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Klinik Çalışma

Value of early unilateral decompressive craniectomy in patients with severe traumatic brain injury

Ciddi travmatik beyin yaralanmalı olgularda erken tek taraflı dekompresif kranyektominin yeri

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BACKGROUND
The aim of our study was to evaluate the results and effectiveness of early decompressive craniectomy in the treatment of severe traumatic brain injury.

METHODS
We conducted a prospective study to investigate the clinical and radiological results of early unilateral decompressive craniectomy in 33 patients with severe traumatic brain injury. The mean area of the craniectomy, potential expansion volume of the decompressed brain, and distance between the lower border of the craniectomy and the temporal cranial base were calculated from computed tomography scans. Clinical results were analyzed with modified Rankin Scale (mRS).

RESULTS
Time to surgery after trauma was 3.1±1.9 hours. There was a direct proportionality correlation between the area of the craniectomy and the calculated volume ($p<0.0001$). There was also a significant correlation between the state of the mesencephalic cisterns after craniectomy and the distance of the craniectomy to the base of the cranium ($p<0.01$). Assessment of overall one-year clinical outcome demonstrated favorable outcome (mRS 0-3) in 48.5% of patients.

CONCLUSION
The high overall morbidity and mortality rates demonstrated in our group despite the performance of early decompressive procedures reflect the severity of the underlying injuries.

Key Words: Decompressive craniectomy; traumatic brain injury/ outcome.

AMAÇ
Travmatik ciddi beyin yaralanması tedavisinde erken yapılan dekompresif kranyektominin sonuçlarını ve etkinliğini değerlendirmektir.

GEREÇ VE YÖNTEM
Travmatik ciddi beyin yaralanması olan 33 olguda, erken yapılan tek taraflı dekompresif kranyektominin klinik ve radyolojik sonuçlarını araştırmak için prospektif bir çalışma yapıldı. Ortalama kranyektomi alanı, dekomprese edilmiş beynin potansiyel genişleme hacmi ve kranyektominin alt sınırı ile temporal kraniyal taban arasındaki mesafe bilgisayarlı tomografilerden hesaplandı. Klinik sonuçlar modifiye Rankin skalası (mRS) ile değerlendirildi.

BULGULAR
Travma ile cerrahi girişim arasındaki zaman 3,1±1,9 saatti. Kranyektomi alanı ile hesaplanan hacim arasında doğrudan orantılı korelasyon vardı ($p<0,0001$). Ayrıca, kranyektomi sonrası mezensefalik sisternaların durumu ile kranyektominin kraniyal tabana olan uzaklığı arasında da anlamlı korelasyon vardı ($p<0,01$). Bir yıllık klinik sonuçların değerlendirmesi olguların %48,5'inde olumlu sonuç sağladı (mRS 0-3).

SONUÇ
Gerçek anlamda erken dekompresyon uygulanmasına rağmen, olgularımızda yüksek oranda morbidite ve mortalite görülmesi altta yatan yaralanmanın ciddiyetini yansıtmaktadır.

Anahtar Sözcükler: Dekompresif kranyektomi; travmatik beyin yaralanması/sonuç.

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Severe traumatic brain injury (TBI) is often accompanied by early death due to transtentorial herniation. Neurosurgical therapy aims to minimize the secondary brain damage after a severe head injury. Restoration of cerebral perfusion by surgical enlargement of the intracranial space is the primary goal of decompression. When a significant mass lesion is present, the role of emergency surgical evacuation and decompression is obvious. However, the management of cases in which severe TBI is coupled with refractory intracranial hypertension in the absence of a mass lesion is more controversial. On the other hand, decompressive craniectomy may be the only method available in developing countries with limited intensive care unit, monitoring resources, and qualified personnel.

The purpose of this study was to evaluate the value of early decompression by correlating the values of parameters such as distance of the lower border of the craniectomy to the temporal cranial base, calculated area of the craniectomy and gain of space for the swelling brain, and relationship between those values and clinical outcome in patients who underwent decompressive craniectomy for severe TBI at a single institution.

