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Surface Area of Decompressive Craniectomy Predicts Bone Flap Failure after Autologous Cranioplasty: A Radiographic Cohort Study

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Abstract

Skull bone graft failure is a potential complication of autologous cranioplasty after decompressive craniectomy (DC). Our objective was to investigate the association of graft size with subsequent bone graft failure after autologous cranioplasty. This single-center retrospective cohort study included patients age ≥18 years who underwent primary autologous cranioplasty between 2010 and 2017. The primary outcome was bone flap failure requiring graft removal. Demographic, clinical, and radiographic factors were recorded; three-dimensional (3D) reconstructive imaging was used to perform accurate measurements. Univariate and multi-variate regression analysis were performed to identify risk factors for the primary outcome. Of the 131 patients who underwent primary autologous cranioplasty, 25 (19.0%) underwent removal of the graft after identification of bone flap necrosis on computed tomography (CT); 16 (64%) of these were culture positive. The mean surface area of craniectomy defect was 128.5 cm^2 for patients with bone necrosis and 114.9 cm^2 for those without bone necrosis. Linear regression analysis demonstrated that size of craniectomy defect was independently associated with subsequent bone flap failure; logistic regression analysis demonstrated a defect area >125 cm^2 was independently associated with failure (odds ratio [OR] 3.29; confidence interval [CI]: 0.249-2.135). Patient- and operation-specific variables were not significant predictors of bone necrosis. Our results showed that increased size of antecedent DC is an independent risk factor for bone flap failure after autologous cranioplasty. Given these findings, clinicians should consider the increased potential of bone flap failure after autologous cranioplasty among patients whose initial DC was >125 cm^2.

Keywords: autologous; bone flap; cranioplasty; infection; resorption

Introduction

Patients with pathologies such as traumatic brain injury (TBI),[1,2] malignant middle cerebral artery infarction,[3,4] hemorrhagic stroke,[5,6] or aneurysmal subarachnoid hemorrhage[7–9] may develop intracranial hypertension that is refractory to conservative treatments. For these patients, decompressive craniectomy (DC) is a standard treatment. During this surgical treatment for elevated intracranial pressure (ICP), a large portion of cranium is removed to allow for the mass effect and swelling;[10] autologous bone flaps are typically cryopreserved or subcutaneously implanted until the time of cranioplasty. After a patient's neurological and medical condition has stabilized and there

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is no longer concern for elevated ICP, the skull defect is repaired by cranioplasty. The primary goals of cranioplasty are to restore the cerebral protective function of the skull and for craniofacial cosmesis. Some patients also experience an improvement in neurological functioning[11–14] and a restoration of cerebrospinal fluid (CSF) dynamics.[15–19]

The use of an autologous bone flap has long been considered the gold standard for cranioplasty,[20–25] yet autologous cranioplasty complications occur at a rate of 15% to 40%.[26–33] The most common complications are surgical site infection (SSI) and aseptic necrosis or bone flap resorption (BFR). BFR is common, occurring in up to 90.2% of patients,[34] but BFR necessitating the removal of bone flap, that is, bone flap failure, is estimated to occur in 1.4% to 32.0% of patients.[28–30,34–37] Bone flap necrosis secondary to SSI occurs at a rate of 4.6% to 16.4%.[26,28,29,32,35]

Previous studies have attempted to identify risk factors for the development of bone flap failure including age, presence of hydrocephalus, and timing of cranioplasty; however, there is no consensus regarding which factor is the most predictive. Trephination defect size has been studied, but the studies were unable to accurately measure the surface area and relied on two-dimensional (2D) estimations.[29,36,38] We sought to examine the role of the size of the craniectomy defect and multiple other clinical factors on subsequent bone flap failure after autologous cranioplasty using a large cohort of patients from a single center. We hypothesized that the larger the size of craniectomy defect, the more likely bone flap failure would be observed after autologous cranioplasty.

