Efficacy of neuroendoscopic evacuation of traumatic intracerebral or intracerebellar hematoma

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Objective: To describe the successful neuroendoscopic evacuation of traumatic intracerebral or intracerebellar hematoma.

Methods: Five patients with a traumatic intracerebral or intracerebellar hematoma underwent neuroendoscopic surgery. The clinical evaluation included pre- and postoperative intracranial pressure (ICP), the Glasgow Coma Scale (GCS) score on admission and at discharge, and the Glasgow Outcome Scale score at discharge. The indications of neuroendoscopic evacuation are: progressive increase in ICP or progressive worsening of consciousness level resistant to conservative therapies, and computed tomography findings such as fourth ventricle displacement and compression of the ambient and quadrigeminal cisterns caused by an intracerebral or intracerebellar hematoma. All the patients were placed in the supine position for the procedure.

Results: The mean GCS score on admission was 9.6, and that at discharge was 13. Except for one case with traumatic intracerebral hematoma that was operated on without ICP monitoring, the mean preoperative ICP was 26 mmHg, and the mean postoperative ICP was 14.25 mmHg. The mean duration of the procedure was 77.2 minutes. The outcomes were moderate disability in 2 patients, severe disability in 2, and death in 1. That patient died of sepsis. There were no complications related to the neuroendoscopic procedures.

Conclusions: Neuroendoscopic evacuation of a traumatic intracerebral or intracerebellar hematoma offers minimally invasive surgery and may be a quick and safe life-saving procedure.

Key words: brain injury, neuroendoscopic surgery, traumatic intracerebral hematoma, traumatic intracerebellar hematoma

Introduction

Neuroendoscopic surgery has been established as a minimally invasive method for the treatment of intracerebral hematoma.1,2 Neuroendoscopic surgery has also been used for traumatic acute subdural or chronic subdural hematomas.3,5 However, the use of endoscopic surgery for traumatic intracranial hematomas remains controversial. A series of endofiberscopic removals of traumatic intracranial hematomas in 180 patients included 8 patients with a traumatic intracerebral hematoma.6 However, to our knowledge, no reports have described in detail the evacuation procedures for traumatic intraparenchymal hematomas, such as traumatic intracerebral or intracerebellar hematomas. We describe the treatment of 4 patients with a traumatic intracerebral hematoma and 1 patient with an intracerebellar hematoma that were all successfully evacuated by neuroendoscopic surgery.

Materials and Methods

Patient population

From December 2012 through May 2015, 432 patients with head trauma were admitted to Kitasato University Hospital. The present study focused on 54 patients with traumatic intracerebral or intracerebellar hematoma. Five of these 54 patients, 4 men and 1 woman aged 21–68 (mean 40.6) years, underwent neuroendoscopic surgery (Table 1). Injury mechanisms were traffic accidents in 3 patients and falls in 2 patients. The Glasgow Coma Scale (GCS) score upon admission was 3–14 (mean 9.6).
Injury severity score upon admission was 26−43 (mean 31.2). Cases 2, 3, and 5 had suffered multiple injuries to the trunk. According to the criteria for intracranial pressure (ICP) monitoring, an ICP monitor (Camino; Integra NeuroSciences, Plainsboro, NJ, USA) was placed in patients with a supratentorial lesion. Except for Case 5, initial ICP was 8−50 mmHg (mean, 26.25 mmHg).

Operative indications
In Kitasato Hospital, evacuation of a traumatic intracerebral or intracerebellar hematoma is performed through a craniotomy according to the guidelines for the surgical management of traumatic brain injury, which summarize the operative indications as: signs of progressive neurological deterioration; signs of mass effect on computed tomography (CT); GCS scores of 6−8 with frontal or temporal contusions greater than 20 cm³ with midline shift of at least 5 mm and/or cisternal compression on CT, and any lesion greater than 50 cm³; uncontrollable high ICP despite conservative therapy; and sustained ICP greater than 30 mmHg.7,8

In contrast to these indications for craniotomy, we emphasize neuroendoscopic evacuation under the following conditions: progressive increase in ICP or progressive worsening of consciousness level resistant to conservative therapies, and CT findings such as fourth ventricle displacement and compression of the ambient and quadrigeminal cisterns caused by intracerebellar hematoma. The 4 patients with a traumatic intracerebral hematoma had persistently high ICP, despite surgical procedures including evacuation of the hematoma, external decompression, and ventricular drainage, and conservative therapies including sedation, brain hypothermia, mannitol administration, and body temperature control. The patient with the traumatic intracerebellar hematoma presented with a progressive increase of the hemorrhagic component, fourth ventricle displacement, and cisternal compression. Therefore, neuroendoscopic surgery was performed. Written informed consent was obtained from each patient or family member before performing the neuroendoscopic procedures.

