A Study on the Indication of External Decompressive Hemicraniectomy for Acute Subdural Hematomas

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Summary

Fourteen cases of surgically treated acute subdural hematomas were studied. The indication of external decompressive hemicraniectomy for these cases of acute subdural hematomas was studied in detail according to postoperative intracranial pressure (pICP) changes in combination with their preoperative neurological, cardiorespiratory and angiographic findings.

The pICP changes were classified into three groups according to the maximum values of intracranial pressures and the patterns of the pressure changes.

Group one: pICP increased rapidly and progressively immediately after the operation, and then it reached the level of mean arterial blood pressure. This rapid increase of pICP in this group seemed to be the result of cerebral vasomotor paralysis. There was no effective treatment for these cases.

Group two: pICP remained in the range of 40 to 70 mmHg. Preoperative neurological examinations of these cases showed semicoma or coma not having the signs of brain stem dysfunction. Preoperative angiograms revealed the delay of circulation time and fairly good filling of small arteries in an early arterial phase. Operative findings in these cases showed that extensive cerebral contusion affected more than two lobes without any definitive brain swelling. Postoperative intracranial pressure might have continued to increase as shown in Group one, if external decompressive hemicraniectomy had not been performed for these cases. It was considered that external decompressive hemicraniectomy should be restricted to this group.

Group three: pICP remained 10 to 30 mmHg. Indication of external decompressive hemicraniectomy was not suggested for cases in this group.

Key words: Acute subdural hematoma, acute intracranial hypertension, external decompressive hemicraniectomy, postoperative intracranial pressure

Introduction

The mortality rate of acute subdural hematoma still remains high. Its principal treatment consists mainly of subdural clot removal and control of acute brain swelling. External decompressive hemicraniectomy has contributed to control acute intracranial hypertension and this surgical procedure can be applied even when the acute intracranial hypertension was caused by massive brain edema without intracranial hematoma.3)

During the past 10 years, we routinely performed external decompressive hemicraniectomy in about 100 cases of acute subdural hematoma, whenever the preoperative level of consciousness was coma or semicoma. This surgical procedure was carried out in 14 recent cases under continuous monitoring of intracranial pressure (ICP) and several vital parameters. The pathophysiology of acute intracranial hypertension by monitoring several vital parameters and the ICP have been previously reported.14,15) The purpose of the present paper is to conduct further study to
obtain strict indication of external decompressive hemicraniectomy for patients with acute subdural hematoma from postoperative changes of ICP data in combination with each patient’s preoperative condition.

**Clinical Material and Methods**

Fourteen patients with acute subdural hematoma without primary brain stem injury, who were admitted to Kurume University Hospital during the past 4 years were studied. Nine were male and five female. Age distribution was 5 to 82 with an average of 42.1 years.

According to preoperative neurological examinations, all cases had some clinical signs of transtentorial herniation caused by supratentorial mass lesions. In this paper, transtentorial herniation was divided into two stages according to the manifestations of clinical signs: herniation was categorized as early if clinical signs consisted mainly of the impairment of third cranial nerve such as dilated pupil with sluggish light reaction; and as late if clinical signs consisted of cardiorespiratory symptoms such as hyperventilation, irregular respiration, bilateral dilated pupils, decerebrate rigidity and systemic hypotension.

All the cases underwent unilateral large external decompressive hemicraniectomy extending over the fronto-temporo-parieto-occipital region and evacuation of hematoma. The detail of this operative procedure of unilateral external decompression and the method of monitoring the ICP were reported in previous papers.

Intracranial pressure was monitored at least for 72 hours after the operation by Ladd’ Fiber Optic ICP Measuring System (Model 1005), the sensor of which was placed in the epidural space under the bone edge of decompressive hemicraniectomy.

**Results**

Five patients in this series survived, giving an overall survival rate of 35.7%. Four of the survivors fully recovered and returned to normal life within 3 months after the operation. One, (CN-6), had an additional ventriculo-peritoneal shunting operation under the diagnosis of posttraumatic normal pressure hydrocephalus. The other survivor, (CN-12), remained in a vegetative state. Good recovery was, therefore, obtained in 28.6% of the whole series of this study.

