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Decompressive Craniectomy for Intracranial Pathologies in the Intensive Care Unit : Effects on Mortality and Clinical Course

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Objective : Decompressive craniectomy (DC) is a surgical procedure in which a portion of the skull overlying edematous and vulnerable brain tissue is removed to reduce intracranial pressure. In cases where all other treatment modalities fail, DC can be life-saving. However, this procedure is not a "panacea;" it can prolong the death process and condemn the patient to a life in need of care, with severe neurological sequelae.

Methods : This multicenter, retrospective study recorded patients' comorbidities, Glasgow coma scale (GCS), Acute physiology and chronic health evaluation II (APACHE II) and Sequential organ failure assessment (SOFA) scores, as well as relevant laboratory parameters. Patients were categorized into three groups according to the Glasgow outcome scale.

Results : Among 201 patients, 41.3% expired, 33.8% survived with severe vegetative sequelae, and 24.9% had mild to moderate sequelae. Non-survivors had significantly lower preoperative and postoperative GCS scores (median 7 vs. 9, p=0.010; 3 vs. 10, p<0.001) and higher 24-hour peak glucose levels (186 vs. 164 mg/dL, p=0.009). Steroid therapy was ineffective. Preoperative GCS scores were significantly lower in patients who later developed severe sequelae compared to those with better outcomes (median 8 vs. 11, p<0.001). Postoperative GCS scores were also lower in this group (8 vs. 13, p<0.001).Additionally, APACHE II (22 vs. 18, p=0.002) and SOFA scores (4 vs. 3, p<0.001) were higher, indicating greater disease severity.

Conclusion : DC can be a lifesaving procedure in selected patients with appropriate indications and appropriate timing. However, the possibility of this procedure resulting in high mortality and severe sequelae cannot be ignored. Therefore, the clinical outcomes that the patient and their families would find acceptable should be carefully considered.

Key Words : Decompressive craniectomy · Glasgow coma scale · APACHE · Sequential organ failure assessment · Glasgow outcome scale.

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INTRODUCTION

Decompressive craniectomy (DC) involves the surgical removal of a portion of the skull that surrounds the edematous and at-risk brain tissue. DC can be performed either primarily or secondarily[3]. It is a method employed to reduce intracranial pressure, improve cerebral compliance, and cerebral blood flow when medical treatment is insufficient[7]. This procedure is performed to control intracranial hypertension and break the pathophysiological vicious cycle that leads to secondary brain damage. Otherwise, the continuation of this process can result in brain herniation and brainstem death. Therefore, DC can play a life-saving role when all other treatments have failed. However, DC is not a panacea. The procedure may delay the process of death, leading to patients surviving with severe neurological sequelae[3]. This condition may lead to a low quality of life, which can be described as a "life that is not truly a life," where the individual is unaware of their surroundings and bedridden; it may also result in negative consequences for the patient's relatives, such as a heavy psychosocial burden and burnout.

With this study, we planned to investigate the effects of DC, on which a complete consensus has not yet been reached, on the intensive care process and mortality.

MATERIALS AND METHODS

This study was approved by the Institutional Research Board of Haydarpaşa Numune Training and Research Hospital (Approval number : HNEAH-KAEK 2022/126).

After obtaining approval from the local ethics committee (HNEAH-KAEK 2022/126), a pre-designed data collection form was distributed to the participating centers for data recording. This multicenter, retrospective study included the intensive care units (ICUs) of five training and research hospitals. Study data were obtained through retrospective review of medical records of patients admitted to the participating centers between 2019 and 2022 Patients aged ≥18 years who underwent DC due to intracranial hemorrhage (epidural or subdural), parenchymal lesions, or cerebral ischemia and were subsequently followed in the ICU were included in the study. Parenchymal lesions were defined as intraparenchymal hemorrhages and traumatic or non-traumatic parenchymal mass lesions causing significant cerebral edema and mass effect requiring DC. In addition, the indication for DC was primarily determined based on clinical deterioration and radiological evidence of life-threatening intracranial hypertension. DC was considered in patients with worsening neurological status (e.g., a decline in Glasgow coma scale [GCS] score or pupillary abnormalities) and/or neuroimaging findings of marked cerebral edema, mass effect, or midline shift who were unresponsive to optimized medical therapy. Primary DC was performed during initial lesion surgery or as an upfront intervention when malignant cerebral edema was anticipated, whereas secondary DC was undertaken as a rescue procedure following failure of maximal medical management. Trauma patients with concomitant extracranial injuries were included in the study, provided that intracranial pathology was the primary indication for DC and ICU admission. Patients younger than 18 years, those with pre-existing severe neurological deficits, and those with incomplete clinical data were excluded. Although minor technical variations existed between centers, DC was uniformly defined across all institutions as a large frontotemporoparietal craniectomy with an approximate bone flap diameter of ≥12 cm, combined with duroplasty using either autologous or synthetic materials. In accordance with this definition, our study evaluated the ICU course and discharge outcomes of patients who underwent decompressive surgery for intracranial pathologies.