MATERIALS AND METHODS

Patient Population

This prospective observational study was undertaken for analysis of 33 patients with severe TBI, who underwent early decompressive craniectomy between August 2004 and August 2007. By referring to their clinical and neuroimaging records, the following information was documented for each patient: sex, age, intracranial pathology, concurrent injuries, clinical status on admission, time to surgery after trauma, pre- and postoperative midline shift, the state of the ventricles and perimesencephalic cisterns, distance of the lower border of the craniectomy to the temporal cranial base, calculated area of the craniectomy and gain of space for the swelling brain, hospitalization time, and early and late neurological outcome.

Severe TBI was identified by a Glasgow Coma Scale (GCS) score of <8 after initial resuscitation. All patients underwent axial cranial computed tomography (CT) after initial airway and hemodynamic management.

An early decompressive craniectomy was performed within the first eight hours in patients according to the following criteria: a GCS score <8 and the existence of clinical signs of cerebral herniation (absence of pupillary reflexes), correlated with abnormalities in CT scan including hematoma, appearance of diffuse or unilateral brain swelling, and/or cerebral herniation. The intracranial pressure (ICP) in these patients could not be measured before the decompressive craniectomy was performed because of limited sickbeds in the intensive care unit, ICP monitoring resources, and qualified personnel in our hospital. Decompressive craniectomy was not performed in patients who had primary fatal brainstem damage, bilateral dilated and fixed pupils, or life-threatening concomitant medical disease.

Surgical Procedures and Follow-Up Examinations

In cases in which surgery was performed to evacuate a mass lesion, a wide unilateral hemicraniectomy was performed, including removal of bone in the frontal, temporal, and parietal regions. Special care was taken to remove the temporal bone down to the base of the cranium. The dura mater was opened widely to the extent of bone decompression with an eventual emptying of a large extra-axial collection. Subsequently, a dural graft was placed to increase the available volume before closure. The bone flap was placed in a subcutaneous abdominal pocket until subsequent cranioplasty. When surgery was performed to treat diffuse or unilateral cerebral edema without a mass lesion, a similar wide craniectomy was performed, ipsilateral to the hemisphere in which the greatest swelling was observed on CT scans. Then duraplasty was performed, and the bone flap was not replaced.

Computed tomography scans were obtained at admission and about 24 hours after surgery. Subsequent scans were performed almost weekly or if new symptoms developed or symptoms worsened (Figs. 1, 2).

For data recording purposes, using the septum pel-

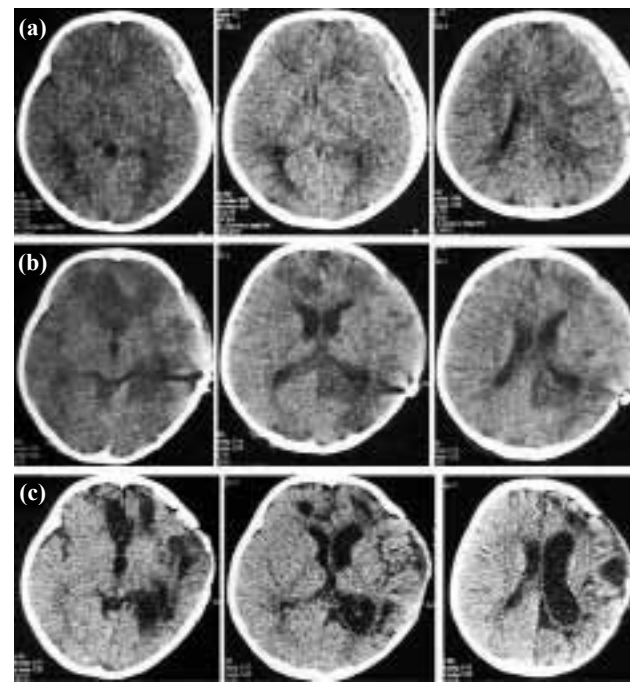

Fig. 1. Preoperative **(a)**, 24-hour **(b)** and 10-day **(c)** CT scans obtained in a 4-year-old female with TBI undergoing early unilateral decompressive craniectomy as the primary therapy.

