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Determinants of reoperation after decompressive craniectomy in patients with traumatic brain injury: A comparative study

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ABSTRACT

Objectives: Reoperation after decompressive craniectomy (DC) in patients with traumatic brain injury (TBI) remains a dilemma and the risk factors are to be identified. The aim of the current study was to determine the determinants and risk factors of reoperation after DC in patients with TBI.

Patients and methods: This retrospective case-controlled study was conducted during a 4-year period from September 2013 to October 2017 in a level I trauma center affiliated with Shiraz University of Medical Sciences in southern Iran. We included all the adult (≥18 years) patients with TBI who underwent primary or secondary DC in our center during the study period. Those who underwent reoperation were compared to those who underwent DC only regarding the demographic findings, clinical features and neuroimaging findings. A univariate and multivariate logistic regression analysis was performed to determine the determining factors of reoperation.

Results: Overall we included 371 patients with mean age of 36.45 ± 14.18 years. Among the patients there were 325 (87.6%) men and 46 (12.4%) women. The reoperation in patients undergoing DC due to TBI was associated with primary DC ($p = 0.039$) and higher Marshall grade ($p = 0.027$). Those who underwent reoperation after DC for TBI had significantly higher ICU ($p = 0.007$) and hospital LOS ($p = 0.001$) and lower 6-month GOSE ($p = 0.010$). Age ($p < 0.001$), GCS ($p < 0.001$) and pupils ($p = 0.027$) were predictors of outcome in reoperation group. Reoperation in primary DC group was associated with pupil reactivity ($p = 0.002$) and number of episodes with INR above 1.5 ($p = 0.037$)

Conclusion: Reoperation after DC for TBI is associated with primary DC, and Marshall grade. The reoperation after DC is associated with worse outcome and longer ICU and hospital stay. The age, GCS and pupil reactivity are the main predictors of outcome in those with reoperation after DC for TBI.

1. Introduction

Traumatic brain injury (TBI) is a critical public health and socioeconomic problem throughout the world [1–3]. It is the leading cause of mortality and disability among young individuals in high-income countries and the most common cause of mortality and years of potential life lost (YPLL) of individuals between 18 and 44 years in developing countries [4,5]. Worldwide, the incidence of TBI is rising sharply, mainly because of increase in use of motor vehicles in low and middle income countries [1]. TBI will surpass many diseases as the major cause of death and disability by the year 2020 [6]. It is often referred to silent epidemic [7]: silent insofar as patients are not vociferous because of the invisibility of symptoms and low awareness of the chronicity of its sequelae and insofar as society in general is largely unaware of the magnitude of the problem.

Decompressive craniectomy (DC) is among the available surgical treatments in patients with TBI suffering from intracranial evacuatable pathologies (primary DC) or refractory intracranial hypertension (secondary DC); however, its role in decreasing mortality and morbidity is controversial which is subjected to large scale randomized clinical trials

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[8–11]. The outcome of DC in patients with TBI however is dependent on several factors and several complications have been associated with the procedure [12,13]. The progression of the intracranial hematomas and formation of the new intracranial pathologies such as epidural and subdural hematomas is among the important complication of the DC [14–17]. The natural course and determinants of new hematoma formation after DC has been the subject of several researches but still little is known accordingly [18,19]. Some factors such as coagulopathies determined by increased fibrinolysis and prolonged bleeding time secondary to disseminated intravascular coagulation (DIC) have been identified [12,20,21]. In addition, the reoperation or bilateral operation in TBI have been associated with poor and more unfavorable outcome [18]. The studies addressing the reoperations after DC due to new hematoma formation are limited to small cases series [18] or case reports [14,16,17,19]; while there is a lack of appropriate evidence regarding the determinants and prognosticators of these conditions. In addition, the DC is being performed in some centers in great number and knowledge of its complication and secondary hematoma formations will help us in preventing them and improving the prognosis. Thus, the aim of this study is to determine the characteristics and etiology of reoperations after DC in patients with TBI and also to investigate the determinants and the prognostic factors of these patients.