Methods

Patient selection

This was a retrospective cohort study of adult patients age ≥18 years treated between January 1, 2010, and December 31, 2017, at University of Texas Health San Antonio University Hospital, a Level-1 trauma center and Joint Commission–certified comprehensive stroke center; the institutional review board approved the study (IRB protocol 15-0808H) with a waiver of informed consent. Patients were identified using a prospectively maintained neurosurgical database. All patient information was de-identified and analyzed in compliance with Health Insurance Portability and Accountability Act regulations. Each patient underwent DC, after which the bone flap was stored at −70°C in a freezer. In all cases, autologous bone flaps were thawed and soaked in betadine solution before reinsertion. The primary outcome of interest was bone flap failure, which was defined as bone necrosis visible on follow-up post-operative computed tomgraphy (CT) scan that required removal. Necrosis was defined based on radiographic evidence of demineralization of bone compared with immediate post-operative imaging; all images were reviewed by an attending neuroradiologist and an attending neurosurgeon. Scanning was performed at normal clinical follow-up time frames (1 month, 3 months, 6 months, 1 year) and in patients presenting with symptoms of bone flap failure (boggy scalp) or infection (fever, drainage, etc.).

Data acquisition

Patient demographic information including history of diabetes, smoking, or alcohol abuse and body mass index (BMI) was collected. Clinical information included indication for DC. Surgical details included whether or not there was fragmentation of the bone flap, operative time for cranioplasty, time to cranioplasty, size of craniectomy defect, and whether the patient required CSF diversion by external ventricular drain (EVD) placement or ventriculoperitoneal shunt placement (VPS).

The surface area of the craniectomy defect was calculated using three-dimensional (3D)-reconstructed renderings of the skull derived from thin-cut CT imaging without contrast performed after the initial DC operation (Fig. 1A,B). Calculations to measure the surface area of the craniectomy defect were made using iterative segmentation calculations using Phillips software (Fig. 1C) and confirmed with surface area calculations, which were performed by calculating an area of an ellipse. All patients diagnosed with bone flap failure requiring removal had intraoperative cultures taken of both their epidural tissue and the bone flap. Bone necrosis secondary to BFR was defined as lack of organism growth on microbiological analysis, whereas infectious bone necrosis yielded a microorganism on culture.

Statistical analysis

Data were summarized using means and standard deviations for continuous variables and counts and frequencies for categorical variables. Comparisons were made with Student's *t* test for continuous variables. A univariate regression analysis was performed to identify associations with bone flap failure. Both linear regression and logistic regression analyses were performed to identify independent risk factors for

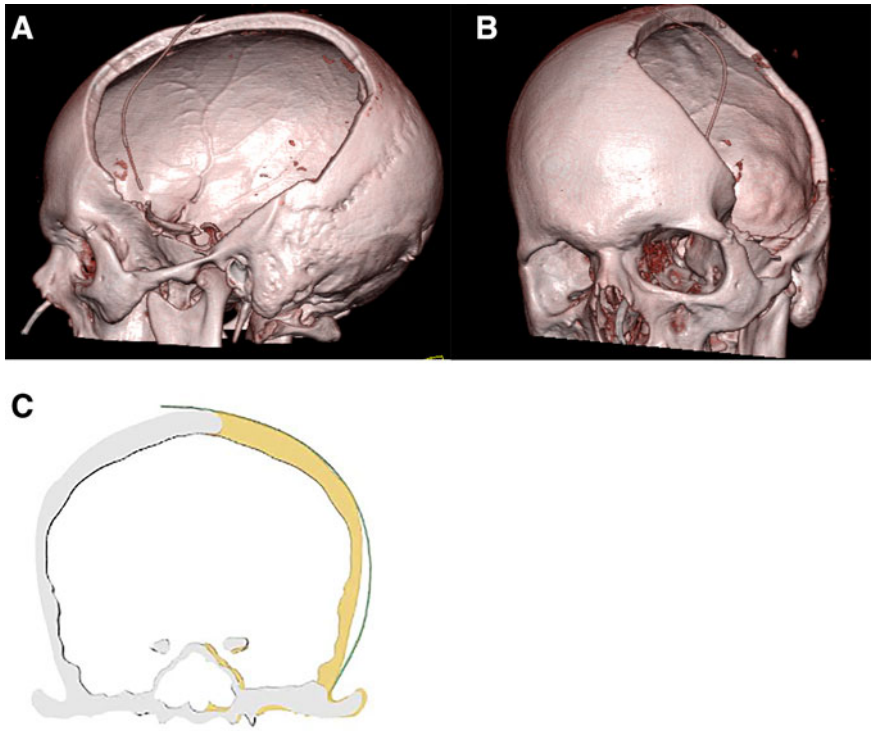

FIG. 1. **(A,B)** 3D reconstruction of a CT scan taken post-operatively after decompressive hemicraniectomy. 3D reconstructions were created for all patients and used to measure the surface area of the autologous bone graft. **(C)** Illustration of a coronal image demonstrating the left cranial defect. 3D, three dimensional; CT, computed tomography.