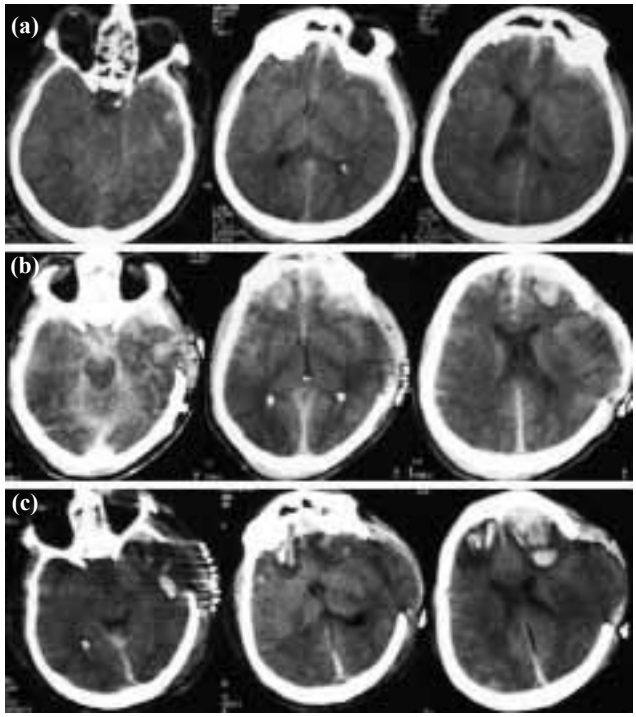

Fig. 2. Preoperative **(a)**, 24-hour **(b)** and one-week **(c)** CT scans obtained in a 32-year-old male with TBI undergoing early unilateral decompressive craniectomy as the primary therapy.

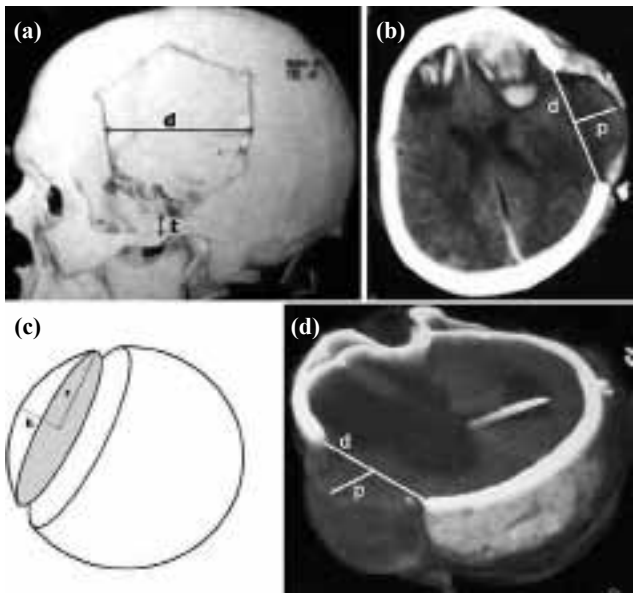

Fig. 3. (a) CT reconstruction of a skull after surgery shows craniectomy area; **(b)** CT scan of a brain after craniectomy shows expanded brain tissue; **(c)** Figure symbolizes spherical segment above the craniectomy; **(d)** CT reconstruction of a brain after surgery shows expanded brain tissue as a spherical segment above the craniectomy. *d* = diameter of the craniectomy, *h* = height of the perpendicular line (p), *p* = perpendicular line with the longest distance from the diameter (d) to the dural flap, *r* = radius of the hypothetical circle (d/2), *t* = distance of the craniectomy to the temporal cranial base.

lucidum as the midline structure, the largest extent of the midline shift was measured. The distance (t) between the lower border of the craniectomy and the temporal cranial base was documented. The changes in the qualitative CT features (the visibility of the ventricles and perimesencephalic cisterns) after decompressive craniectomy were examined and assessed as improved, unchanged, or deteriorated. The mean area of the craniectomy was calculated via a previously established formula as follows:[1] The diameters (d) of the craniectomy were measured in millimeters for each CT scan slice. The CT slice with the largest diameter was then taken, and the perpendicular line (p), the longest distance from the horizontal diameter to the dural flap, was measured (Fig. 3).