The database recorded comorbidities that could affect the clinical course, early postoperative GCS scores, Acute physiology and chronic health evaluation II (APACHE II) scores, Glasgow outcome scale (GOS), highest blood glucose level, lowest serum sodium level, peak body temperature, and Sequential organ failure assessment (SOFA) scores on the day of surgery, as well as the duration of mechanical ventilation and length of ICU stay. Infections, one of the major causes of mortality during prolonged ICU stays, were documented based on culture results.

Table 1. GOS scoring system and descriptions

GOS score	Description
1	Death
2	Vegetative state
3	Severe disability — conscious but severely disabled, dependent
4	Moderate disability — disabled but independent
5	Good recovery — no disability, minor deficits; independent

GOS : Glasgow outcome scale

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ICU discharge outcomes were classified using the GOS. Patients were grouped as follows : group 1, expired (GOS 1); group 2, vegetative state or severe disability with major sequelae (GOS 2–3); and group 3, moderate disability or good recovery (GOS 4–5). The effects of the specified parameters on the defined outcome groups were evaluated comparatively (Table 1).

Statistical analysis

Data normality was assessed using the Shapiro-Wilk test. Normally distributed variables were expressed as mean±standard deviation, and non-normally distributed variables as median (interquartile range, IQR). Categorical variables were presented as counts and percentages. Group comparisons used the chi-square or Fisher's exact test for categorical data, and Student's t-test or Mann-Whitney U test for continuous data. Logistic regression identified predictors of severe sequelae or vegetative state, presented as odds ratios (ORs) with 95% confidence intervals (CIs). Survival was analyzed with Cox proportional hazards models. Analyses were performed using SPSS ver. 26.0 (IBM Corp., Armonk, NY, USA), with $p<0.05$ deemed significant.

RESULTS

The study included 201 patients (72 females, 35.8%; 129 males, 64.2%). Survival was similar between sexes (56.9% vs. 59.7%; $p=0.766$). GOS outcomes showed 41.3% expired, 17.4% in a vegetative state, 16.4% severe sequelae, 14.9% moderate disability, and 10.0% good recovery. Grouped outcomes indicated 41.3% expired, 33.8% severe, and 24.9% favorable recovery (Table 2).

Female patients accounted for 35.8% (n=72) of the study population. The mortality rate among females was 43.1%, compared to 40.3% in males; this difference was not statistically significant ($p=0.766$). The presence of certain comorbidities was associated with varying mortality outcomes. Diabetes mellitus was present in 21.9% (n=44) of patients, with a mortality rate of 54.5% compared to 37.6% among non-diabetic patients, a difference approaching statistical significance ($p=0.056$). Hypertension was observed in 52.7% (n=106) of patients and was significantly associated with increased mortality (48.1% vs. 33.7%; $p=0.045$). Heart failure, present in 9.5% (n=19) of cases, was strongly linked to higher mortality (73.7% vs. 37.9%; $p=0.003$). Similarly, chronic kidney disease (8.0%; n=16) and cancer (9.5%; n=19) were associated with significantly higher mortality rates (68.8% and 68.4%, respectively) compared to patients without these conditions ($p=0.032$ and $p=0.015$, respectively). A history of stroke (14.9%; n=30) was also associated with increased mortality (60.0% vs. 38.0%; $p=0.028$). Trauma was identified in 26.4% (n=53) of patients, but its presence did not significantly affect mortality (34.0% vs. 43.9%; $p=0.255$). Other comorbidities showed no significant association. Mortality was notably higher in patients with parenchymal hemorrhage (50.5%) compared to those admitted with infarction (31.4%) or epidural/subdural hemorrhage (33.3%) (Table 2).