bone flap failure. The variables included in the regression models were those of clinical interest as potential independent risk factors.[23,29,31,34–37,39–42] Variables included in the linear regression analysis included those showing as statistically significant on univariate analysis or those with clinical significance surface area of craniectomy defect, age at cranioplasty, preoperative Glasgow Coma Scale (GCS) score, operative time, and time to cranioplasty. Variables included in the logistic regression model included cranioplasty defect >125 cm^2, diabetes, smoking history, alcohol abuse, BMI >25, fragmentation, and the need for CSF diversion after cranioplasty. Statistical significance was established using a cutoff of $p < 0.05$.

Results

Patient characteristics and operative factors

Of the 281 patients identified, 150 patients were excluded because they underwent non-autologous cranioplasty, were lost to follow-up, or had inadequate initial imaging to allow for 3D reconstructions and craniectomy defect surface area calculations (Fig. 2). Thirteen different neurosurgeons performed the 131 cranioplasty surgeries at a mean of 111.1 ± 58.9 days after initial DC.

Patient demographics at time of autologous cranioplasty are detailed in Table 1. The mean patient age was 38.5 ± 13.9 years and 72% were male. One-hundred sixteen patients (89%) had experienced a TBI and 15 had experienced malignant stroke requiring DC. Bifrontal DC had been performed in 2 (1.5%) patients, the other 129 patients underwent unilateral hemicraniectomy. A total of 81 (62%) patients were obese (BMI >25), 19 (14.5%) had diabetes, 43 (32.8%) had hypertension, 15 (11.5%) had hyperlipidemia or cardiovascular disease, 90 (68.7%) were smokers at the time of autologous cranioplasty, and 36 (27.4%) had a diagnosis of alcohol abuse.

Autologous cranioplasty operations had a mean operative time of 130.4 ± 60.1 min (range 44–490 min). The mean surface area of craniectomy defect was

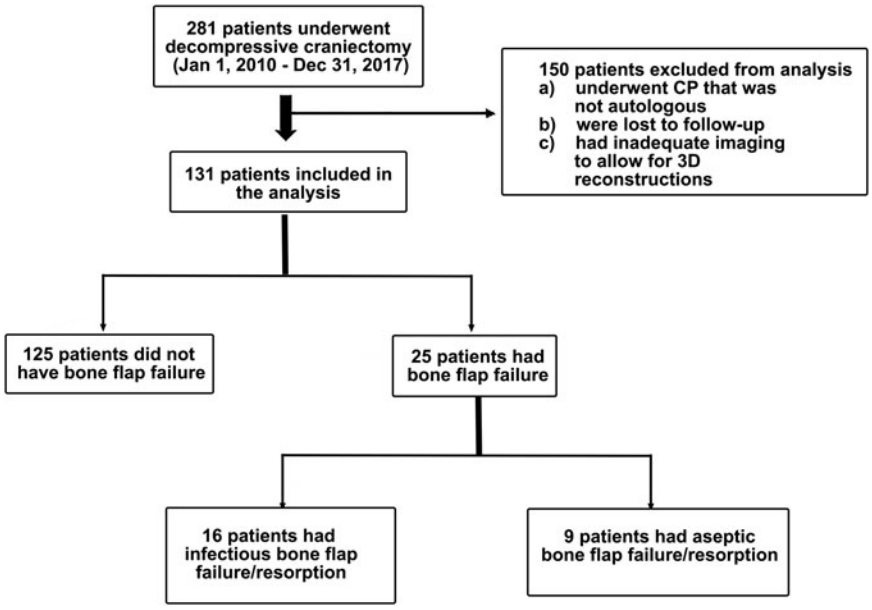

FIG. 2. Flow diagram illustrating cohort selection, inclusion, and exclusion. 3D, three dimensional; CP, cranioplasty.