2. Materials and methods

2.1. Study population

This retrospective case controlled study was conducted during a 4-year period from September 2013 to December 2017 in Shahid Rajaei hospital, a large level I trauma center affiliated with Shiraz University of Medical Sciences, located in southern Iran which is a referral trauma center for approximately 5 million populations. We included all the adult patients within the age range of 18–70 years with TBI who underwent DC either for evacuation of an intracranial pathology (primary DC) or refractory intracranial hypertension (secondary DC). Those who developed postoperative new hematomas requiring surgical evacuation were considered as the study group while those who did not underwent a second operation were considered as controls. All the patients were consecutively included in the study and survived for at least 48 h after the surgery. We have excluded those with multiple craniofacial injuries, those with multi-organ and systemic severe injuries and those who were brain death on admission. We excluded those who died due to systemic injuries within 48 h after surgery. Those with history of previous cranial surgeries and those on anticoagulants and antiplatelet agents before the injury were also excluded from the study. The study protocol was approved by the medical ethics committee and the institutional review board (IRB) of Shiraz University of Medical Sciences (IR.SUMS.REC.1396.523). Due to the retrospective nature of the study, no informed written consents were required.

2.2. Study protocol

All the patients were initially evaluated by a neurosurgery resident and the findings were recorded in the data gathering forms available in the emergency room. We recorded the demographics (age, and gender), mechanism of injury, time between the injury and surgery, the baseline clinical status (GCS after resuscitation, pupil reactivity, lateralizing signs) and the baseline laboratory information including the fibrinogen level, platelets and international normalized ratio (INR). We also recorded the initial CT-scan findings based on the Marshal classification [22]. The type and location of primary and contralateral hematoma, presence of a skull fracture and brain swelling overlying the contralateral site were also reviewed. The midline shift was also recorded on the preoperative CT-scan. All the patients with prolonged INR (value ≥ 1.5) were treated with appropriate amount of fresh frozen plasma (FFP) before reoperation.

2.3. Surgery

All the patients underwent decompressive craniectomy surgery using the standard technique described elsewhere [13]. The operations were performed by the neurosurgery residents and attending physicians according to the hospital protocols using a traumatic skin incision. The bone flap was packed in a sterile plastic bag and restored in standard bone bank. The hematoma was evacuated and the dura was primarily repaired in those with intracranial hematomas without significant brain swelling. But in those who had significant brain swelling or were operated due to intractable intracranial hypertension, secondary dural repair with pericardial fascia or synthetic dural substitutes were performed. All the patients were admitted to intensive care unit (ICU) and received conservative management. Serial postoperative CT-scan and neurological examination was performed and the need for operation was determined according to changes in neurological examination or CT-scan. The indications for reoperation included any extra-axial hematoma thicker than 15 mm and/or midline shift of more than 5 mm. The location of the new hematomas was recorded as the ipsilateral and contralateral site. The etiology of the reoperation was also determined.

2.4. Follow-up and outcome

The number of operations performed after the decompressive craniectomy and the type of operation was recorded. All the patients received conservative therapy according to 4th edition of the guidelines for management of severe traumatic brain injury [23]. Those with proposed low GCS underwent early tracheostomy within 7 days of the injury [24]. Patients were given a helmet and instructions for avoiding the pressure ulcers. We followed the patients in an outpatient basis based on a monthly schedule. Cranioplasty was performed 6 weeks to 3 months after the operation based on the resolution of brain swelling and CT-scan findings utilizing a proposed technique described elsewhere [25]. The outcome of the patients was determined using the Glasgow outcome scale extended (GOSE) at 6 month follow-up. We also recorded the ICU and hospital length of stay (LOS). Unfavorable outcome was defined as dead or persistent vegetative state (PVS) and favorable outcome was defined as otherwise.

