Contralateral Subdural Effusion Secondary to Decompressive Craniectomy Performed in Patients with Severe Traumatic Brain Injury: Incidence, Clinical Presentations, Treatment and Outcome

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Key Words

Traumatic brain injury · Decompressive craniectomy · Subdural effusion, complication, incidence rate

Abstract

Objective: This study was performed to better understand postoperative contralateral subdural effusion, an uncommon but serious complication secondary to decompressive craniectomy in patients with head trauma. **Subjects and Methods:** Data from medical records of 169 patients who underwent decompressive craniectomy after head trauma between 2003 and 2006 were collected. The data included demographics, clinical presentations, treatment and outcome. **Results:** Of the 169 patients, 11 (6.5%) had contralateral subdural effusion. On the average, this complication was found 14 days after decompressive craniectomy. Of the 11 patients, conservative treatment was effective in 7 with a gradual resolution which lasted 52.7 days on average. The effusion in the remaining 4 patients led to progressive deterioration of clinical presentation, and surgical intervention was necessary: subduroperitoneal shunting in 3 cases and burr hole drainage in the remaining 1 case. **Conclusions:** Our findings confirmed that postoperative contralateral subdural effusion was not an uncommon complication secondary to decompressive craniectomy. Most contralateral subdural

effusions resolved spontaneously after conservative management, but surgical management may be necessary if the patients develop deteriorating clinical manifestations or the subdural effusion has an apparent mass effect.

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Introduction

Refractory intracranial hypertension, resulting from severe traumatic brain injury (STBI), is one of the leading causes of death in patients with head trauma and always poses a great challenge to the neurosurgeon. Although there is no conclusive evidence on whether or not decompressive craniectomy has a beneficial effect, the procedure is widely performed as a last resort when patients with STBI develop uncontrolled intracranial pressure (ICP) [1–3]. Decompressive craniectomy involves removal of a large bone flap above the affected brain hemisphere and simultaneous dura opening to create more space for the swelling brain [4]. A simultaneous decompressive duraplasty, a technically simple procedure with low incidence of complications, is recommended in some institutions [5, 6]. In this article, we report a group of cases who developed contralateral subdural effusion after decompressive craniectomy in patients with traumatic brain in-

Fig. 1. In this 69-year-old patient, a right subdural effusion contralateral to the cranial defect was discovered a week after decompressive craniectomy. The subdural effusion was managed conservatively and was gradually reabsorbed spontaneously.

Fig. 2. In this 49-year-old patient, a left subdural effusion with manifest clinical presentations and marked brain midline shift was discovered 40 days after surgical decompression, and the patient became unconscious again. Consequently, a subduroperitoneal shunt was performed for this contralateral subdural effusion.

jury. To our knowledge, this is the first report on a group of patients with head trauma who developed contralateral subdural effusion after decompressive craniectomy, and we hope that this report will be helpful for the understanding and management of this complication.

Subjects and Methods

From 2003 to 2006, a total of 169 patients underwent decompressive craniectomy for elevated ICP with or without removal of a clot or contusion after head trauma at the Institute of Brain Medicine, Zhejiang University, PR China. Of these, 11 patients had the complication of contralateral subdural effusion secondary to decompressive craniectomy. The medical records of these patients including the demographics, clinical presentations, treatment and outcome were collected and analyzed.

Results

Among the 169 patients who underwent decompressive craniectomy after head trauma, 11 (6.5%) had contralateral subdural effusion. The information about the 11 patients is given in table 1. The average age of the patients was 47.4 years (range 9–70 years); 9 were male and 2 female. Traumatic subarachnoid hemorrhage was found in 8 (72.7%) patients. In addition, the average time from the procedure of decompressive craniectomy to the onset of contralateral subdural effusion was 14 days (range 1–40 days).

After decompressive craniectomy, 10 patients showed improvements in clinical presentations. The remaining 1 patient who suffered from severe head trauma with an initial Glasgow Coma Scale score of 4 died 30 days after the operation (case No. 7 in table 1). A deterioration of clinical presentation or onset of new symptoms/signs re-

lated to subdural effusion was seen in 8 cases. The new symptoms included headache ($n = 5$), vomiting ($n = 3$), worsening of conscious state ($n = 3$), paralysis ($n = 1$) and convulsion ($n = 1$). Prompt computer tomography (CT) scan revealed a contralateral subdural effusion. As for the remaining 3 patients without apparent worsening of clinical manifestations, the contralateral subdural effusion was discovered by a conventional CT scan after surgical decompression.