The area of the craniectomy (a) was determined by integrating the diameter (d) of each CT scan slice of a craniectomy and multiplying the obtained values by the slice thickness.

The volume above the craniectomy was calculated as follows: A hypothetical circle was assumed and therefore transformed the formula

$$A_{circle} = \pi \cdot r^2$$

to

$$r = \sqrt{A_{circle} / \pi}$$

where A_{circle} is the calculated area of the craniectomy and r represents the radius of the hypothetical circle. The height of the perpendicular line to the dural flap (p) was used to calculate the volume (V) as the spherical segment above the craniectomy by the formula

$$V = \frac{h^2 \cdot \pi}{3} \cdot (3r - h)$$

where h represented the height of the perpendicular line (p) and r represented the radius of the hypothetical circle.

Surviving patients were followed up regularly at intervals of 1, 3, 6, and 12 months. The outcomes of surgery in the long-term follow-up were assessed for all surviving patients according to the criteria used by the modified Rankin Scale (mRS). Favorable outcome was defined as mRS of 0, 1, 2 and 3 (good or slight to moderate neurological disability). Poor outcome was defined as mRS of 4, 5 or 6 (moderate severe to severe neurological disability or death) determined at the late postoperative clinic visits.

Statistical Analysis

Statistical calculations were performed using the GraphPad Prism V.3 program for Windows. Besides standard descriptive statistical calculations (mean ± standard deviation), one-way analysis of variance was

used in the comparison of groups, the post hoc Newman-Keuls multiple comparison test was used to compare subgroups, and the chi-square test was performed during the evaluation of qualitative data. Statistical significance was established at a probability value of <0.05.

RESULTS

Of these 33 patients, 25 were males (76%) and 8 were females (24%); their mean age (± SD) was 34.42±22.09 (range, 4-80 years). During the study period, 12246 patients were examined by the emergency service, 5414 of whom suffered some degree of head injury and 419 of whom required a craniotomy. Of the patients in whom a craniotomy was performed for trauma, 25 patients underwent a decompressive craniectomy in conjunction with removal of a mass lesion and 8 patients underwent a decompressive craniectomy for diffuse or unilateral brain edema. Patients suffered various forms of trauma, including motor vehicle accidents, motor vehicle-pedestrian accidents, falls, assaults, and bicycle accidents. All patients had various volumes of acute subdural hematoma with or without other intracranial pathologies. The concurrent injuries included pneumothoraces, hemothoraces, pulmonary contusions, splenic injury, liver lacerations, and orthopedic injuries. The initial mean GCS score was 5 points. Time to surgery after trauma was 3.1±1.9 hours (median, 2.5 hours).

Radiographic Analysis

The average area of craniectomy was 70.9±15.8 cm^2 (median, 67.9 cm^2; range, 51.5-113 cm^2). The mean distance between the lower border of the craniectomy and the temporal cranial base was 17.9±8.4 mm (median, 17 mm; range, 7-35 mm). The average distance from the horizontal diameter to the dural flap (perpendicular line) was 27.8±5.4 mm (median, 28.5 mm; range, 14-38 mm). The calculated mean volume (as spherical segment above the craniectomy) was 69.1±16.4 cm^3 (median, 67.5 cm^3; range, 46.1-107.2 cm^3).

There was a direct proportionality correlation between the area of the craniectomy and the distance to the dural flap ($p<0.001$) and between the area of the craniectomy and the calculated volume ($p<0.0001$).

The average of the midline shift before surgery was 20.6±5.5 mm (median, 20 mm; range, 13-31 mm). The average decrease in the midline shift after decompression was -17.3± -5 mm (median, -17 mm; range, -10 to -28 mm). There was a significant improvement in the degree of midline shift ($p<0.0001$). There was no correlation between changes in midline shift and the size of the craniectomy ($p>0.05$).

The visibility of the ventricles did not demonstrate a significant improvement ($p>0.05$). The visibility of the perimesencephalic cisterns improved significantly after craniectomy ($p<0.001$). Decreases in the midline shifts correlated significantly with an improvement in the visibility of the perimesencephalic cisterns ($p<0.01$). There was also a significant correlation between the state of the mesencephalic cisterns after craniectomy and the distance of the craniectomy to the base of the cranium ($p<0.01$).