$114.9 \pm 21.2\ cm^2$ (range 59.9–165.7 cm^2); the mean surface area in those who had bone flap failure was 128.5 cm^2, which was significantly higher than that of those who did not (111.8 cm^2; $p < 0.001$). Bone flap fragmentation was noted in 19 cases (14.5%). In the cases of bone fragmentation, the individual pieces were rejoined during the autologous cranioplasty and affixed with cranial plating systems before implantation. Thirteen (9.9%) patients required EVD placement at the time of autologous cranioplasty. VPS was required in 14 (10.6%) patients; 5 of these patients had the procedure performed before cranioplasty and 9 during or after the autologous cranioplasty operation.

Table 1. Univariate Analysis Comparing Patients with Bone Flap Failure and Those without Failure

Variable	*Total cohort (n = 131)*	*No failure (n = 106)*	*Failure (n = 25)*	P-*value*
Age at cranioplasty (years)	38.5 ± 14.0	38.8 ± 14.2	37.1 ± 12.8	0.57
Operative time (min)	130.4 ± 60.1	126.5 ± 61.1	146.6 ± 51.4	0.14
Area of bone removal (cm^2)	114.9 ± 21.2	111.8 ± 19.7	128.5 ± 21.5	<0.001
Time to cranioplasty (days)	111.1 ± 58.9	110.3 ± 59.3	114.4 ± 55.5	0.75
Required EVD	14 (10.6%)	11 (10.3%)	3 (12.0%)	0.81
Required VPS	14 (10.6%)	11 (10.3%)	3 (12.0%)	0.81
Trauma	116 (88.5%)	95 (89.6%)	21 (84.0%)	0.43
Malignant stroke	15 (11.4%)	11 (10.3%)	4 (16.0%)	0.43
Fragmentation	19 (14.5%)	14 (13.2%)	5 (20.0%)	0.39
CSF diversion after cranioplasty	27[a] (20.6%)	20 (18.8%)	7 (28.0%)	0.31
Diabetes	19 (14.5%)	17 (16.0%)	2 (8.0%)	0.31
Hyperlipidemia, cardiopulmonary disease	15 (11.5%)	13 (12.2%)	2 (8.0%)	0.55
Hypertension	43 (32.8%)	35 (33.0%)	8 (32.0%)	0.92
Smoker	90 (68.7%)	81 (76.4%)	9 (36.0%)	0.27
Infection	16 (12.2%)	0 (0%)	16 (64.0%)	<0.001
Alcohol abuse	36 (27.4%)	32 (30.1%)	4 (16.0%)	0.15
BMI >25	81 (61.8%)	65 (61.3%)	16 (64.0%)	0.80
Cranioplasty area >125 cm^2	44 (33.6%)	30 (28.3%)	14 (56.0%)	0.01

[a]One patient required EVD and VPS.
BMI, body mass index; CSF, cerebrospinal fluid; EVD, external ventricular drain; VPS, ventriculoperitoneal shunt.

Bone necrosis and bone flap surface area

Of the 131 patients who underwent cranioplasty, 25 (19.0%) patients developed bone flap failure of the reinserted bone flap in a mean time of 228.5 ± 178 days after cranioplasty. Six of 43 patients (13.9%) who underwent early cranioplasty (within <90 days) had bone flap failure, whereas 20/88 (22.7%) patients who underwent late cranioplasty (>90 days) had bone flap failure. Aseptic BFR was identified in 9 (36%) patients and 16 (64%) patients had bone necrosis secondary to infection. Four (16%) of the infected bone flaps that were cultured at the time of bone flap removal grew a combination of organisms, including methicillin-susceptible *Staphylococcus aureus* (MSSA), methicillin-resistant *S. aureus* (MRSA), *Bacteroides fragiles*, *Serratia marcescens*, and *Cutibacterium acnes*. Of those bone flaps in which only a single organism was isolated, 5 (20%) grew *C. acnes*, 2 (8%) grew MRSA, 2 (8%) grew *Enterobacter cloacae*, 1 (4%) grew *Klebsiella pneumoniae*, 1 (4%) MSSA, and 1 (4%) grew coagulase-negative *Staphylococcus*.

