Using titanium mesh to replace the bone flap during decompressive craniectomy: A medical hypothesis

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A R T I C L E   I N F O

Keywords:
Decompressive bone flap replacement
Decompressive craniectomy
Cranioplasty
Traumatic brain injury
Intracranial hemorrhage

A B S T R A C T

Decompressive craniectomy (DC) plays a significant role in treating refractory intracranial hypertension. During this surgical procedure, part of the skull is removed and the underlying dura mater is open, which can effectively release intracranial pressure. However, in some cases, the decision whether or not to remove the bone flap relies on the surgeon’s personal experience. Positive decisions are usually made to avoid massive postoperative cerebral edema and infarction, which can lead to overtreatment. The procedure is related to many side-effects, which may affect the recovery of neurological function. Patients who have survived have to be anesthetized and undergo secondary cranioplasty 3 or 6 months later. Despite its technical simplicity, complications associated with cranioplasty are hard to ignore. Therefore, there is a need for a new surgical procedure combining decompressive craniectomy and cranioplasty. Acute expansion of the skin flap is limited, and the compensatory capacity of the skull after DC depends on the volume of the bone flap at the early stage. The titanium mesh is thin and strong, does not take up extra space provided by bone flap. Therefore, we put forward the concept of Decompressive Bone Flap Replacement. During this procedure, neurosurgeons resect the massive bone flap, open the dura mater, remove the hematoma in a similar manner to a standard craniotomy and then use titanium mesh shaped appropriately to replace the bone flap. Compared with traditional DC, it can ensure the integrity of the skull without affecting the effect of decompression. This paper presents 2 cases of DC and reviews the literature sustaining our hypothesis.

Introduction

Refractory intracranial hypertension (ICH) can be caused by traumatic brain injury (TBI), or intracranial hemorrhage, intracranial infection or brain tumor, and fails to respond to traditional medical treatments such as dehydration drugs, hyperosmotic drugs, and diuretics. Decompressive craniectomy (DC) is suggested when intracranial pressure fails to respond to medical treatments. The procedure of DC was first described by Kocher in the treatment of TBI by removal of an area of the skull to expand the potential cranial volume. It can effectively release intracranial pressure and increase cerebral perfusion pressure in patients with severe TBI and ICH [1,2]. It consists of 2 major parts: first, the intracerebral hematoma and necrotic brain tissue are removed, and second the bone flap is removed. DC is a life-saving procedure [3,4], which may also lead to a series of complications because of the pathological and physiological changes induced by removing the bone flap; these include skull defect, subdural hygroma, hydrocephalus, cortical herniation, paradoxical herniation, encephalocele, and seizures among others [5,6]. So, whether or not to remove the bone flap is controversial. In some cases, removing the bone flap could be overtreatment. Shimamura (2011) et al thought that DC was not required for rescue in ICH if the hematoma could be removed completely [7]. Tsermoulas (2016) et al found removing the bone flap after acute subdural hematoma evacuation was not associated with better outcome [8]. Yang (2012) et al thought it was important to decide whether or not to perform DC according to the intraoperative findings [9]. Information regarding preoperative midline shift, the initial Glasgow Coma Scale (GCS) score, and presence of intraventricular hemorrhage (IVH) was helpful in deciding if the bone flap should be removed [10]. In clinical practice, the positive decision of removing the bone flap is usually made during surgery to avoid fatal complications like malignant brain swelling and massive cerebral infarction.
After bone flap removal, the secondary cranioplasty can ensure an adequate biomechanical protection for the underlying brain, maintain stable intracranial pressure, and re-establish cerebrospinal fluid dynamics and cerebral blood flow [11,12]. Cranioplasty offers cosmetic and protective benefits for patients after the procedure of DC and contributes to the improvement of neurological functions [13,14]. Autologous bone, hydroxyapatite, polyetheretherketone, polymethylmethacrylate, and titanium are frequently used in the procedure [15]. Despite the technical simplicity of cranioplasty, the procedure can result in many complications, including infection, postoperative hydrocephalus, resorption of the flap, seizure, and postoperative hematoma, which may require surgical treatment [14,16]. In a few cases, massive brain swelling after cranioplasty has been fatal [17]. For elderly people, the risk of anesthesia cannot be ignored, and a new surgical approach to accomplish the decompressive effect and avoid the side-effects following DC is needed.