Clinical Analysis

The mean hospitalization time was 18.4±6.7 days for surviving patients (median, 17.5 days; range, 9-43 days). At the time of discharge, 45.5% of the 33 patients had a favorable outcome, and 54.5% of patients had a poor outcome. Nine patients (27.3%) died, including four of the eight patients who underwent decompressive surgery for cerebral edema in the absence of a mass lesion. Of the 24 survivors, five patients (20.8%) required a tracheostomy and nine (37.5%) required a gastrostomy. Posttraumatic shunt-dependent hydrocephalus developed in seven (21.2%) of the surviving patients. Posttraumatic epilepsy requiring antiepileptic agents developed in 14 (42.4%) of the patients. In all 24 survivors, the patient's own bone was replaced once the craniectomy site was soft and the patient's long-term survival was apparent. The mean time to replacement of the bone was 2.5 months.

The average follow-up duration was 14 months (range, 10-19 months). At the late postoperative visits, while the neurological state had minimally improved, only 48.5% had a favorable outcome and 51.5% of the patients had a poor outcome. Our series showed that patient age and initial neurological condition are correlated with the clinical outcome ($p<0.001$). The outcome of patients younger than 50 years or with an initial GCS score ≥5 was significantly better than that of older patients or those with an initial GCS score <5.

DISCUSSION

Severe TBI is associated with a high mortality and morbidity. Management of TBI aims to minimize the secondary brain damage by maintaining cerebral perfusion pressure (CPP) and reducing ICP. The exact role, timing and efficacy of decompressive craniectomy remain controversial.[1-5] Guidelines recommend monitoring ICP in patients with a GCS score ≤8, maintaining ICP <25 mm Hg, and maintaining CPP >70 mm Hg, by using hemodynamic support, sedation, external ventricular drainage of cerebrospinal fluid, mannitol, and moderate hyperventilation. Decompressive craniectomy is often the final option in the management of posttraumatic intractable intracranial hypertension.[6] Because this old neurosurgical procedure is usually the only available method in our country with limited intensive care unit, monitoring resources and qualified personnel, we conducted this study to investigate the changes in the CT features and relationship between

these changes and clinical outcome in patients undergoing a decompressive surgical procedure.

We calculated the area of the craniectomy and gain of space for the swelling brain. Thus, we obtained potential expansion volume of the decompressed brain. Our results showed that the extent of the craniectomy directly correlated with the distance to the dural flap and volume of the brain above the craniectomy. A larger craniectomy proved too much volume for the swollen brain. A study of Eisenberg et al.,[7] based on information from 753 patients, indicated that midline shift, regardless of the underlying pathology, is a very strong predictor of abnormal ICP. They also showed an association between degree of midline shift and risk of dying. Conversely, other studies[2,8,9] found no or only poor correlation between the degree of midline shift and the level of ICP. However, Münch et al.[1] demonstrated a strong correlation between the increase in CPP and the decrease in the midline shift. Even though ICP measurements before the decompression were not available for our patients, we found a significant decrease in the midline shift after craniectomy. Compressed or obliterated mesencephalic cisterns and small or asymmetrical ventricles are thought important because they are an indication of brain swelling and herniation. Several studies[7,10,11] concluded that the state of the mesencephalic cisterns predicts outcome following TBI. Eisenberg et al.[7] also showed that the risk of elevated ICP in patients with abnormal cisterns was increased three-fold compared to patients with normal cisterns. In our series, the state of the perimesencephalic cisterns significantly improved after craniectomy related to the distance of the craniectomy to the base of the cranium. In addition, we found that decreases in the midline shifts correlated significantly with an improvement in the visibility of the perimesencephalic cisterns. The results of our study confirm that a large craniectomy down to the base of the cranium prevents brainstem compression by the temporal lobe and therefore improves patient outcome. According to the data reported by Tabaddor et al.,[9] ventricular compression is a very strong predictor of abnormal ICP. Conversely, Eisenberg et al.[7] demonstrated no correlation between the small or asymmetrical ventricles and ICP or death. We did not obtain a significant improvement in the visibility of the ventricles after decompression.