There were no statistically significant differences in the baseline characteristics when comparing cohorts (Table 1). Linear regression analysis demonstrated that the continuous variable of surface area of craniectomy defect had a significant association ($p < 0.001$) with bone flap failure, but age at cranioplasty, pre-operative GCS score, operative time, and time to cranioplasty did not (Table 2). Patients who experienced failure of their autologous bone graft were significantly more likely to have a pre-operative craniectomy defect of >125 cm^2 (56% vs. 28.%; $p = 0.008$). Logistic regression analysis demonstrated that a craniectomy defect >125 cm^2 was an independent predictor of eventual bone flap failure (OR 3.29; confidence interval [CI]: 0.249–2.135) after autologous cranioplasty (Table 3). No other variables were found to be significant.

Table 2. Linear Regression Analysis Demonstrating Association of Continuous Variables on the Outcome of Bone Flap Failure

Variable	P-*value*
Bone flap area	<0.001
Age at cranioplasty	0.418
Pre-operative GCS score	0.830
Operating time	0.914
Time to cranioplasty	0.763

GCS, Glasgow Coma Scale.

Table 3. Logistic Regression Analysis Demonstrating the Association of Categorical Variables on the Outcomes of Bone Flap Failure

Variable	*OR*	*95% CI*	P-*value*
>125 cm^2	3.29	0.25–2.14	**0.01**
Diabetes	0.47	−2.37–0.86	0.36
Smoker	0.78	−1.26–0.76	0.63
Alcohol abuse	0.48	−2.0–0.55	0.26
BMI >25	1.27	−0.74–1.22	0.63
Fragmentation	1.72	−0.67–1.75	0.38
CSF diversion after cranioplasty	1.08	−1.02–1.18	0.89

Boldface values indicate statistical significance at $p < 0.05$.
BMI, body mass index; CI, confidence interval; CSF, cerebrospinal fluid; OR, odds ratio.

Discussion

Autologous bone flaps are more commonly used in cranioplasty than other materials because of the low cost and exact fit to the cranial defect;[20–25] however, high rates of complications, most commonly BFR and SSI, have been reported.[28–30,34–37] Consistent with these past studies, 19.0% of patients in this series experienced bone flap failure requiring removal.

Based on our experience at a large, metropolitan academic medical center, we hypothesized that the size of the antecedent cranial defect was associated with subsequent bone flap failure after autologous cranioplasty. Some previous studies have determined that craniectomy defect size is a possible risk factor, whereas others have found no correlation.[29,36,38] These negative studies included 372, 207, and 58 patients, respectively, but they used 2D techniques in estimating the surface area of the bone defect, which is a significant limitation. Because of the curvature of the skull, these calculations likely underestimated the true surface area of the defect. The current study parallels the findings of Schoekler and Trummer[38] but is the first to use 3D reformats to analyze the craniectomy defect. Our results demonstrate that the surface area of the pre-operative craniectomy defect is significantly associated with subsequent development of bone flap failure, with a value of >125 cm^2 serving as a threshold at which the incidence of complications from bone flap failure is increased.

Previous studies focused on BFR or aseptic necrosis have identified younger age and the presence of bone fragmentation or VPS as independent risk factors.[23,29,34–36,41,42] Although we did not find that bone fragmentation was correlated with bone flap failure, it has been shown by many studies to be a significant risk factor[35–37,42,43] and could be explained by a similar mechanism. Others have found smoking, short time to cranioplasty, and long storage times to

be potential risk factors.[29,35,37] Previous studies on SSI in cranioplasty have identified diabetes, longer cranioplasty operation duration, and shorter time to cranioplasty as risk factors for SSI leading to bone flap necrosis.[31,39,40]

Bone flap failure complications are observed more commonly among pediatric patients.[23,29,34–37,41–44] Several reports have demonstrated that age <18 years is a risk factor for bone resorption,[37,44] whereas others have demonstrated that age <30 years is a significant risk factor.[29,42] In this study with an adult cohort, there were not significant differences in age between cohorts. An additional factor that can influence bone flap failure is time from decompression to cranioplasty. Schuss and colleagues[37] found that cranioplasty performed within 2 months of DC had a significantly higher risk of aseptic bone necrosis. Conversely, Brommeland and associates[35] found that a longer storage time increased the risk of BFR. In our study, there was no association between time to cranioplasty and bone flap failure, which previous studies have shown as well.[29,36,38,44] Most patients (64%) in our cohort experienced bone flap failure as a result of infection; in a retrospective study of 754 autologous cranioplasty operations, Morton and co-workers[31] found the only independent risk factor for bone flap infection was performing cranioplasty <14 days from DC. In the current study, three patients underwent autologous cranioplasty within 14 days of their initial decompression, none of whom experienced bone flap failure requiring removal. Our data demonstrated no difference in timing of cranioplasty between patients with bone flap failure (mean 114.4 days) and and those with no failure (mean 110.4 days), so we were not able to determine a clinically meaningful cutoff in this data set; future studies should focus on this metric. In this study, 13.9% who underwent early cranioplasty (within <90 days) versus 22.7% who underwent late cranioplasty experienced bone flap failure; this trend differs from previous reports, and further study on this subject should focus on this metric.