No significant age difference was found between survivors

Table 2. Comparison of demographic characteristics and comorbidities between survivors (GOS 2–5) and non-survivors (GOS 1)

Patient characteristic	Survivors, GOS 2–5	Non-survivors, GOS 1	*p*-value
Sex			
Female	41 (56.9)	31 (43.1)	0.766
Male	77 (59.7)	52 (40.3)	
Diabetes mellitus	20 (45.5)	24 (54.5)	0.056
Hypertension	55 (51.9)	51 (48.1)	0.045*
Coronary artery disease	20 (48.8)	21 (51.2)	0.159
Heart failure	5 (26.3)	14 (73.7)	0.003*
COPD/asthma	7 (58.3)	5 (41.7)	1.000
Chronic kidney disease	5 (31.3)	11 (68.8)	0.032*
Valvular disease	3 (50.0)	3 (50.0)	0.693
Arrhythmia	9 (45.0)	11 (55.0)	0.233
Cancer	6 (31.6)	13 (68.4)	0.015*
Autoimmune disease	0 (0.0)	1 (100.0)	0.413
Pulmonary vascular disease	2 (40.0)	3 (60.0)	0.651
Hypothyroidism	5 (62.5)	3 (37.5)	1.000
Stroke	12 (40.0)	18 (60.0)	0.028*
Dementia	2 (66.7)	1 (33.3)	1.000
Other neurological disease	11 (78.6)	3 (21.4)	0.161
Obesity	2 (100.0)	0 (0.0)	0.513
Cirrhosis	2 (100.0)	0 (0.0)	0.513
Trauma	35 (66.0)	18 (34.0)	0.255
Admission reason			0.036*
Infarction	35 (68.6)†	16 (31.4)†	
Epidural/subdural hematoma	34 (66.7)†	17 (33.3)†	
Parenchymal lesion	49 (49.5)†	50 (50.5)‡	

Values are presented as number (%). *Significant *p*-values (<0.05). †‡Cells with different superscript symbols indicate statistically significant differences. GOS : Glasgow outcome scale, COPD : chronic obstructive pulmonary disease

and non-survivors, with median ages of 61 years (IQR, 48–71) and 62 years (IQR, 54–74), respectively (p=0.140). However, neurological status differed significantly between groups. Preoperative GCS were lower in non-survivors (median, 7; IQR, 5–10) than in survivors (median, 9; IQR, 6–12; p=0.010), and this difference became more pronounced postoperatively, with non-survivors showing markedly reduced scores (median, 3; IQR, 3–6) compared to survivors (median, 10; IQR, 7–13; p<0.001). Corticosteroid use was similar between groups, with both median daily and total doses of 0 mg (IQR, 0–4; p=0.546 and p=0.402, respectively). Laboratory findings revealed comparable postoperative 24-hour sodium levels (p=0.190) but significantly higher glucose levels in non-survivors (median, 186 mg/dL; IQR, 151–240) than survivors (median, 164 mg/dL; IQR, 141–205; p=0.009). Postoperative fever did not differ (p=0.355). Disease severity indicators were higher in non-survivors, with elevated APACHE II (median, 22 vs. 20; p=0.001) and Surgery day SOFA scores (median, 5 vs. 4; p<0.001). Survivors had longer ICU stays (median, 19 vs. 9 days; p=0.010), while mechanical ventilation duration was comparable (p=0.987). The use of mannitol (52.7%) and anticoagulants (28.9%) showed no significant mortality association (p=0.668 and p=0.117, respectively). Similarly, mortality did not differ between primary and secondary decompressive surgeries (p=0.197) (Table 3).

Table 3. Comparative analysis of laboratory parameters, GCS scores, and treatment variables in relation to mortality, comparing survivors (GOS 2–5) with non-survivors (GOS 1)

Patient characteristic	Survivors, GOS 2–5	Non-survivors, GOS 1	*p*-value
Age (years)	61 (48–71)	62 (54–74)	0.140
Preop GCS	9 (6–12)	7 (5–10)	0.010*
Postop GCS	10 (7–13)	3 (3–6)	<0.001*
Steroid per day	0 (0–4)	0 (0–4)	0.546
Steroid (mg)	0 (0–4)	0 (0–4)	0.402
Postop 24 hours low sodium	138 (136–141)	139 (135–144)	0.190
Postop 24 hours high glucose	164 (141–205)	186 (151–240)	0.009*
Postop 24 hours high fever	37 (37–37)	37 (37–37)	0.355
APACHE II	20 (13–25)	22 (18–30)	0.001*
Surgery day SOFA	4 (3–5)	5 (4–7)	<0.001*
ICU stay duration (days)	19 (8–41)	9 (5–30)	0.010*
Mechanical ventilation duration (days)	10 (3–33)	9 (4–26)	0.987
Mannitol	64 (60.4)	42 (39.6)	0.668
Anticoagulant	29 (50.0)	29 (50.0)	0.117
Decompressive surgery, primary	55 (53.9)	47 (46.1)	0.197
Decompressive surgery, secondary	63 (63.6)	36 (36.4)	-

