INTRODUCTION

Intracranial mass lesions in head traumas are typically classified into three categories: epidural hematoma (EDH), subdural hematoma (SDH), and intracerebral hematoma (ICH). Management guidelines have been established to determine when to operate on each type of mass lesion. However, no universal criteria for the treatment of multiple lesions currently exist. Management in patients with multiple lesions who suffered from a certain type of predominant pathology was reported to not be significantly different from that of patients with only one lesion of the same type. Simultaneous surgery for multiple lesions is a rare situation.

Bilateral surgery has rarely been published in case reports for sequential evolution of a contralateral hematoma after removal of a first one. The rate of bilateral surgery in brain trauma operations cannot be estimated, but lesions requiring consecutive contralateral surgery were almost always EDHs. When each of multiple masses has sufficient volume to increase the intracranial pressure, they should be removed simultaneously. In this report, we present our experiences of single-session operations for bilateral traumatic intracranial hematomas.

MATERIALS AND METHODS

Operative cases for bilateral intracranial hematomas were retrieved in a prospectively collected database of head injuries for a recent 5-year period. In total, 666 cases of acute intracranial hematomas were admitted to our institution and 203 cases (30.4%) underwent evacuation of the hematomas and/or decompressive craniectomies. We retrospectively reviewed the medical records and the computed tomography (CT) scans of patients who underwent surgery. Among the operative cases, only eight (3.9%) underwent operations for bilateral intracranial hematomas in a single session. Injury mechanism, initial Glasgow Coma Scale score, types of intracranial lesions, surgical methods, and Glasgow outcome scale were evaluated.

Conclusion: Bilateral intracranial hematomas that should be removed in a single-session operation are rare. Epidural hematomas almost always occur in these cases and should be removed first to prevent the hematoma from growing during the surgery. Then, the other hematoma, contralateral to the EDH, can be evacuated with a small craniotomy.
A single session was defined as evacuation of bilateral intracranial supratentorial hematomas with separate scalp incisions and under the same general anesthesia. Operations for bifrontal craniotomy/craniectomy and chronic subdural hematoma were excluded. Patients who suffered a secondary intracranial hematoma after a spontaneous intracerebral hemorrhage were also excluded. Retrieved data included age at presentation, gender, mechanism of injury, Glasgow Coma Scale score on admission, Glasgow outcome scale at discharge, and types of intracranial hematoma and operation methods on each side.

RESULTS

All the cases were males, with mean age of 54.8 (range, 15–80) years. The most common mechanism of injury was a fall (four cases); there were two cases of pedestrian traffic accidents, one case of a slip, and another of unknown trauma (Table 1). The types of intracranial lesions consisted of EDH/ICH in five, EDH/EDH in one, EDH/SDH in one, and ICH/SDH in one (Table 1). Interestingly, no midline shift was found in brain CT scans, but obliteration of the basal cisterns, compression of the lateral ventricle, and effacement of cortical sulci was observed. These findings meant the elevated intracranial pressure exerted equally by both sides. All of the cases except one had an EDH. The EDHs were usually evacuated with a craniotomy. All the ICHs were removed through a burr hole craniostomy and the site of the SDH was managed with decompressive craniectomy and hematoma evacuation. When the patients had an EDH, the EDH was always evacuated first. We designed to avoid serious complications like an expansion of the EDH during the surgery. No re-expansion of a hematoma occurred intraoperatively or postoperatively. All patients, except the one who was severely injured, survived.

Illustrative cases

Case 5

A 48-year-old man came to the emergency room in semi-comatose consciousness. He had fallen from a construction elevator, about 3 m high. Severe scalp swelling was seen in his left parietal and a linear skull fracture was seen in the left temporo-parietal area. Both pupils were fixed with no light reflex and his motor responses showed abnormal flexion to painful stimuli.

On a CT scan, a large EDH in the left parietal over four times the thickness of the skull and an ICH in the right temporal lobe were found (Fig. 1). The basal cisterns and cortical sulci were obliterated, but no midline shift was seen.

He underwent emergency decompressive surgery and hematoma evacuation. We planned to perform the EDH first with a decompressive craniectomy and then to aspirate the ICH via a burr hole. We considered the decompressive surgery essential because of his poor consciousness and the obliteration of the cisterns and cortical sulci.

