gilsbach JM, Kreitschmann I: Quality of assistance. The authors thank Ms. Mami Itoh for her editorial can compromise the treatment of vasospasm.

11) Kassell NF, Torner JC, Haley EC Jr, Jane JA, Adams

12) Le Roux PD, Elliott JP, Newell DW, Grady MS, Winn


23) Shimoda M, Oda S, Shibata M, Tominaga J, Kittaka


References


Acknowledgments

The authors thank Ms. Mami Itoh for her editorial assistance.

Neurol Med Chir (Tokyo) 50, October, 2010

Neurol Med Chir (Tokyo) 50, October, 2010
K. Kazumata et al.


25) Starke RM, Komotar RJ, Kim GH, Kellner CP, Otten ML, Hahn DK, Michael Schmidt J, Sciacca RR, May-


27) Su CC, Saito K, Nakagawa A, Endo T, Suzuki Y, Shi-


29) Tokuda Y, Inagawa T, Katoh Y, Kumano K, Ob-
bayashi N, Yoshioka H: Intracerebral hematoma in patients with ruptured cerebral aneurysms. Surg


Address reprint requests to: Ken Kazumata, M.D., Department of Neurosurgery, Teine Kijeinkai Hospital, 1–jo 12–chome 1–ban 40–go, Maeda, Teine–ku, Sapporo 006–8555, Japan.

e-mail: kazumata@tkeijinkai.gr.jp