Neuroendoscopic evacuation of traumatic intracerebral or intracerebellar hematoma
To facilitate the procedure, the patient was placed in the supine position with the head resting on a horseshoe frame. The same skin incision was used in cases of traumatic intracerebral hematoma located on the ipsilateral side as the craniectomy. The site of the burr hole was based on the hematoma location and the proposed trajectory in cases of traumatic intracerebral hematoma on the contralateral side of the craniectomy. The burr hole was placed midway between the inion and the mastoid process for the evacuation of the traumatic intracerebellar hematoma. The transparent sheath (Neuroport, regular size; Olympus Corp., Tokyo) was inserted into the surface of the target hematoma via a burr hole. A rigid-rod neuroendoscope with 2.7-mm or 4-mm outer diameter and 0-degree viewing angle (EndoArm Neuroendoscopy System; Olympus Corp.) was introduced through the transparent sheath. Neuroendoscopic evacuation was performed under ICP monitoring in all cases of a traumatic intracerebral hematoma. Artificial cerebrospinal fluid (Artcereb; Otsuka Pharmaceutical Co., Tokyo) was used as the irrigation fluid.

Table 1. Case summary, surgical data, and outcome

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Location of hematoma</th>
<th>Injury mechanism</th>
<th>GCS score</th>
<th>ISS</th>
<th>Multiple injuries</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>On admission</td>
<td>At discharge</td>
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<tr>
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<td>TA</td>
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<tr>
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<td>Fall</td>
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<td>25</td>
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<tr>
<td>5</td>
<td>67, M</td>
<td>Rt. CH</td>
<td>Fall</td>
<td>14</td>
<td>15</td>
<td>36</td>
</tr>
</tbody>
</table>

*After a 9-day hospital course, this patient died of sepsis.
GCS, Glasgow Coma Score; ISS, Injury Severity Score; ICP, intracranial pressure; GOS, Glasgow Outcome Score; FL, frontal lobe; CH, cerebellar hemisphere; TA, traffic accident; D, dead; SD, severe disability; MD, moderately disabled
Results

Puncture of the hematoma was successful at the first attempt in all the cases. The hematoma contents were soluble in high-pressure water in the central portion, but the external portion consisted of hard tissue in Cases 1 and 2. Neuroendoscopic evacuation of a hematoma in the central portion was relatively easy with minimal intraoperative bleeding, but the hard tissue could not be removed with suction. The hematoma content was soft and easily aspirated in Cases 3–5. Confirmation of decreased ICP was the indication to stop the procedure in the 4 patients with traumatic intracerebral hematoma. In Case 5 with traumatic intracerebellar hematoma, visual confirmation of slack and pulsation of the cerebellum was the best indication to stop the procedure. There was no intraoperative or postoperative bleeding in any of the patients.

Table 1 shows the pre- and postoperative ICP, time from hospital presentation to surgery, duration of the procedure, and outcome. Except for Case 5, preoperative ICP was 25–27 (mean 26) mmHg, and postoperative ICP was 12–16 (mean 14.25) mmHg. ICP finally decreased to 5–10 (mean 8.25) mmHg just before monitor removal. Time from hospital presentation to endoscopic surgery was 2–38 (mean 18) hours. Duration of surgery was 30–100 (mean 77.2) minutes. Except for Case 4, GCS score at discharge was 9–15 (mean 12.3), and the outcomes were: moderate disability in 2 patients, severe disability in 2, and death in 1. The Case 4 patient died of sepsis. There were no complications related to the endoscopic procedures.

Case 2
A 21-year-old man suffered craniofacial and thoracic injuries in a bicycle accident. On admission, the patient had a GCS score of 11 (E3V3M5). CT revealed a thin left acute epidural hematoma and bifrontal contusions (Figure 1A). His level of consciousness decreased from GCS score of 11 to 8 during the initial 5 hours of hospitalization. Repeat CT showed enlargement of the left acute epidural hematoma and bifrontal contusion. An ICP monitor was placed. The opening pressure was 32 mmHg. Evacuation of the epidural hematoma and left external decompression were performed, and an external ventricular drainage was inserted (Figure 1B). The patient was treated medically to control the ICP postoperatively. However, the difficulty in controlling the ICP gradually increased. CT showed evolution of the contusion with accumulation of edema fluid in the right frontal lobe and disappearance of the subarachnoid space in the quadrigeminal and ambient cisterns (Figure 1C, D). Endoscopic evacuation of the right frontal hematoma was performed. Subsequently, the ICP could be controlled. Postoperative CT revealed improved midline shift and appearance of the subarachnoid space in the quadrigeminal and ambient cisterns (Figure 1E, F). After an 86-day hospital course, the patient was discharged for further rehabilitation with a GCS score of 13.