Table 1. Summary of operative cases for the bilateral traumatic intracranial hematomas in single session

<table>
<thead>
<tr>
<th>Age/gender</th>
<th>Injury mechanism</th>
<th>GCS</th>
<th>Type of intracranial hematomas</th>
<th>Operation method</th>
<th>Skull fracture</th>
<th>GOS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>EDH (1)</td>
<td>SDH (2)</td>
<td>cr/e</td>
<td>cr/e</td>
</tr>
<tr>
<td>1 57/M</td>
<td>Falling</td>
<td>9</td>
<td>EDH (1)</td>
<td>SDH (2)</td>
<td>cr/e</td>
<td>cr/e</td>
</tr>
<tr>
<td>2 15/M</td>
<td>Falling</td>
<td>8</td>
<td>EDH (1)</td>
<td>EDH (2)</td>
<td>cr/e</td>
<td>cr/e</td>
</tr>
<tr>
<td>3 80/M</td>
<td>Falling</td>
<td>14</td>
<td>ICH (2)</td>
<td>EDH (1)</td>
<td>Burr hole</td>
<td>cr/o</td>
</tr>
<tr>
<td>4 71/M</td>
<td>Pedestrian TA</td>
<td>14</td>
<td>ICH (2)</td>
<td>EDH (1)</td>
<td>Burr hole</td>
<td>cr/o</td>
</tr>
<tr>
<td>5 48/M</td>
<td>Falling</td>
<td>5T</td>
<td>ICH (2)</td>
<td>EDH (1)</td>
<td>Burr hole</td>
<td>cr/e</td>
</tr>
<tr>
<td>6 44/M</td>
<td>Unknown</td>
<td>13</td>
<td>EDH (1)</td>
<td>ICH (2)</td>
<td>cr/o</td>
<td>Burr hole</td>
</tr>
<tr>
<td>7 59/M</td>
<td>Slip down</td>
<td>4T</td>
<td>ICH (2)</td>
<td>SDH (1)</td>
<td>Burr hole</td>
<td>cr/e</td>
</tr>
<tr>
<td>8 65/M</td>
<td>Pedestrian TA</td>
<td>4T</td>
<td>ICH (2)</td>
<td>SDH (1)</td>
<td>Burr hole</td>
<td>cr/e</td>
</tr>
</tbody>
</table>

(1) & (2) : operation order as (1) is first and (2) is later. GCS : Glasgow Coma Scale, GOS : Glasgow Outcome case, TA : traffic accident, EDH : epidural hematoma, ICH : intracerebral hematoma, SDH : subdural hematoma, cr/o : craniotomy, cr/e : craniectomy, MD : moderate disability, SD : severe disability, M : male

Fig. 1. CT scans of case 5. Preoperative CT images (A and B) show a large epidural hematoma in the left temporo-parietal area and an intracerebral hematoma in the right temporal lobe. The basal cisterns and cortical sulci are obliterated but no midline shift was seen. A linear skull fracture is seen on the left temporal bone (C). The hematomas were removed through a craniectomy in the left temporo-parietal and a burr hole in the right temporal area. The cisterns can be seen in the postoperative CT image (D).
sulci. To remove the EDH in the left temporo-parietal, a large craniotomy should be performed. So, a decompressive craniectomy was carried out on the left side. We made a large U-shaped scalp incision on the left temporo-parietal area for the removal of the EDH, which was caused by a torn middle meningeal artery. Then, a vertical linear scalp incision was made on the right temporal for the evacuation of the ICH. Each of the hematoma surgeries was performed with a separate head position and surgical drape. Evacuation of the hematomas proceeded uneventfully (Fig. 1). He underwent autograft cranioplasty at 2 months after the decompressive surgery. His consciousness recovered fully, but unfortunately he suffered deafness in both ears.

Case 7
A 59-year-old man was admitted in semi-comatose consciousness. He had been drunk and slipped the day before he came to the emergency room. A scalp contusion was found in the right temporal and a linear skull fracture was seen in the same area as the scalp injury. His systolic blood pressure was 180 mm Hg. He showed abnormal flexion to painful stimuli. No light reflex was observed in either pupil. On a CT scan, a large EDH in the right parietal over three times the thickness of the skull and an ICH in the left temporal lobe was found (Fig. 2). The basal cisterns were obliterated and both lateral ventricles were compressed. No midline shift was seen. We planned to address the EDH first, with an appropriate craniotomy, and then to aspirate the ICH via a burr hole. The skull bone flap of the right temporo-parietal was restored because the dura was not very tense after the EDH evacuation. The middle meningeal artery was torn beneath the skull fracture. Then, the left frontal scalp was incised for small craniotomy and aspiration of the ICH in the left frontal lobes. Each hematoma surgery was performed using a different head position and surgical drape. Evacuation of the hematomas was uneventful (Fig. 2). He recovered to continue his normal life without assistance, but he needed to take antiepileptic drugs for post-traumatic epilepsy.