Values are presented as median (interquartile range) or number (%). *Significant *p*-values (<0.05). GCS : Glasgow coma scale, GOS : Glasgow outcome scale, preop : preoperative, postop : postoperative, APACHE II : Acute physiology and chronic health evaluation II, SOFA : Sequential organ failure assessment, ICU : intensive care unit

Table 4. Demographic and comorbid characteristics of patients by outcome severity : vegetative state/severe disability (GOS 2–3) vs. moderate disability/good recovery (GOS 4–5)

Patient characteristic	Severe disability/ vegetative state, GOS 2–3	Good recovery/ moderate disability, GOS 4–5	*p*-value
Sex			
Female	25 (61.0)	16 (39.0)	0.696
Male	43 (55.8)	34 (44.2)	
Diabetes mellitus	12 (60.0)	8 (40.0)	1.000
Hypertension	36 (65.5)	19 (34.5)	0.136
Coronary artery disease	12 (60.0)	8 (40.0)	1.000
Heart failure	2 (40.0)	3 (60.0)	0.649
COPD/asthma	4 (57.1)	3 (42.9)	1.000
Chronic kidney disease	4 (80.0)	1 (20.0)	0.394
Valvular disease	3 (100.0)	0 (0.0)	0.261
Arrhythmia	6 (66.7)	3 (33.3)	0.731
Cancer	2 (33.3)	4 (66.7)	0.399
Pulmonary vascular disease	2 (100.0)	0 (0.0)	0.507
Hypothyroidism	3 (60.0)	2 (40.0)	1.000
Stroke	8 (66.7)	4 (33.3)	0.555
Dementia	0 (0.0)	2 (100.0)	0.177
Other neurological disease	8 (72.7)	3 (27.3)	0.351
Obesity	2 (100.0)	0 (0.0)	0.507
Cirrhosis	1 (50.0)	1 (50.0)	1.000
Trauma	14 (40.0)	21 (60.0)	0.015*
Admission reason			0.281
Infarction	23 (65.7)	12 (34.3)	
Epidural-subdural hemorrhage	16 (47.1)	18 (52.9)	
Parenchymal lesion	29 (59.2)	20 (40.8)	

Values are presented as number (%). *Significant *p*-values (<0.05). GOS : Glasgow outcome scale, COPD : chronic obstructive pulmonary disease

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Among 118 survivors, 50 (42.4%) had normal to moderate sequelae, while 68 (57.6%) developed severe sequelae or a vegetative state. Severe outcomes occurred in 61.0% of females and 55.8% of males, versus 39.0% and 44.2% with better outcomes, respectively; the difference was not statistically significant (p=0.696).

Comorbidities were also examined. Diabetes mellitus was present in 20 survivors (16.9%), with 60.0% experiencing severe outcomes versus 57.1% of non-diabetic patients (p=1.000). Hypertension was identified in 55 survivors (46.6%), with 65.5% developing severe sequelae compared to 50.8% of non-hypertensive patients (p=0.136). A history of trauma (n=35; 29.7%) was associated with a significantly lower rate of severe outcomes (40.0% vs. 65.1% in non-trauma patients; p=0.015). Other comorbidities, including coronary artery disease, heart failure, chronic obstructive pulmonary disease/asthma, chronic renal failure, arrhythmia, cancer, prior stroke, dementia, and hypothyroidism, were not significantly associated with functional outcomes (Table 4).