Patients who develop shunt-dependent hydrocephalus may also be at increased risk for aseptic bone necrosis[36,37,42,43]; however, a large meta-analysis demonstrated that hydrocephalus was associated with early cranioplasty but not with BFR or SSI.[45] A potential explanation is that placement of a VPS disrupts the contact between the dura and the bone graft, decreasing bone growth and allowing for a higher rate of BFR.[43] However, other studies are consistent with our results and did not find the presence of a VPS to be an independent risk factor for bone flap failure.[35,41,46]

The size of the bone flap removed during DC is predicated on the need to remove enough bone to achieve an adequate decompression to prevent/relieve intracranial hypertension. Given the time-sensitive and life-saving nature of DC, consideration of potential later risk of bone flap failure from the size of decompression should not be a factor. However, given the increased propensity of subsequent bone flap failure after autologous cranioplasty among patients with bone flaps >125 cm^2 in this study and the cost related to subsequent revision surgeries, future studies could focus on performing cranioplasty with titanium or poly-ether-ether-ketone implants in lieu of autologous cranioplasty for large defects. A recent randomized controlled trial by Honeybul and colleagues[23] comparing autologous and titanium cranioplasty in 64 patients showed that titanium cranioplasty was associated with better cosmetic and functional outcomes without increasing overall costs. Further studies should evaluate whether synthetic flaps could reduce the flap failure rate in larger defects.

There are several limitations to the current study. This was a retrospective cohort study, so the accuracy of the data is subject to recall bias. There were a large number of patients excluded from the study because of lack of follow-up and imaging to calculate the craniectomy defect; lack of long-term follow-up is common in the trauma population.[47] Although this was a single-center study, it represents the experience of 13 neurosurgeons in a large metropolitan area and there may have been practice variations with respect to timing to bone flap replacement, size of craniectomy, threshold for removal, and definitions of resorption. There are certainly technical surgical risk factors specific to an individual surgeon that may increase the risk for bone flap failure, but given the heterogeneity in surgeon practice in the current study, we were not able to identify any trends among individual surgeons and bone flap failure requiring removal; this represents a future area of potential study. Despite the limitations, we believe the finding of bone flap size as an independent predictor of bone flap failure is noteworthy and should be investigated further.

Conclusion

We demonstrated that larger craniectomy defects, specifically those >125 cm^2, were independently associated with bone flap failure requiring removal. In patients who undergo DC and require removal of large flaps, consideration should be given to performing

cranioplasty with alternative materials. Future studies comparing autologous cranioplasty with synthetic cranioplasty are necessary with common data elements and focus on cost-effectiveness and efficacy.

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Authors' Contributions

Authors contributions are as follows; conception and design: R.G., data collection: C.W.J., M.I., T.T.P., and T.F.; statistical analysis: V.M.R. and C.W.J.; writing of article manuscript: V.M.R. and R.G.; critical editing of article manuscript: V.M.R., C.W.J., and R.G.; review of article manuscript: all authors.

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Author Disclosure Statement

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Abbreviations Used

2D = two-dimensional
3D = three-dimensional
BFR = bone flap resorption
BMI = body mass index
CI = confidence interval
CP = cranioplasty
CSF = cerebrospinal fluid
DC = decompressive craniectomy
EVD = external ventricular drain
GCS = Glasgow Coma Scale
ICP = intracranial pressure
IRB = institutional review board
MSSA = methicillin-susceptible *Staphylococcus aureus*
MRSA = methicillin-resistant *Staphylococcus aureus*
OR = odds ratio
SSI = surgical site infection
TBI = traumatic brain injury
VPS = ventriculoperitoneal shunt