Large cranial defects can result from decompressive craniectomy performed for rapid relief of intractable intracranial hypertension. Decompressive craniectomy reduces the risk of death in patients experiencing severe brain edema. However, patients with large cranial defects may experience complications, including sinking flap syndrome and syndrome of the trephined. A large cranial defect is one of the indications for cranioplasty. According to prior practice, this procedure is commonly performed 3–6 months after craniectomy because of infection risks or unresolved brain swelling. Recently, the purpose of cranioplasty has changed, from cosmetic or protective effects to therapeutic effects. We performed early cranioplasty in an effort to diminish complications from large cranial defects. The aim of this study was to assess the safety and efficacy of early cranioplasty after decompressive craniectomy. Considering the hospitalization periods and complications that proceed from large cranial defects, another goal of this study is the prompt reintegration of these patients into their normal living environments.

Materials and Methods

From January 2009 to December 2010, decompressive craniectomies were performed on 82 patients in our de-
department. Of these patients, 40 patients were excluded for the following reasons: 15 patients due to death or follow-up loss, 7 patients due to decompressive craniectomy for malignant infarction, and 18 patients due to subarachnoid hemorrhage. Consequently, 42 patients with traumatic brain injury underwent cranioplasty in our department. Because 6 patients underwent decompressive craniectomy at other hospitals, a total of 36 patients were enrolled in this study. Group I included 15 patients who underwent early cranioplasty within 6 weeks. Group II included 21 patients who underwent delayed cranioplasty 6 weeks after decompressive craniectomy.

Data on all patients were gathered upon enrollment. In all patients, brain computed tomographic (CT) scans were performed for evaluation of changes in brain swelling, fluid collection, and ventricle dilatation. In addition, laboratory results, including white blood cell counts, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) levels were assessed in order to identify infections. We also gauged on whether or not patients underwent duraplasty with artificial dura during decompressive craniectomy. All patients, except one, underwent decompressive craniectomy using artificial dura. Postoperative fluid collection was monitored with brain CT scans. All cranioplasties were performed using an autologous bone flap and, titanium mesh plates or clamp systems. The autologous bone flap was frozen and stored at −78°C. Sites of marginal bone defects, caused by the autologous bone graft, were reconstructed with polymethylmethacrylate (PMMA) in some patients.

Procedure outcomes were evaluated one month after cranioplasty using the Barthel index of activity of daily living (ADL). To evaluate the safety of early cranioplasty, we compared the ratio of infection, subdural fluid collection, and ventricle dilatation in the early cranioplasty group (Group I) and the delayed cranioplasty group (Group II).

Results

Thirty-six patients (28 males, 8 females) who underwent cranioplasty after decompressive craniectomy for traumatic brain injury were included in this study; 21 patients had suffered acute subdural hemorrhage and 15 traumatic intraparenchymal hemorrhage. The mean age of all patients was 52.06 ± 15.09 (range, 19–75) years. Mean periods between decompressive craniectomy and cranioplasty of Groups I and II were 35.20 ± 3.76 (29–42) and 62.95 ± 14.82 (44–102) days (Table 1).

Differences in preoperative GCS score between Groups I and II were not statistically significant (p=0.759). Mean Barthel indices of ADL approximately one month after cranioplasty in Groups I and II were 65.67 ± 5.30 (55–75) and 47.86 ± 10.67 (30–75). Differences between the two groups were statistically significant (p<0.05)(Table 2).

Of the 11 patients with subdural fluid collection before cranioplasty, fluid collection disappeared in 10 on postoperative brain CT scan. Newly developed subdural fluid collection was found in 3 patients (Group I: 1 patient, Group II: 2 patients); however, this disappeared within one month. In the 3 patients with dilatation of the lateral ven-

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**TABLE 1.** Characteristics of the early cranioplasty group (I) and the delayed cranioplasty group (II)

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>15</td>
<td>21</td>
</tr>
<tr>
<td>Age (years)*</td>
<td>51.40 ± 14.97 (19–69)</td>
<td>52.52 ± 15.52 (19–75)</td>
</tr>
<tr>
<td>Duration (days)*</td>
<td>35.20 ± 3.76 (29–42)</td>
<td>62.95 ± 14.82 (44–102)</td>
</tr>
<tr>
<td>Sex ratio (M : F)</td>
<td>13 : 2</td>
<td>15 : 6</td>
</tr>
<tr>
<td>Preoperative GCS score*</td>
<td>8.87 ± 4.02 (3–15)</td>
<td>8.38 ± 5.03 (3–14)</td>
</tr>
<tr>
<td>Duraplasty</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Fixation materials (Plate : Clamp)</td>
<td>9 : 6</td>
<td>16 : 5</td>
</tr>
<tr>
<td>Usage of PMMA</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

*values are reported as the mean ± standard deviation (range)

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**TABLE 2.** Outcome and complications in both groups

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barthel index* (mean ± SD, range)</td>
<td>65.67 ± 5.30 (55–75)</td>
<td>47.86 ± 10.67 (30–75)</td>
</tr>
<tr>
<td>Infection</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Subdural fluid collection</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Ventricleomegaly</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05: unpaired t-test; significant difference between group I and group II; SD: standard deviation
tricle underlying the cranial defect after decompressive craniectomy, ventricular dilatation was not aggravated in the follow-up period.