Age distribution was similar between patients with severe sequelae and those with more favorable outcomes, with median ages of 61 years (IQR, 51–73) and 60 years (IQR, 45–71), respectively (p=0.439). However, neurological status differed markedly. Preoperative GCS were significantly lower in patients with severe sequelae (median, 8; IQR, 5–10) compared to those with better outcomes (median, 11; IQR, 9–13; p<0.001), and this difference persisted postoperatively (median, 8; IQR, 6–10 vs. median, 13; IQR, 10–14; p<0.001). Corticosteroid administration patterns were comparable between groups, with both median daily and total doses of 0 (IQR, 0–4), showing no statistical sig-

Table 5. Comparison of laboratory values, GCS, and treatment parameters in patients with vegetative state/severe disability (GOS 2–3) vs. moderate disability/good recovery (GOS 4–5)

Patient characteristic	Severe disability/ vegetative state, GOS 2–3	Good recovery/ moderate disability, GOS 4–5	*p*-value
Age (years)	61 (51–73)	60 (45–71)	0.439
Preop GCS	8 (5–10)	11 (9–13)	<0.001*
Postop GCS	8 (6–10)	13 (10–14)	<0.001*
Steroid per day	0 (0–4)	0 (0–4)	0.203
Steroid (mg)	0 (0–4)	0 (0–4)	0.166
Postop 24 hours low sodium	138 (136–141)	138 (136–140)	0.795
Postop 24 hours high glucose	168 (146–217)	157 (134–196)	0.179
Postop 24 hours high fever	37 (37–37)	37 (37–37)	0.630
APACHE II	22 (16–25)	18 (10–22)	0.002*
Surgery day SOFA	4 (3–5)	3 (2–4)	<0.001*
ICU stay duration (days)	30 (14–48)	11 (5–21)	<0.001*
MV duration (days)	24 (6–44)	6 (1–10)	<0.001*
Mannitol	43 (67.2)	21 (32.8)	0.026*
Anticoagulant	13 (44.8)	16 (55.2)	0.132
Decompressive surgery, primary	28 (50.9)	27 (49.1)	0.194
Decompressive surgery, secondary	40 (63.5)	23 (36.5)	-

Values are presented as median (interquartile range) or number (%). *Significant *p*-values (<0.05). GCS : Glasgow coma scale, GOS : Glasgow outcome scale, preop : preoperative, postop : postoperative, APACHE II : Acute physiology and chronic health evaluation II, SOFA : Sequential organ failure assessment, ICU : intensive care unit, MV : mechanical ventilation

Table 6. Multivariable survival analysis

Predictor	HR	95% CI for HR	*p*-value
Admission reason			0.016*
Infarction vs. parenchymal lesion	0.37	0.15–0.94	0.037*
Epidural/subdural hemorrhage vs. parenchymal lesion	1.09	0.55–2.18	0.802
Diabetes, yes vs. no	0.81	0.42–1.56	0.523
Coronary artery disease, yes vs. no	0.80	0.41–1.57	0.514
Heart failure, yes vs. no	3.97	1.86–8.47	<0.001*
Chronic kidney disease, yes vs. no	1.45	0.57–3.67	0.437
Cancer, yes vs. no	2.18	1.03–4.60	0.041*
Prior stroke, yes vs. no	1.15	0.58–2.29	0.692
Other neurological disease, yes vs. no	0.47	0.11–1.96	0.302
Anticoagulant use, yes vs. no	1.16	0.64–2.11	0.631
Decompressive surgery, secondary vs. primary	0.62	0.34–1.12	0.114
Age (per years)	0.99	0.97–1.01	0.426
Postoperative GCS (per points)	0.74	0.66–0.83	<0.001*
Post-op 24-hour sodium low (per mmol/L)	1.03	0.99–1.07	0.224
Post-op 24-hour glucose high (per mg/dL)	1.004	1.000–1.008	0.048*
APACHE II (per points)	1.01	0.98–1.04	0.631
SOFA on surgery day (per points)	1.13	1.04–1.24	0.006*

*Significant *p*-values (<0.05). HR : hazard ratio, CI : confidence interval, GCS : Glasgow coma scale, APACHE II : Acute physiology and chronic health evaluation II, SOFA : Sequential organ failure assessment

nificance (p=0.203 and p=0.166, respectively). Laboratory results indicated similar postoperative 24-hour sodium levels (p=0.795) and slightly higher, though not significant, glucose levels in the severe sequelae group (median, 168 vs. 157 mg/dL; p=0.179). Postoperative fever showed no group difference (p=0.630). Disease severity measures were higher among patients with severe sequelae, as reflected by elevated APACHE II (median, 22 vs. 18; p=0.002) and Surgery day SOFA scores (median, 4 vs. 3; p<0.001). Intensive care parameters also differed substantially : patients with severe sequelae had longer ICU stays (median, 30 vs. 11 days; p<0.001) and mechanical ventilation durations (median, 24 vs. 6 days; p<0.001). Mannitol use was associated with a higher incidence of severe sequelae (67.2% vs. 32.8%; p=0.026), while anticoagulant therapy showed a non-significant trend toward more favorable outcomes (p=0.132) (Table 5).