The hypothesis

Based on the considerations mentioned above, we put forward the concept of Decompressive Bone Flap Replacement (DBFR). During the procedure, the neurosurgeon resects the massive skull bone, opens the dura mater, removes intracerebral hematoma and necrotic brain tissue, and expands the dura mater in a similar manner to standard DC. Then the condition of the brain tissue is assessed and restoration and fixation of the bone window with prefabricated titanium mesh is performed. Both bone flap removal and cranioplasty are accomplished at the same time. The procedure of DBFR is capable of preserving the skull integrity and achieving a decompressive effect. Comprehensive methods are utilized to decrease intracranial pressure after DBFR, including the reverse Trendelenburg position, lumbar drainage of cerebrospinal fluid, mild hypothermia therapy, and other traditional conservative treatments. After acute phase, the intracranial pressure gradually returns to the normal range, and the implanted titanium mesh can protect brain tissue from swings caused by atmospheric pressure or gravity. At the same time, the increased intracranial space will be occupied by normal brain tissue or encephalomalacia foci with accompanying gliosis. Therefore, DBFR itself does not induce new neurological dysfunction.

Case description

Case 1: A 50-year-old male was transported to the emergency department by ambulance after head trauma from a motorcycle accident. The CT scan showed multiple brain contusions, intracerebral hematoma, subdural hematoma, and subarachnoid hemorrhage. Cerebral edema was obvious. He was admitted to the department of neurosurgery in a conscious state. The score for GCS was 14 points. Colleagues performed conservative treatment for the patient, including using hemostatic and dehydrating agents and neurotrophy medication. Neurological deficits were detected. The patient’s GCS score decreased to 12 points 5 h later, and CT scans showed the intracerebral hematoma had increased and latent contusions had emerged; cerebral edema was observed. After serious consideration, colleagues agreed to continue conservative treatments. Next day, the CT scan revealed aggravation of the intracerebral hematoma and brain edema. The patient underwent emergency craniotomy. Considering the risk of delayed intracranial hematoma and brain edema, colleagues finally decided to remove the bone flap. After the surgery, traditional treatment continued to be used in order to control brain edema and prevent late hematoma and intracranial infection. In addition, lumbar puncture was utilized to drain the subarachnoid hemorrhage. The patient recovered well without any defects in neurological function. Three months later, he received cranioplasty (Fig. 1).

Case 2: A 61-year-old male was admitted to the neurology department for sudden confusion and had a GCS score of 9 points and NIHSS score of 15 points; in addition, CT scans showed massive cerebral infarction in the distribution of the middle cerebral artery. Intravenous thrombolytic therapy was carried out. Nine hours later, the GCS score had decreased to 6 points with unilateral mydriasis, and a CT scan showed the area of cerebral infarction was expanded. The patient underwent emergency DC and was transferred to the intensive care unit (ICU). Despite all our efforts, the patient became comatose and died soon after his family refused further treatments on the third day (Fig. 2).

In these 2 cases, both the patients underwent DC in crisis situations. Although he suffered from multiple brain injuries, the first patient recovered well after DC without delayed hematoma or massive cerebral edema. According to postoperative CT series, cerebral edema was under control, and none of factors that could induce intracranial hypertension were detected. So, DC was deemed to be overtreatment in this case. The patient had to undergo cranioplasty 3 months later. It would be hard to criticize the neurosurgeon for removing the bone flap, who made the decision according to the intraoperative situation. Afterall, putting the bone flap back is with a high risk of elevated ICP. On the contrary, replacing it with titanium mesh would solve the problem and that’s what DBFR does. Would DBFR increase the mortality rate? In case 2, intracranial pressure continued to increase after DC because the infarct area was enlarged, and forced brain tissue to shift against the bone window, but not out of the bone window. As in many other cases, there was no significant expansion of the skin flap observed in the early stage after the operation. Thus, we can assume that DBFR would not increase the mortality rate even in the worst situation.

The compensatory capacity of the skull after DC is decided by the volume of the bone flap and additional space by shift of the skin flap. Acute scalp flap expansion is restricted [18–20]. Further, because of scalp swelling, temporal muscle hematoma, dural surface tension, additional space by skin flap shift is limited. To solve this problem, Akutsu (2013) et al used artificial dermis to expand the area of the scalp flap in DC; however, it takes 1 to 6 months for the skin flap to complete epithelialization [21]. On the other hand, enlarging the volume of the bone flap can increase cranial compensatory volume immediately. Based on volumetric formula, both increase in bone flap size and thickness can enlarge the volume. There are conflicting results concerning the relationship between cranial thickness and sex, age, and general body build. The cranial thickness may increase with age [22–24] and have no significant relationship with the sex, weight, or stature of an individual [25,26]. The average thickness of the bone flap is relatively constant for adult people. Generally, cranial thickness ranged from 1.1 to 13.6 mm, with an average thickness of 5.0 mm at 7 cm from the midline [27]. Bone flap diameter is about 15 cm in a standard DC procedure and about 17 cm in decompressive hemicraniectomy (DHC). The maximum volume of craniectomy bone in standard DC is 95.5 ± 24.8 mL, and 131.8 ± 32.9 mL in DHC [28]. Thus, the first step of DBFR is similar to DHC, which can provide a large size of bone flap.