None of our patients presented with symptoms or signs of infection, such as fever and elevated level of laboratory findings, including leukocyte, ESR, and CRP during the postoperative one month follow-up period.

**Discussion**

Decompressive craniectomy is one of the most important methods for management of refractory intracranial hypertension after severe traumatic brain injury. However, the value of decompressive craniectomy in achieving a better outcome remains controversial and certain complications may follow the operation.\(^7,14\)

In 1945, Gardner reported on a syndrome characterized by severe headache, dizziness, undue fatigability, poor memory, irritability, epilepsy, discomfort, and psychiatric symptoms observed in patients with large cranial defects, which he called “syndrome of the trephined”. Occurrence of the syndrome of the trephined is frequent after a large craniectomy and is a well-known indication for cranioplasty. It is believed to be related to atmospheric pressure transmitted through the unsupported scalp; it was also called “syndrome of the sinking skin flap” by Yamaura and Makino in 1977.\(^6\) After cranioplasty, symptoms related to this syndrome can be relieved to different degrees in some patients; however, the procedure is not always reliably effective.

In addition, the large craniectomy would also lead to significant changes in the dynamics of local cerebral blood flow. In the early phases after decompressive craniectomy, perfusion of brain tissue underlying the cranial defect increases with reduced ICP; however, soon after the process, the sinking skin flap transmits atmospheric pressure to the underlying brain tissue and lead to a low cortical perfusion, compared with the contralateral brain, as well as a disturbance of venous drainage, which would be partially rectified after the cranioplasty.\(^7,14\)

The hydrodynamic information about cerebrospinal fluid (CSF) before and after cranioplasty have been studied.\(^6\) Changes in CSF hydrodynamics after large craniectomy lead to dilatation and shift of the ipsilateral lateral ventricle, hydrocephalus, and subdural collections. Cranioplasty is helpful for the correction of these disturbances as well as partial relief of symptoms.\(^2\)

Winkler et al.\(^11\) demonstrated that chronic decompressive craniectomy impairs not only postural blood flow regulation in the ipsilateral hemisphere, but also cerebrovascular reserve capacity in the brain as a whole. Cranioplasty improves both postural blood flow regulation and cerebrovascular reserve capacity. Therefore, cranioplasty resulted in marked improvement of metabolic activity.

At present, cranioplasty may be performed not only for cosmetic reasons, but also for its therapeutic effects, particularly for patients with huge cranial defects after decompressive craniectomy.\(^7,10\) Commonly, performance of cranioplasty 3 months after craniectomy is recommended, and if the patient has a history of intracranial infection or open craniocerebral injury, the procedure can delayed for at least 6 months after the first surgery. However, some authors have advanced the idea of early cranioplasty after decompressive craniectomy to alleviate complications from craniectomy.\(^4,8\) Some authors reported that early cranioplasty provides a satisfactory securing dissection plane during operative procedures, compared with later cranioplasty, without causing additional complications, including infection, subdural hygroma, and brain parenchymal damage, in selected cases.\(^4,12\) Liang et al. reported that early cranioplasty was safe and assisted in improvement of patient’s neurological function and prognosis. In addition, early cranioplasty has an advantage in dissection for cranioplasty.\(^8\) Early cranioplasty performed before massive scar formation reduces operative time by facilitating soft tissue dissection. Beauchamp et al. suggested that early cranioplasty would lower the overall cost of care by eliminating the need for additional hospital admissions.\(^1\)

In this study, early cranioplasty was effective in improving ADL of patients. The Barthel index of ADL was found to be significantly higher in the early cranioplasty group. In addition, early cranioplasty does not increase relative risk of complications, such as infection or fluid collection. In addition, fixation materials and usage of bone cement (PMMA) have no effect on the rate of cranioplasty infection.\(^1\)

The limitations of our study included its retrospective nature, small sample size, and lack of long-term follow-up data. A prospective, randomized, controlled study with extended follow-up duration will be needed to fully establish efficacy of early cranioplasty.

**Conclusion**

We consider that with appropriate selection of patients, early cranioplasty for large cranial defects after decompressive craniectomy will be a safe and helpful strategy for improvement of the neurologic function of patients.
with severe traumatic brain injury.

■ The authors have no financial conflicts of interest.

REFERENCES