Case 3
A 25-year-old woman suffered craniofacial and thoracic injuries and injuries of the extremities after being hit by a train. On admission, she had a GCS score of 3. She had anisocoria with dilation of the right pupil. Initial CT revealed multiple skull fractures, a right acute epidural

<table>
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<tr>
<th>ICP (mmHg)</th>
<th>ICP before monitor removal (mmHg)/Time to removal (day)</th>
<th>Time from hospital presentation to endoscopic surgery (hrs)</th>
<th>Duration of surgery (min)</th>
<th>GOS score at discharge</th>
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<td>Post-operative</td>
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<td>12</td>
<td>10/7</td>
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</table>
Figure 1. Case 2. A. Initial computed tomography (CT) scan demonstrating a thin left acute epidural hematoma (arrowhead) and bifrontal contusions with small bleeds in the so-called salt-and-pepper pattern (arrows). B. CT scan after external decompression showing enlargement of the traumatic intracerebral hematoma (TICH) in the right frontal lobe (arrow). C, D. CT scans after external decompression depicting the right frontal hematoma with the formation of a niveau-like shadow (arrow) and disappearance of the subarachnoid space in the quadrigeminal and ambient cisterns (arrowhead). E, F. CT scans after endoscopic evacuation of the TICH revealing reduction of the hematoma (arrow) and appearance of the subarachnoid space in the quadrigeminal and ambient cisterns (arrowhead).

Figure 2. Case 3. A. Initial CT scan demonstrating multiple fractures, a right acute epidural hematoma (arrowhead), traumatic subarachnoid hemorrhage (black arrow), and pneumocephalus (white arrow). B. CT scan after external decompression showing enlargement of the TICH in the right frontal lobe (white arrow). C. CT scan after endoscopic evacuation of the TICH depicting reduction of the hematoma and improvement of the midline shift.
hematoma, traumatic subarachnoid hemorrhage, and pneumocephalus (Figure 2A). Evacuation of the right acute epidural hematoma and external decompression were immediately performed, and an external ventricular drainage was inserted. Additionally, an ICP monitor was placed. The opening pressure was 8 mmHg. CT after external decompression revealed enlargement of the hematoma in the right frontal lobe and midline shift (Figure 2B). The ICP had increased and was difficult to control with conservative therapies. Endoscopic evacuation of the hematoma in the right frontal lobe through the decompressive craniotomy was performed (Figure 2C). Postoperatively, the ICP decreased. After a 172-day hospital course, the patient was discharged to a nearby hospital with a GCS score of 9.

Case 5
A 67-year-old man fell from a 20-meter cliff into a mountain stream. The patient suffered multiple craniofacial and thoracic fractures and hepatic rupture. The patient was airlifted by a helicopter to our hospital. A thoracic drainage tube was inserted for pneumothorax on the trip to the hospital. On admission, his GCS score was 14 (E3V5M6). CT showed a hematoma in the right cerebellar hemisphere (Figure 3A). His GCS score decreased from 14 to 10 during the initial 3 hours of hospitalization. Follow-up CT revealed enlargement of the hemorrhagic component and mass effect (Figure 3B, C). Endoscopic evacuation of the hematoma in the right cerebellar hemisphere was performed. The patient was placed in the supine position with the head resting on a horseshoe frame. The burr hole was placed midway between the inion and mastoid process (Figure 3D). Postoperative CT showed remarkable improvement in fourth ventricle displacement and cisternal compression (Figure 3E, F). His GCS score had recovered to normal on the fourth postoperative day.