**DISCUSSION**

Multiple intracranial hemorrhages could be a common phenomenon in traumatic brain injury. However, operations for traumatic intracranial hematomas are usually unilateral. Bilateral operations have been reported in bifrontal decompressive craniectomies for brain swelling or sequential contralateral removal of a newly developed hematoma after a one-side evacuation of an intracranial hematoma. In our series of patients over 5 years, only 1.2% of the head-injury patients with hematomas underwent simultaneous operations for bilateral intracranial hematomas in a single session.

The delayed development of a contralateral EDH after evacuation of an acute SDH is a rare phenomenon. However, many neurosurgeons are familiar with the possibility. When brain swelling occurs even with complete removal of a SDH, neurosurgeons almost always suspect the development of an EDH on the contralateral side. Immediate postoperative CT scans or contralateral diagnostic burr holes are then essential, and a sequential contralateral craniotomy would follow.

The EDH may commonly be responsible for the worsening neurological status, rather than the underlying brain injury, and evacuation of the EDH may have value in improving the prognosis in these patients with severe head injuries. However, it is unfortunate that delayed treatment of a large contralateral EDH can cause postoperative neurological deterioration and even be a cause of death.

Intracranial traumatic hematomas can evolve with time. Also, evacuation of an intracranial hematoma may facilitate the growth of other hematomas existing in a preoperative CT scan. The reduction in intracranial pressure due to evacuation of a first hematoma may also play an important role in hematoma growth due to the release of mechanical pressure, which may be hemostatic. Almost all reports of contralateral hematoma growth after surgery were related to EDH. Rapid growth of an EDH contralateral to a hematoma evacuation may cause intraoperative brain swelling, which may lead to confusion between primary brain edema and contralateral EDH development. Also, a contralateral EDH could cause deterioration in neurological status and even death. The development of growing hematomas or a new lesion should be ruled out by repeated CT scans after surgery. Intraoperative imaging, such as CT and ultrasound, can also be valuable for detecting any new lesion during surgery.
However, intraoperative imaging tools may not always be available and the growth of other hematomas is a rare phenomenon. It may be better to consider how to avoid the development of problematic hematoma growth in head injury patients.

For the surgical treatment for EDH, there are insufficient data to support one method. However, craniotomy provides a more complete evacuation of the hematoma. To completely remove the EDH, a well-designed scalp incision and craniotomy is typically required. The craniotomy should be sufficiently large to visualize all of the hematoma. When an EDH grows beyond the hematoma area estimated in the preoperative CT, inadequate removal of the EDH may result. Unlike the EDH operation, surgery for ICH and SDH can typically be performed safely through a small craniotomy of less than the hematoma area if the bleeding can be anticipated preoperatively.

In our cases, the most common combination was EDH/ICH, in five cases. Only one case showed EDH/EDH. No midline shifting was found. An EDH could grow on removing a contralateral hematoma because of the release of a tamponade effect. All the cases of EDH were accompanied by skull fractures, caused by the original coup injury. When the dural artery is torn, rapid enlargement of the EDH would be expected. Furthermore, the brain injury could be aggravated when a contralateral EDH develops rapidly. Also, even though an EDH does not grow that much during contralateral hematoma evacuation, incomplete removal of the hematoma may be inevitable because of the rather smaller craniotomy. Thus, we propose that the EDH evacuation should be accomplished as the first step in removal of bilateral intracranial hematomas. An adequate craniotomy should be performed for the removal of the EDH; a smaller craniotomy or burr hole will then be sufficient to evacuate the contralateral intracranial hematoma.

**CONCLUSION**

Bilateral intracranial hematomas, requiring simultaneous removal in a single session, are rare. An EDH always occurs in these cases. To avoid problematic situation of contralateral hematoma growth, the EDH should be firstly dealt with an appropriate craniotomy. Then, the other hematoma, contralateral to the EDH, can be evacuated by means of a small craniotomy.

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**References**