Thirty-day mortality was observed in 68 of 201 patients (33.8%). In multivariable Cox regression analysis adjusting for demographic, clinical, and perioperative variables, admission diagnosis remained an independent determinant of survival (Wald χ^2=8.32; df=2; p=0.016). Patients admitted with cerebral infarction had a 63% lower risk of death compared to those with parenchymal hemorrhage (hazard ratio [HR], 0.37; 95% CI, 0.15–0.94; p=0.037), whereas those with epidural or subdural hematomas showed no significant difference (HR, 1.09; p=0.80). Among comorbidities, cardiac dysfunction had the most pronounced adverse impact, as pre-existing heart failure nearly quadrupled the 30-day mortality risk (HR, 3.97; 95% CI, 1.86–8.47; p<0.001). Active malignancy also increased the risk, doubling the hazard (HR, 2.18; 95% CI, 1.03–4.60; p=0.041). Postoperative neurological recovery was strongly predictive; each one-point increase in the GCS reduced mortality by 26% (HR, 0.74; p<0.001). Disease severity contributed independently, with each additional point in the Surgery day SOFA score increasing mortality risk by 13% (HR, 1.13; p=0.006). Elevated postoperative glucose modestly increased hazard (HR, 1.004; p=0.048), while sodium level, APACHE II score, age, and secondary surgery were not significant predictors. Model diagnostics confirmed no proportional hazards violations, and the concordance index (Harrell's C=0.83) indicated strong predictive accuracy (Table 6).

Table 7. Multivariable analysis of predictors for severe sequelae or vegetative state

Predictor	OR	95% CI for OR	p-value
Hypertension, yes vs. no	1.13	0.38–3.41	0.823
Mannitol use, yes vs. no	0.95	0.33–2.70	0.921
Trauma, yes vs. no	0.13	0.03–0.52	0.004*
Decompressive surgery, secondary vs. primary	1.19	0.42–3.44	0.742
Anticoagulant use, yes vs. no	0.22	0.06–0.75	0.016*
Steroid dose, mg/day	1.18	0.89–1.57	0.257
Preoperative GCS (per point)	1.00	0.82–1.23	0.995
Postoperative GCS (per point)	0.73	0.59–0.90	0.004*
Postoperative 24-hour highest glucose (mg/dL)	1.001	0.991–1.011	0.899
APACHE II (per point)	1.05	0.98–1.13	0.149
SOFA on surgery day (per point)	1.29	0.93–1.78	0.125
Mechanical ventilation duration (days)	1.02	0.99–1.05	0.254
Admission reason	0.82	0.42–1.62	0.570

*Significant p-values (<0.05). OR : odds ratio, CI : confidence interval, GCS : Glasgow coma scale, APACHE II : Acute physiology and chronic health evaluation II, SOFA : Sequential organ failure assessment

The multivariate analysis revealed key predictors associated with functional outcomes after decompressive surgery. Traumatic etiology emerged as a significant favorable factor, with trauma patients showing 87% lower odds of severe disability or vegetative state compared to non-trauma cases (OR, 0.13; 95% CI, 0.032–0.517; p=0.004). Anticoagulant therapy also demonstrated a strong protective effect, reducing the likelihood of severe impairment by 78% (OR, 0.22; 95% CI, 0.064–0.753; p=0.016). Postoperative GCS was an independent prognostic factor; each one-point increase decreased the risk of severe sequelae by 27% (OR, 0.727; 95% CI, 0.586–0.902; p=0.004), emphasizing the importance of early postoperative neurological status.

Conversely, hypertension, mannitol administration, preoperative GCS, disease severity indices (APACHE II and Surgery day SOFA), type of decompressive surgery, duration of mechanical ventilation, and postoperative hyperglycemia were not significantly associated with functional outcomes (p>0.05), indicating that postoperative neurological and etiological factors may have stronger prognostic relevance than general clinical parameter (Table 7).

DISCUSSION

DC involves surgically removing part of the skull to relieve

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pressure on swollen or compromised brain tissue. Its effects on clinical outcomes and quality of life remain debated. This study aimed to evaluate the intensive care outcomes of DC, the factors influencing these outcomes, and its effects on mortality and recovery. Although evidence supports its life-saving potential, uncertainties regarding indications and patient selection persist[22]. Several studies report reduced mortality and improved outcomes, especially with early intervention[4,11], while others note unfavorable results[6].