The contralateral effusion resolved gradually under conservative management in most of the patients (7/11; fig. 1); the average time from the onset of contralateral subdural effusion to resolution was 52.7 days (range 16–151 days) based on the CT image. However, the contralateral subdural effusion in the remaining 4 patients contributed to progressive clinical deterioration (fig. 2), and a surgical procedure was performed (subduroperitoneal shunting for 3 cases, and burr hole drainage for the remaining 1).

The outcome for these 11 patients is also shown in table 1. The prognosis was based on a Glasgow Outcome Scale score which was assessed 6 months after the head trauma.

Discussion

Decompressive craniectomy is now considered as an important procedure for the management of refractory intracranial hypertension, and over recent decades, a number of studies have shown that this procedure is effective in decreasing ICP and preventing transtentorial herniation after STBI [7, 8]. Generally, decompressive craniectomy is a technically simple procedure with few surgical complications [4, 9–11]. Tabaddor and LaMor-

Table 1. Patients developing contralateral subdural effusion after decompressive craniectomy performed for severe head trauma

No.	Sex/age, years	GCS score	tSAH	Dura-plasty	Symptoms and signs	Time days	Outcomes
1	M/52	4	Yes	No	The patient remained unconscious after DC and did not respond to his surroundings; no deterioration of clinical condition was found	29	Conservative management was performed, and the subdural effusion resolved 22 days after onset; GOS 2
2	M/56	7	Yes	Yes	The patient became conscious 16 days after DC but he complained of progressive deterioration with headache and frequent vomiting 3 days after his recovery of consciousness	20	Conservative management was performed, and the subdural effusion resolved 26 days after onset; GOS 3
3	M/49	6	Yes	No	The patient was awake and alert 30 days after DC, but the conscious state worsened gradually and he lost consciousness again	40	Subduroperitoneal shunting was performed, and the subdural effusion disappeared after the surgery; GOS 3
4	F/70	6	No	Yes	The patient was awake and alert 2 days after DC, but the symptom of headache remained and worsened	6	Conservative management was performed, and the subdural effusion disappeared 31 days after onset; GOS 3
5	M/48	7	No	Yes	The patient was awake and alert 1 day after DC, but headache and vomiting worsened, and an upper-extremity paralysis developed	3	Subduroperitoneal shunting was performed, and the subdural effusion disappeared after the surgery; GOS 4
6	F/29	6	Yes	Yes	The patient was lethargic after DC; worsening of the conscious state was discovered 5 days later	5	Drainage of the effusion with a burr hole was performed, and the subdural effusion disappeared after the surgery; GOS 3
7	M/9	4	Yes	Yes	The patient remained comatose after DC; he developed pneumonia and systemic infection response syndrome, and died 30 days after surgical decompression	7	Conservative management was performed, and the subdural effusion resolved 16 days after onset; GOS 1
8	M/37	5	No	Yes	The patient regained consciousness on the day when DC was performed, but he complained of progressively worsening headache and vomited frequently	1	Conservative management was performed, and the subdural effusion resolved 51 days after onset; GOS 4
9	M/69	9	Yes	Yes	The patient was awake and alert 3 days after the first procedure, but he still complained of persistent headache	7	Conservative management was performed, and the subdural effusion resolved 151 days after onset; GOS 4
10	M/42	5	Yes	Yes	The patient remained unconscious after DC but responded to pain stimuli; the clinical condition improved, but 2 weeks later his conscious state began to worsen and convulsions developed	15	Subduroperitoneal shunting was performed, and the subdural effusion disappeared after the surgery; GOS 2
11	M/60	4	Yes	Yes	The clinical condition improved after DC, and no worsening was found	21	Conservative management was performed, and the subdural effusion resolved 72 days after onset; GOS 3

GCS = Glasgow Coma Scale; tSAH = traumatic subarachnoid hemorrhage; DC = decompressive craniectomy; time = the time ranging from surgical decompression to discovering contralateral subarachnoid effusion; GOS = Glasgow Outcome Scale, the score of which was calculated 6 months after head trauma except for the patient who died within a month after trauma.

gesse [9] reported harmful effects of large craniectomy on brain functions, suggesting early cranioplasty; Andre et al. [10] reported a temporal muscle hematoma. Wagner et al. [11] reported an unexpectedly high occurrence of direct craniectomy-related parenchymal bleeding (41.6%) and ischemic lesions (28.4%). They concluded that the size and shape of the edges of the craniectomy flap were important factors related to a decompression effect and postoperative complications, and that flaps too small in diameter may cause additional hemorrhagic or ischemic lesions. Likewise, sharp bone edges were found to be associated with craniectomy-related lesions.