It has been hypothesized that the damaging cycle of extensive edema caused by elevated ICP, which results in ischemia of neighboring brain tissue and further infarction, may be interrupted by surgical decompression. Experimental studies proved decompressive craniectomy to be the most effective measure in terms of reduction of ICP and survival rate.[12-14] On the other hand, the increase in brain edema after decompressive craniectomy led to a discussion in the neurosurgical literature and to questioning the usefulness of the procedure when treating severely brain-injured patients. Cooper et al.[15] reported a reduction in ICP in experimental cold injury but also observed a clear increase in brain edema after decompression. Hatashita and Hoff[16] showed that decompressive craniectomy associated with arterial hypertension may lead to significant brain edema in cats after experimental brain injury. Studies by the same authors in nontraumatized cats with craniectomies showed a significant reduction in ICP with an increase in compliance in comparison with normal control animals.[17] Yamakami and Yamaura[18] investigated the effect of decompressive craniectomy on regional blood flow in contusions using single-photon emission CT (SPECT) in five patients with severe brain injury. The patients underwent craniectomy when ICP could not be controlled by medical therapy. Within 24 hours of decompression, the SPECT analysis showed a perfusion deficit in all patients that correlated with the contusions. In the region of brain decompression, an area of mild hyperperfusion was seen at the same time. One week after decompression, the region of hyperperfusion had increased in size and intensity. A CT scan obtained at one week revealed massive brain swelling in the decompression area. One month after decompression, no hyperperfusion remained, and a CT scan obtained at that time did not show any brain swelling. Patients showed significant clinical improvement within this period of time. Even within the first postoperative week, while brain swelling was at its maximum, there was no negative effect on the neurological status of the patients. They concluded that the focal cerebral blood flow increase may protect the brain from secondary ischemic cell damage (lactate and potassium clearance).

Burkert and Plaumann[12] demonstrated not only ICP reduction after decompression but also a decrease in intracerebral oxygen tissue pressure (PaO_2) after brain swelling occurred. The change in PaO_2 was even more sensitive to brain swelling than the rise in ICP. After decompression, the PaO_2 increased but only in patients in whom craniectomy was performed early and extensively. When craniectomy was performed in the late phase of ICP elevation and decompression was too limited, the PaO_2 rose but did not reach normal values. According to Guerra et al.,[19] the craniectomy should be performed early, before the severe impairment of brain perfusion occurs, and should yield a wide decompression. Ho et al.[20] investigated cerebral oxygenation, vascular reactivity, and neurochemistry following decompressive craniectomy for severe TBI. Decompressive craniectomy resulted in a significant reduction in the mean ICP and cerebrovascular pressure reactivity index to autoregulatory values in patients. In addition, the durations of abnormal brain

tissue oxygenation were significantly reduced after decompressive craniectomy. However, they obtained poor outcome in 68.7% of the patients.

The cause of the poor results despite the large use of decompression reported in the literature is probably an erroneous indication or timing of the procedure. It is important to perform a sufficiently wide craniectomy with dura opening and duraplasty to achieve sufficient decompression without herniation before irreversible ischemic brain damage occurs. Otherwise, irreversible ischemic damage to the brain exists with no chance of recovery.[19,21-24] There was no indication for decompression craniectomy in our patients with irreversible brainstem signs or herniation, neurological pons signs or fatal primary brain injury. The high overall mortality rate demonstrated in our group (39.4%), despite the fact that rapid decompressive procedures were performed, reflects the severity of the underlying injuries and is in accordance with the data from the Traumatic Coma Data Bank (TCDB) published by Marshall et al.[25] in 1991. The timing of surgery, which ranged from 1 to 8 hours in our study, is a very important factor that may demonstrate the true effect of decompressive craniectomy.

In conclusion, decompressive craniectomy is simple and safe. The procedure leads to the fastest relief by immediate reduction in intracranial hypertension and has the lowest rate of complications. The craniectomy should be performed early, before the severe impairment of brain perfusion occurs, and should yield a wide decompression. Special care should be taken to remove the temporal bone down to the floor of the middle fossa to maximize the extent of decompression at the level of the perimesencephalic cisterns.

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