2.5. Statistical analysis

All the statistical analysis was performed using the statistical package for social sciences (SPSS Inc., Chicago, Illinois, USA) version 22.0. data are presented as mean ± SD as appropriate. The parametric variables with normal distribution were compared using independent *t*-test while the proportions were compared using the chi-square test. Parametric variables without normal distribution were compared using Mann-Whitney *U* test The odds ratio (OR) with 95% confidence interval (CI) was also reported for non-parametric comparisons. We ran a univariate analysis to determine the risk factors for reoperation in our series. We also performed a multivariate logistic regression analysis in order to compensate for confounders and confirm the results. A two-sided p-value of less than 0.05 was considered statistically significant.

3. Results

Overall, we included 317 adult patients who underwent primary or secondary DC in our center during the study period with mean age of 36.45 ± 14.18 (ranging from 18–70) years. Among the patients there were 325 (87.6%) men and 46 (12.4%) women. The frequency of reoperation was 55 (14.8%) among which 22 (5.9%) patient underwent ipsilateral surgeries while 33 (8.9%) underwent contralateral reoperation. The reoperation etiology was EDH in 25 (45.5%) patients and contralateral DC in 23 (41.8%). The EDH was in the site of previous DC in 16 (29.1%) patients while it was in a new location in 9 (16.3%) patients (6 contralateral frontoparietal and 3 posterior fossa EDH). The

Table 1
The baseline characteristics of the 371 patients who underwent decompressive craniectomy in our center during the study period.

Variable	Value
Age (years)	36.45 ± 14.18
Gender	
Men (%)	325 (87.6%)
Women (%)	46 (12.4%)
Mechanism of injury	
Motor-vehicle accident (%)	283 (72.3%)
Fall (%)	69 (18.6%)
Assault (%)	19 (9.1%)
Type of decompressive craniectomy	
Primary (%)	329 (88.7%)
Secondary (%)	42 (11.3%)
Marshall Classification	
I (%)	11 (2.9%)
II (%)	16 (4.3%)
III (%)	18 (4.8%)
IV (%)	29 (7.9%)
V (%)	297 (80.1%)
Reoperation	
Ipsilateral (%)	22 (5.9%)
Contralateral (%)	33 (8.9%)
Reoperation type	
Epidural hematoma (%)	25 (45.5%)
Decompressive Craniectomy (%)	23 (41.8%)
Intraparenchymal Hemorrhage (%)	7 (12.7%)
GCS Score	7.75 ± 3.48
Severe (%)	271 (73.1%)
Moderate (%)	84 (22.6%)
Mild (%)	16 (4.3%)
Pupil reactivity	
Reactive (%)	247 (66.6%)
Anisocoric (%)	88 (23.7%)
Fixed (%)	36 (9.7%)
Intracranial hematoma	
Cerebral contusions (%)	112(30.2%)
Subdural hematoma (%)	94 (25.3%)
Subarachnoid hemorrhage (%)	86 (23.2%)
Intraparenchymal hematoma (%)	49 (13.2%)
Epidural hematoma (%)	30 (8.1%)

GCS: Glasgow Coma Scale.

contralateral DC was performed due to intractable intracranial hypertension in 14 (25.4%) patients and acute SDH in 9 (16.3%). In 7 (12.7%) patients intraparenchymal (IPH) was evacuated in the site of preoperative deep cerebral contusion. The baseline characteristics of the patients are summarized in Table 1.