The brain injury contains primary and secondary injuries. Primary injury occurs immediately after trauma, including damage to the cerebral cortex and medulla, and intracranial vessel. Owing to lack of repair capability of nerve cells, therapeutic options are limited. Secondary injury occurs hours to days after the initial trauma. It is mainly composed of brain edema and intracranial hematoma. Brain edema after TBI or ICH is the leading cause of secondary brain injury, which is linked to high mortality [29]. According to the Monro-Kellie Doctrine, development of brain edema rapidly leads to an increase in intracranial pressure when autonomic regulation is impaired. The highest intracranial pressure was detected during the 3 to 5 days after injury [30]. Despite DBFR, postoperative intracranial pressure after DBFR may remain at a relatively high level for a longer period of time compared to regular DC or DHC. Traditional methods are needed after the surgery, including physical and medical treatments. In addition, follow-up CT scan is worthwhile to evaluate the intracranial situation.

Posttraumatic cerebral infarction (PTCI) is a severe complication in craniotomy. According to the study of Su (2017) et al, the incidence of
PTCI following DC was 31.2% [31], and it can induce massive brain edema and lead to unacceptable outcomes. PTCI may directly result in failure of surgical decompression in DBFR. Although knowing the risk factors including hyperthermia, traumatic subarachnoid hemorrhage, thrombocytopenia, and abnormal prothrombin time [32], it is impossible to confirm PTCI before DBFR. It is essential to define the indications for DBFR.

**Indications for DBFR**

The indications for DBFR are as follows: 1. Patient with massive hematoma (traumatic or non-traumatic); 2. Unilateral pupil mydriasis; 3. Vital signs remain stable without any pressor agent; and 4. Without injury to cerebral draining veins or to important blood supply arteries. Contraindications are as follows: bilateral pupil mydriasis; open craniospinal injury; open cranio-cerebral injury; massive cerebral infarction (primary or secondary); aged under 18 years old.

**Design for titanium mesh**

The type of materials which are used in cranioplasty have been mentioned above. Titanium has the advantage of being strong, thin, and biocompatible, which is perfect for DBFR. In secondary cranioplasty, the neurosurgeon shapes titanium mesh to fit the size of bone window by handwork, or alternatively uses customized titanium mesh shaped according to the three-dimensional CT reconstruction of the patient. Titanium mesh provides protection for the brain and maintains a stable intracranial pressure in this situation. However, in DBFR, maintaining the enlarged volume of the skull in a certain range is the chief requirement for titanium mesh in order to guarantee the decompressive effect; an inappropriate shape of concave mesh would decrease the compensatory capacity of the skull. It is critical that the concave shape of the titanium mesh is similar to, or larger than that of the contralateral skull (Fig. 3). Because the anatomical shape of the skull is similar among humans and both sides of the cranium are symmetrical, titanium mesh shares a similar shape with the appearance of the contralateral skull. Hence, it can be fabricated in advance and pre-shaped to different sizes of a universal model in the factory. Moreover, it is important to reserve holes on the titanium mesh for the drainage tube. In the future, with the development of 3-D printing technology and material science, the neurosurgeon can proceed with DBFR using customized replacement material.
replacement craniectomy. A: Using regular titanium mesh to replace bone flap. B: Using curvature enlarging titanium mesh to replace bone flap. TM: Titanium mesh; CETM: Curvature enlarging titanium mesh. Post-operational diagrammatic drawing for decompressive bone flap replacement craniectomy. Fig. 3.

Key points of DBFR

The DBFR procedure combines DC and cranioplasty. The first step of DBFR is similar to DHC, and in order to ensure the decompressive effect of DBFR, a large skin flap and bone flap are needed. During removal of the intracerebral hematoma, It is important to protect cerebral draining veins and important blood supply arteries. Then, the dura mater is expanded with the artificial dural graft, making sure the concave shape of the titanium mesh is appropriate, and the dura mater is closely adhered to the mesh. Finally, if necessary the galea is cut open or the temporalis muscle cut off to decrease the surface tension of the skin.

Potential risks of DBFR

First, the large size of skin flap and bone flap need additional surgical time and anesthesia, which could result in more blood loss, infection, or a cardiovascular event. Second, delayed hematoma (subdural, epidural, or intracranial), posttraumatic cerebral infarction, and surgical region infection may require surgical drainage or secondary craniotomy. Finally, the physical appearance of the head could be permanently asymmetrical, which may hinder good recovery of the patient.

Consequences of the hypothesis

If our hypothesis is correct, neurosurgeons can reduce the incidence of complications caused by intracranial pressure imbalance or dynamic changes in cerebrospinal fluid. The patient would have no need for secondary cranioplasty. In fact, DBFR can be complementary to DC, but it cannot replace the role of DC in treating ICH. Little evaluation has been done for this new surgical procedure, and further research is still required.

Funding

None.

Declaration of Competing Interest

None.

References