Park et al.[20], in their investigation of early DC outcomes among patients with severe cerebral edema, reported that 48% achieved favorable recovery (GOS score 4 or 5), 30% experienced poor recovery (GOS score 2 or 3), and 22% succumbed to mortality. Schwab et al.[23] demonstrated that early DC, performed at a mean of 21 hours post-infarction, was associated with lower mortality (16%) compared to late intervention (34.4%), emphasizing the importance of timing in optimizing outcomes. Similarly, Mori et al.[16] reported significantly lower six-month mortality in early DC (4.8%) versus late DC (17.2%), with higher preoperative GCS in the early group (11.2 vs. 6.6). Almino Leite et al.[1] found that patients with preoperative GCS ≤8 had a 1.93-fold higher risk of mortality than those with GCS >8. Ammar et al.[2] identified age >65 years, GCS ≤8, SOFA score >5, hypercapnia (>45 mmHg), disseminated intravascular coagulation, and corticosteroid use as independent predictors of mortality in DC patients. Tsai et al.[26] highlighted that preoperative GCS >8 and APACHE II <13 were associated with better clinical outcomes.

In our study, we obtained results similar to those reported in the literature. Surgery day SOFA scores were higher in non-survivors (median, 5; IQR, 4–7) than survivors (median, 4; IQR, 3–5; $p<0.001$). Preoperative GCS was lower in non-survivors (median, 7; IQR, 5–10) compared to survivors (median, 9; IQR, 6–12; $p=0.010$), and this difference became more pronounced postoperatively (median, 3; IQR, 3–6 vs. median 10; IQR, 7–13; $p<0.001$). Notably, each 1-point increase in postoperative GCS reduced mortality by 26% (HR, 0.74; 95% CI, 0.66–0.83; $p<0.001$) and severe disability risk by 27% (OR, 0.727; 95% CI, 0.586–0.902; $p=0.004$). Contrary to Ammar et al.[2], corticosteroid use did not significantly influence outcomes, highlighting the primary prognostic role of patient-specific physiological and neurological parameters following DC. Clinically, our findings underscore the importance of careful patient selection, timely surgical intervention, and rigorous postoperative neurological monitoring. Postoperative GCS serves as a reliable predictor of both survival and functional recovery, guiding intensive care and rehabilitation strategies.

Outcomes after DC are not uniformly favorable, as a substantial proportion of patients survive with severe disability, require long-term care, or remain bedridden. Honeybul et al.[13] reported that DC patients had modified Rankin scores of 4 or 5, indicating high risk of poor outcomes. Similarly, Howard et al.[14] evaluated 40 DC patients, finding 22 deaths, six with low Glasgow outcome scale-extended (GOSE), and only 12 (30%) achieving high GOSE, representing 70% with unfavorable functional outcomes. Nambiar et al.[19] observed that 68% of DC patients experienced poor outcomes, with high admission APACHE II scores as the strongest predictor of low GOSE and 6-month mortality. Cooper et al.[5], in 155 patients, demonstrated that early bifrontotemporoparietal DC reduced intracranial pressure and ICU stay but was associated with 70% poor functional outcomes. Our study yielded results consistent with the literature. According to the GOS outcomes, 41.3% of the patients died, 17.4% remained in a vegetative state, and 16.4% developed severe sequelae (a total of 33.8% with GOS 2–3), while 14.9% had moderate disability and 10.0% achieved good recovery (a total of 24.9% with GOS 4–5). APACHE II scores were significantly higher in non-survivors, with a median of 22 (IQR, 18–30) versus 20 (IQR, 13–25) in survivors ($p=0.001$). Similarly, surgery-day SOFA scores were elevated in non-survivors (median 5; IQR, 4–7 vs. median, 4; IQR, 3–5; $p<0.001$). Each one-point increase in Surgery day SOFA score raised mortality risk by 13% (HR, 1.13; $p=0.006$), highlighting that APACHE II and SOFA scores are reliable predictors of clinical course, mortality, and functional outcomes following DC. One of the major limitations of our study is its retrospective design and intercenter differences in follow-up, which precluded the availability of standardized 3–6 months or 12-month functional outcome data. Consequently, our findings are limited to early ICU outcomes and early functional status.