We demonstrated that the reoperation in patients undergoing DC due to TBI was associated with primary DC [*p = 0.039;* OR (95%CI): 2.18 (1.23–3.89)] and higher Marshall grade (*p = 0.027*). There was no association between the age (*p = 0.687*), gender (*p = 0.315*), mechanism of injury (*p = 0.721*), GCS (*p = 0.206*), pupil reactivity (*p = 0.080*) and the reoperation after DC for TBI (Table 2). Neither on admission INR (*p = 0.357*) nor the number of episodes with INR > 1.5 were associated with reoperation after DC (*p = 0.062*). We did not find any difference between those undergoing reoperation regarding the on admission level of fibrinogen (*p = 0.358*) and the rate of fibrinogen level ≥ 150 mg/dL (*p = 0.095*). Those who underwent reoperation after DC for TBI had significantly longer ICU (*p = 0.007*) and hospital LOS (*p = 0.001*). The outcome of the patients based on the 6-month GOSE was found to be significantly lower in those who underwent reoperation (*p = 0.010*). The reoperation was associated with higher rates of mortality (43.6% *vs.* 29.1%), vegetative state (10.9% vs. 4.9%), and low severe disability (9.1% vs. 5.3%). Reoperation was also associated with lower rate of upper good recovery (16.3% *vs.* 24.7%). The outcome measures are summarized in Table 3.

The univariate analysis in those who underwent reoperation after DC for TBI revealed that unfavorable outcome was associated with higher age (*p < 0.001*), higher rate of severe TBI (*p < 0.001*), lower

Table 2
Univariate analysis for determining the determinant factors of reoperation in 371 patients with traumatic brain injury undergoing decompressive craniectomy.

	No reoperation (n = 316)	Reoperation (n = 55)	p-value
Age (years)	37.22 ± 13.52	36.14 ± 18.62	0.687
Gender			
Men (%)	274 (86.7%)	51 (92.7%)	0.315
Women (%)	42 (13.3%)	4 (7.3%)	
Mechanism of injury			
Motor-vehicle accident (%)	238 (75.3%)	45 (81.8%)	0.721
Fall (%)	63 (19.9%)	6 (10.9%)	
Assault (%)	15 (4.8%)	4 (7.3%)	
Type of decompressive craniectomy			
Primary (%)	278 (87.9%)	51 (92.7%)	**0.039**
Secondary (%)	38 (12.1%)	4 (7.3%)	
Marshall Classification			
I (%)	10 (3.1%)	1 (1.9%)	**0.027**
II (%)	14 (4.4%)	2 (3.6%)	
III (%)	17 (5.4%)	1 (1.9%)	
IV (%)	26 (8.2%)	3 (5.4%)	
V (%)	249 (78.9%)	48 (87.2%)	
GCS	7.34 ± 2.47	7.86 ± 2.8	0.206
Severe (%)	233 (73.7%)	38 (69.1%)	0.159
Moderate (%)	68 (21.5%)	16 (29.0%)	
Mild (%)	15 (4.8%)	1 (1.9%)	
Pupil reactivity			
Reactive (%)	219 (66.6%)	28 (50.9%)	0.080
Anisocoric (%)	80 (23.7%)	8 (14.5%)	
Fixed (%)	17 (9.7%)	19 (34.6%)	
Intracranial hematoma			
Cerebral contusions (%)	93 (29.4%)	19 (34.5%)	0.063
Subdural hematoma (%)	82 (25.9%)	12 (21.8%)	
Subarachnoid hemorrhage (%)	79 (25.0%)	7 (12.7%)	
Intraparenchymal hematoma (%)	41 (12.9%)	8 (14.5%)	
Epidural hematoma (%)	21 (6.8%)	9 (16.5%)	
Laboratory			
INR on admission	1.29 ± 1.42	1.44 ± 1.55	0.357
No. of episodes with INR > 1.5	2.34 ± 3.47	2.46 ± 4.12	0.062
Platelet ($\times 10^9$/L)	219.6 ± 27.4	227.1 ± 84.6	0.174
Fibrinogen level (mg/dL)	139.41 ± 96.7	131.59 ± 96.3	0.358
< 150 mg/dL	177 (56.1%)	34 (61.8%)	0.095
≥150 mg/dL	139 (43.9%)	21 (38.2%)	

GCS: Glasgow Coma Scale; **INR:** International Normalized Ratio.