The average age of the 5 survivors was 39.2 years, and that of the 9 non-survivors was 43.7 years. The intervals from trauma to operation were 4 to 54 hours with an average of 14.4 hours. The average interval from trauma to operation of the 5 survivors was 17.2 hours and that of the non-survivors was 12.6 hours. Differences were not statistically significant.

In all cases, preoperative neurological examinations showed coma or semicoma in the level of consciousness and developing signs of transtentorial herniation: early in 8; late in 6 cases (Table 1).

Postoperative changes of ICP in each case are shown in Figs. 1 and 2. The patients were classified into three groups according to the
<table>
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<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Consciousness</th>
<th>Preoperative conditions</th>
<th>Cardiorespiratory impairment</th>
<th>Trauma to operation (hrs.)</th>
<th>Hematoma (g)</th>
<th>Operative findings</th>
<th>Outcome</th>
<th>Trauma to death (days)</th>
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maximum level of pICP and the patterns of the changes of pressure.

Group one: the pICP increased rapidly and progressively immediately after the operation and reached the level of mean arterial blood pressure (MABP). In some cases, the pICP reached a level above the MABP. The preoperative level of consciousness in Group one was coma or semicoma. In this group, there were signs of late stage of transtentorial herniation: bilateral dilated and fixed pupils in 3/6 cases (50%); decerebrate rigidity in 2/6 (33%); and cardiorespiratory impairment in 4/6 (67%). Cerebral angiograms revealed the delay of circulation time, poor filling of small arteries in an early arterial phase (Fig. 3) and stasis of contrast medium in main arteries in a capillary phase. Operative findings showed marked cerebral contusion and brain swelling over the hemisphere beneath the subdural hematoma. All the patients in the Group one died several hours after the operation.

Group two: the postoperative elevation of ICP remained in the range of 40 to 70 mmHg. The preoperative level of consciousness of the patients in this group were semicoma or coma with signs of early stage of herniation. Cerebral angiograms revealed the delay of circulation time, poor filling of small arteries in an early arterial phase (Fig. 4), but the delay of circulation time. Operative findings showed cerebral contusion localized within one lobe and no definitive sign of brain swelling. All patients in this group survived.

Group three: postoperative elevation of ICP remained between 10 to 30 mmHg. The preoperative level of consciousness of the patients in this group was semicoma or coma with signs of early stage of herniation. Cerebral angiograms revealed almost complete filling of small arteries in an early arterial phase (Fig. 5), but the delay of circulation time. Operative findings of the brain showed cerebral contusion localized within one lobe and no definitive sign of brain swelling. All patients in this group survived.

Discussion

The outcome of the patients with acute subdural hematomas is still extremely disastrous. In this series, the mortality rate was 64.3%, and the good recovery rate was only 28.6%. Important factors which influence the prognosis of the patients were the location and amount of subdural clot, presence or absence of primary brain stem injury and the degree of cerebral contusion. The principle of
Fig. 3  Right common carotid angiograms of a patient in Group one showing poor filling of small arteries (early arterial phase).

Fig. 4  Left common carotid angiograms of a patient in Group two showing fairly good filling of small arteries (early arterial phase).
Fig. 5 Right common carotid angiograms of a patient in Group three showing almost complete filling of small arteries (early arterial phase).

Treatment is how to manage markedly raised ICP caused by the presence of subdural clot, cerebral contusion, and subsequent brain edema.

In a previous paper, we reported that external decompressive hemicraniectomy for patients with acute subdural hematoma should be restricted to those who had unilateral neurological signs caused by unilateral hematoma, and to those in whom the level of consciousness was better than semicoma in cases with bilateral neurological signs. In the present paper, the indication of this surgical procedure was re-investigated in detail on the basis of postoperative ICP changes in combination with preoperative neurological, cardiorespiratory and angiographic findings.

Acute brain swelling in this paper was defined as follows: Upon opening the dura, the brain protruded extensively with pulsation beyond the edge of craniotomy and this was uncontrollable with hyperventilation and intravenous administration of Mannitol. The mortality rate in such cases of acute brain swelling was 100%, and it was considered that there was no effective method for vasomotor paralysis whenever it had actually occurred.