Mortality in patients undergoing DC is influenced by multiple factors, with metabolic parameters playing a key role. Blood glucose levels, in particular, are strongly associated with outcomes. He et al.[12] categorized glucose as ≤6.1 mmol/L (normoglycemia), >6.1–7.8 mmol/L (mild), >7.8–10.0 mmol/L (moderate), and >10.0 mmol/L (severe), finding moderate and severe hyperglycemia linked to higher mortality. Ge et al.[9] reported that glycemic variability independently increased 28- and 90-day mortality. Consistently, our study showed significantly higher postoperative 24-hour glucose in non-survivors (median,

186 vs. 164 mg/dL; p=0.009). Each mg/dL increase in maximum glucose raised mortality risk by 0.4% (HR, 1.004; p=0.048).

Age and hemorrhage type are commonly reported predictors of mortality in patients undergoing DC. In a study conducted by Tang et al.[25], age and subdural hemorrhage were identified as predictors of 30-day mortality in patients undergoing DC. Several studies in the literature have similarly recognized age as a significant prognostic factor in patients requiring DC[15,17,21,28]. Likewise, Goedemans et al.[10] in a study including 204 patients who underwent craniectomy for various indications, found that aneurysmal subarachnoid hemorrhage was associated with poor prognosis. In contrast, our study did not find a statistically significant association between age and mortality. Median age was 61 years in survivors and 62 years in non-survivors (p=0.140), with both groups averaging below 65 years. Mortality varied by admission diagnosis : patients admitted with cerebral infarction had a 63% lower risk of death compared to those with parenchymal hemorrhage (HR, 0.37; 95% CI, 0.15–0.94; p=0.037), whereas the presence of epi- or subdural hematoma did not significantly affect mortality (HR, 1.09; p=0.80). These findings suggest that, while age may influence outcomes in certain cohorts, admission diagnosis appears to be a more relevant prognostic factor in our population.

While some patients undergoing DC achieve favorable functional outcomes, a substantial proportion experience poor functional outcomes, characterized by prolonged ICU stays, increased healthcare costs, and post-discharge dependency with reduced quality of life. Predicting poor functional outcomes is therefore essential for planning and management. Mulisa et al.[18] reported that a preoperative GCS ≤5 was associated with an unfavorable functional outcome (GOSE <4) in 85.7% of patients. This association between low GCS and poor outcomes has been confirmed by multiple studies[8,24,27]. In our study, preoperative GCS was closely associated with mortality, whereas postoperative GCS demonstrated a stronger correlation with functional outcomes. Each 1-point increase in postoperative GCS reduced the likelihood of severe disability by approximately 27% (OR, 0.727; 95% CI, 0.586–0.902; p=0.004), underscoring the prognostic importance of early postoperative neurological assessment.

Faleiro et al.[8] evaluated 30 surviving patients 6 months after DC performed for trauma among 78 cases. According to the GOS, 21 patients (70%) achieved favorable functional outcomes (GOS 4–5), while the remaining patients exhibited poorer recovery profiles[8]. Similarly, in our study, among 35 surviving patients with a history of trauma (29.7%), 40.0% developed severe disability, compared to 65.1% in non-trauma patients (p=0.015). Trauma history was associated with 87% lower odds of severe disability (OR, 0.130; 95% CI, 0.032–0.517; p=0.004), suggesting better functional recovery in trauma patients undergoing DC.

Our study has several limitations. Anticoagulation was predominantly initiated for prophylactic purposes in patients considered to be clinically stable and at low risk of hemorrhagic complications; thus, the observed association is more likely to reflect selection bias rather than a true protective effect. Owing to the retrospective nature of the study and the lack of detailed data regarding anticoagulation indications, timing of initiation, and pathology-specific practice variations, residual confounding cannot be excluded. Accordingly, this finding was interpreted with caution and was not emphasized in the discussion to avoid overinterpretation.

CONCLUSION

DC can be a life-saving intervention in appropriately selected patients with proper timing. However, the potential for high mortality and severe disability cannot be overlooked. Therefore, careful consideration should be given to which clinical outcomes are deemed acceptable by the patient and their family. Awareness of factors that increase the risk of postoperative morbidity and mortality is crucial for predicting potential clinical trajectories and optimizing patient selection.

AUTHORS' DECLARATION

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

Informed consent

This type of study does not require informed consent.

Author contributions

Conceptualization : AS, OE, SB, KTS; Data curation : SB, ASB, BDO, YB, FKargın, SE, FKılıç; Formal analysis : AS, FG,

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