Figure 3. Case 5. A. Initial CT scan obtained on admission demonstrating a hematoma (arrow) in the right cerebellar hemisphere. B, C. Repeat CT scan obtained 3 hours after hospitalization revealed hematoma enlargement with fourth ventricle displacement and cisternal compression. D. The patient was placed in the supine position with the head resting on a horseshoe frame. The burr hole (black arrow) was placed midway between the inion (arrowhead) and the mastoid process (white arrow) in the posterior fossa TICH. E, F. CT scans after endoscopic evacuation of the TICH showing reduction of the hematoma and remarkable improvement in cisternal compression (arrowhead).
Discussion

Neuroendoscopic surgery for traumatic intracranial hematoma is a controversial issue, but these 5 cases indicate that neuroendoscopic surgery can be highly effective for the treatment of traumatic intracerebral or intracerebellar hematoma. The development of neuroendoscopy and associated devices has been important in the prevalence of neuroendoscopic surgery for the treatment of hypertensive intracerebral hemorrhage, particularly considering safety and efficiency. However, few reports have described neuroendoscopic surgery for traumatic intracranial hematomas (chronic subdural and acute subdural hematomas). Neuroendoscopic evacuation of acute subdural hematoma has been applied for elderly patients with brain atrophy and a relatively thick hematoma because the wider subdural space favors endoscopic manipulation. In the present cases, neuroendoscopic surgery was safe and effective for the treatment of traumatic intracerebral or intracerebellar hematomas. However, neuroendoscopic surgery for these conditions still presents some problems, especially the method of hemostasis. Fortunately, our patients could be treated without intraoperative bleeding. We planned to treat any intraoperative bleeding by the application of a combined irrigation sucker (Fujita Medical Instrument, Tokyo) to the bleeding point followed by coagulation with electrical or radiowave methods (Surgi-Max; Ellman International, Inc., Hicksville, NY, USA).

Traumatic intraparenchymal hematoma is categorized into the type without cerebral contusion and edema, and the type with massive edema associated with fusion of small bleeds in the salt-and-pepper pattern. In the former type, the major reason for increased ICP is the intracerebral hematoma, so the object of treatment is surgical removal of the hematoma (e.g., Cases 3−5). In the latter type, increased ICP is caused by contusional hematoma and associated edema, so the treatment must include contusion necrotomy (e.g., Cases 1 and 2). Contusional edema is classified into the early massive type and the delayed pericontusional type, based on the occurrence time and mechanism. The early massive type occurs within 24 hours of injury and is difficult to control with conservative therapy. Early massive edema is caused by accumulation of edema fluid in the central area of contusion. In contrast, the delayed pericontusional type occurs 2 to 3 days after injury, mainly in the white matter. Delayed pericontusional edema is mainly caused by vasogenic edema. Early massive edema is thought to be the main cause of poor clinical outcome. According to analysis of the data obtained from the Japan Trauma Data Bank, patients with cerebral contusion who "talk and deteriorate," treated with conservative therapies or with decompressive surgery, show improvements in the Glasgow Outcome Scale at 6 months after traumatic brain injury, indicating that the mortality rate can be significantly reduced by decompressive surgery. Head CT of Cases 1 and 2 revealed accumulation of edema fluid in the central portion of the contusion. Endoscopic removal of contusional hematoma and associated edematous fluid in the central area of contusion was performed but not for total contusion necrotomy. Neuroendoscopic evacuation was extremely difficult to achieve the external portion which consisted of hard tissue. Also, we consider that removal of the external portion is not essential because neuroendoscopic evacuation of high-pressure fluid in the central portion was enough to decrease the ICP.

Secondary brain injury occurs as the result of secondary insults including systemic factors such as hypotension, hypoxia, infection, anemia, acid-base disorder, electrolytic imbalance, impaired glucose level, and fever. Such systemic factors can be exacerbated by multiple trauma, and invasive surgical procedures might well increase the threat. Therefore, in the present study, neuroendoscopic evacuation was chosen rather than craniotomy due to advanced multiple trauma and poor condition of these 5 patients. Traumatic intracerebral hematoma on the contralateral side to external decompression in Case 2 was approached through a small incision in the right forehead. Neuroendoscopic evacuation is more effective than craniotomy for the treatment of traumatic intracerebral hematoma located on the contralateral side of the craniectomy in patients with advanced multiple trauma and who are in poor condition.

Traumatic intracerebellar hematoma is extremely rare, as the incidence rate was reported to be approximately 0.72% of all head trauma cases. To our knowledge, there have been no reports on the use of endoscopic surgery for traumatic intracerebellar hematoma. Posterior fossa craniectomy requires placing the patient in the prone position, but endoscopic surgery does not. Therefore, the length of time required for the position change can be shortened and enable immediate evacuation of the hematoma. In addition, suspected tension pneumothorax, cardiac tamponade, and intrathoracic or intraperitoneal bleeding caused by severe trauma can be monitored with serial ultrasonography and promptly treated during the evacuation of a hematoma with the patient in the supine position. Therefore, based on these advantages and
results, because it is the easiest and most efficacious method currently available, evacuation by endoscopic surgery should be the first line of treatment for traumatic intracerebellar hematomas.

References