McNealy and Plum observed in detail the clinical manifestations of brain stem dysfunction caused by transtentorial herniation in the cases with supratentorial mass lesions, and classified them into several stages in relation to the process of herniation from the beginning to the endstage. Based on their report as well as other’s, we defined in brief, clinical manifestations of brain stem dysfunction by transtentorial herniation as follows: unilateral dilated pupil with sluggish light reaction was earlier signs of transtentorial herniation (third nerve stage); while sustained hyperventilation, bilateral dilated pupils, and bilateral decerebrate rigidity were later signs of herniation (medullary stage).

Postoperative changes of ICP and some vital parameters of a patient in the Group one are illustrated in Fig. 6. Postoperative ICP increased rapidly, progressively, and finally reached the level of MABP. From these findings, it was considered that the changes of pICP was the result of cerebral vasomotor paralysis in Group one cases. Preoperative level of consciousness was coma with signs of late stage herniation. Cerebral angiograms revealed the delay of circulation time and poor filling of
small arteries in an early arterial phase. Extensive cerebral contusion and acute brain swelling were found at operation, which did not respond to intravenous administration of Mannitol or hyperventilation. Operative result showed that decompression was not effective in these cases. The reason why external decompression was not effective in these patients can be considered likely to be that brain swelling was so extensive after external decompression that cerebral venous return was further impaired at the site of lateral lacuna by bone edge and swollen brain.

The pattern of postoperative changes of ICP as in Group two patients remained in the range of 40 to 70 mmHg. Patients in this group were in semicoma or coma without late signs of transtentorial herniation in preoperative neurological examinations. Cerebral angiograms revealed the delay of circulation time and fairly good filling of small arteries in an early arterial phase. There was cerebral contusion without definitive brain swelling affecting more than two lobes at operation, which responded to intravenous administration of Mannitol or hyperventilation.

According to data reported by Moody et al., 10 dogs with experimentally raised ICP died within 12 hours when they were not treated with external decompression, whereas those subjected to decompression lived 10 days or more. On clinical study, Ranshoff et al. reported that a decreased mortality rate from 90 to 60% and full recovery rate of 28% were observed by performing hemicraniectomy for treatment of acute subdural hematomas. However, they later reported pessimistic results that their total survival rate was 10% and functional recovery rate was 4% in 50 clinical cases with acute subdural hematoma. They further concluded that the indication of this surgical procedure should be restricted to patients who did not have any evidence of brain stem dysfunction, but who were secondarily deteriorated by marked hemispheric edema and/or subdural clot.

In general, ischemic anoxia of the brain is induced by a decrease of cerebral blood flow under the condition of raised ICP. Intracranial pressure is further elevated by an increase of cerebral blood volume due to the dilatation of cerebral vessels responding to ischemic changes of the brain. Pressure gradient between SABP and ICP is rather decreased by a progressive elevation of ICP, despite the elevated systemic blood pressure through the mechanism of vasopressor response. As a result, cerebral vasomotor paralysis is caused by a marked decrease of cerebral blood flow which consequently results in brain swelling.

Hayashi et al. reported in their experimental study that cerebral perfusion pressure was kept at 40 mmHg under the range of 50 to 70 mmHg of epidural pressure because of the buffering effect of craniospinal cavity. When the ICP was further elevated above this pressure range, the perfusion pressure suddenly decreased and blood stasis in the cerebral vessels and a change of cerebrovascular tone to vasoparalytic state developed. According to their results, the cerebral blood flow would be maintained under the epidural pressure ranging from 40 to 70 mmHg, which was also observed in our Group two patients. Unless the process leading to the vasoparalytic state could be prevented by performing external decompression as in our Group two patients, the ICP would continue to increase above this pressure range and finally reach the MABP level as observed in our Group one patients. It should be emphasized, therefore, that the indication of external decompressive hemicraniectomy should be restricted to cases similar to our Group two patients.
The third pattern of postoperative ICP change as in Group three did not show such high elevation. The preoperative level of consciousness of patients in this group was semicona or coma without any signs of late stage transtentorial herniation. Cerebral angiograms revealed delay of circulation time, but complete filling of small arteries in an early arterial phase. Cerebral contusion without brain swelling and responsiveness of the brain to intravenous administration of Mannitol or hyperventilation were clearly noted at operation. There was no positive indication of external decompressive hemicraniectomy in these cases.

References