Subdural effusion is a well-known complication of craniotomy; however, development of contralateral subdural effusion has rarely been reported previously, and it is believed to be an infrequent complication of surgical decompression in patients with severe head trauma [4]. A number of possible mechanisms could be responsible for the development of contralateral subdural effusions: (a) the rapid decrease in the ICP as well as outward herniation would form a pressure gradient between the two hemispheres and lead to the enlargement of the contralateral subdural space and the accumulation of effusion, especially when initially there is a possible rupture in the arachnoid layer after head trauma; in our experience, the more notable the shift of the brain after decompressive craniectomy, the more likely the contralateral subdural effusion (fig. 2); (b) the disturbance of the cerebrospinal fluid (CSF) circulation, especially the problem of absorption, would increase the risk of accumulation of the effusion through the torn portion of the arachnoid layer [12, 13], but the subarachnoid hemorrhage would inhibit the absorption of CSF as was discovered in 8 of our 11 patients; (c) shrinkage of the ipsilateral brain due to intraoperative tissue retraction and inability to reshape would cause a pressure gradient between the two hemispheres and lead to brain shift as well, and play an important role in the enlargement of the contralateral subdural space as well as the accumulation of subdural effusions [4].

Most traumatic subdural effusions were considered as 'clinically silent' [14]; however, most patients (8 of 11) in this series did present an apparent deterioration in clinical manifestations. A risk of delayed or missed diagnosis may exist for the patients without apparent clinical symptoms or signs, especially for those who were in a coma or were intubated. It should be noted that the onset of contralateral subdural effusion could be delayed for even more than 1 month after surgical decompression and could still cause clinical deterioration (as case No. 3 in our study; table 1).

Prevention of contralateral subdural effusion could be effected by decompressive duraplasty or bandaging. Decompressive duroplasty can decrease the influence of CSF circulation and brain shift as was also reported by Yang et al. [13]. After the peak time of cerebral edema, bandaging the cranium may help to prevent cephalocele and brain shift before the performance of cranioplasty.

Suggested methods for the treatment of subdural effusion include conservative management with close observation [15, 16] as was done for 7 of our cases, subdural drainage [17] as was done in 1, subduroperitoneal shunt [18] as was done in the remaining 3 and removal of the subdural effusion by craniotomy [19]. Conservative management is recommended when the effusion is not a large quantity of fluid and when there is no manifest brain shift and deterioration of clinical presentation. However, if signs such as obvious compression of a cerebral ventricle and cistern as well as shift of the midline occur, or the effusion becomes large, surgery should be performed as soon as possible. The separation of the dura and arachnoid membrane caused by the development of subdural effusion could lead to the formation of a new membrane with newly formed vessels, which would result in repeated microhemorrhage and contribute to chronic subdural effusion or hematoma. Apparently, long-range compression and the stimulus of effusion or hematoma are disadvantageous for the brain tissue as well as for the recovery of neural function after head trauma.

Drainage with a burr hole was performed in 1 patient, which was considered as a simple and effective procedure for subdural effusion. However, subduroperitoneal shunting carries the risk of failure when the fluid contains blood. So it should be considered with caution when the effusion has a high density in the CT image, and burr hole drainage may be performed first to let the fluid become clear. In our experience, drainage with a burr hole is technically easier with obviously a smaller surgical trauma. However, it may not work in some conditions, especially when the subdural effusion is combined with a contralateral large cranial defect and a possible pressure gradient between hemispheres that would contribute to reaccumulation of CSF [4]. A subduroperitoneal shunt is suitable for the subdural effusion with a large quantity of fluid. When the subdural effusion increases progressively (suggesting the existence of valves), this method may be the best choice. In addition, Kilincer et al. [4] reported that they performed cranioplasty and subduroperitoneal shunting simultaneously and attained a satisfactory resolution of the effusion which had developed after aneurysm surgery and decompressive craniectomy. The meth-

od of performing shunting plus cranioplasty could resolve the problems of subdural effusion and of the large cranial defect at the same time, and the cranioplasty which would resolve the outward herniation and pressure gradient was also helpful for the resolution of contralateral subdural effusion. However, there is a risk of infection because of the simultaneous performance of two operations. Removal of the subdural effusion with craniotomy is another effective method, but with a larger surgical trauma and a longer surgical time. Craniotomy after surgical decompression on the contralateral side should be considered carefully before adopted. We considered the craniotomy as a last resort, and it was not performed in this group of patients.

Conclusion

Our findings confirmed that postoperative contralateral subdural effusion was not an uncommon complication secondary to decompressive craniectomy. Most of the contralateral subdural effusions resolved spontaneously with conservative management, but surgery may be necessary if the patients develop deteriorating clinical manifestations or the subdural effusion has an apparent mass effect.

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