GCS (*p < 0.001*) and higher rate of fixed pupils (*p = 0.027*). Other parameters including the Marshall grade (*p = 0.823*) and the laboratory markers of coagulation were not found to be associated with outcome in these patients (Table 4). We also ran a multivariate regression model and found that age (*p = 0.018*), GCS (*p < 0.001*) and pupil reactivity (*p < 0.001*) remained significant determinants of outcome in those with reoperation after DC. Table 5 summarizes the determinants of reoperation in those who underwent primary DC. We found that the reoperation in primary DC was associated with pupil reactivity (*p = 0.002*) and number of episodes of INR above 1.5 (*p = 0.037*). Other factors did not predict the reoperation in primary DC. We could not calculate these determinants in those undergoing secondary DC as there were limited number of patients in this subgroup (only 4 patients).

4. Discussion

In this case-controlled study, we have evaluated the rate and risk factors of the reoperation after DC for TBI. In this large series we found that reoperation was encountered in 16.2% of all procedures and is associated with increased length of stay in both ICU and hospital as well

Table 3
The 6-month outcome of 371 patients with traumatic brain injury who underwent decompressive craniectomy in two study groups.

	No reoperation (n = 316)	Reoperation (n = 55)	p-value
Hospital LOS (days)	26.3 ± 8.7	39.6 ± 9.6	**0.001**
ICU LOS (days)	20.1 ± 6.8	28.6 ± 8.4	**0.007**
GOSE			
Dead (%)	105 (29.1%)	24 (43.6%)	**0.010**
Vegetative State (%)	18 (4.9%)	6 (10.9%)	
Low Severe Disability (%)	19 (5.3%)	5 (9.1%)	
Upper Severe Disability (%)	27 (7.5%)	4 (7.3%)	
Low Moderate Disability (%)	16 (4.4%)	1 (1.9%)	
Upper Moderate Disability (%)	46 (12.7%)	2 (3.6%)	
Low Good Recovery (%)	41 (11.4%)	4 (7.3%)	
Upper Good Recovery (%)	99 (24.7%)	9 (16.3%)	

GOSE: Glasgow Outcome Scale Extended; **ICU:** Intensive Care Unit; **LOS:** Length of Stay.

Table 4
The predictors of the unfavorable outcome in 55 patients with traumatic brain injury undergoing decompressive craniectomy and reoperation.

	Favorable (n = 16)	Unfavorable (n = 39)	p-value
Age (years)	22.91 ± 10.63	35.10 ± 9.74	< **0.001**
Gender			
Men (%)	15 (93.7%)	36 (92.3%)	0.972
Women (%)	1 (6.3%)	3 (7.7%)	
Mechanism of injury			
Motor-vehicle accident (%)	12 (75.0%)	33 (84.5%)	0.179
Fall (%)	2 (12.5%)	4 (10.3%)	
Assault (%)	2 (12.5%)	2 (5.2%)	
Type of decompressive craniectomy			
Primary (%)	14 (87.5%)	37 (94.8%)	0.522
Secondary (%)	2 (12.5%)	2 (5.2%)	
Marshall Classification			
I (%)	0 (0.0%)	1 (2.5%)	0.823
II (%)	1 (6.25%)	1 (2.5%)	
III (%)	0 (0.0%)	1 (2.5%)	
IV (%)	1 (6.25%)	2 (5.2%)	
V (%)	14 (87.5%)	34 (87.3%)	
GCS	7.91 ± 3.1	5.98 ± 4.3	< **0.001**
Severe (%)	3 (18.7%)	35 (89.7%)	< **0.001**
Moderate (%)	12 (75.0%)	4 (10.3%)	
Mild (%)	1 (6.3%)	0 (0.0%)	
Pupil reactivity			
Reactive (%)	8 (50.0%)	20 (51.3%)	**0.027**
Anisocoric (%)	2 (12.5%)	6 (15.4%)	
Fixed (%)	6 (37.5%)	13 (33.3%)	
Intracranial hematoma			
Cerebral contusions (%)	6 (37.5%)	13 (33.3%)	0.061
Subdural hematoma (%)	1 (6.3%)	11 (28.2%)	
Subarachnoid hemorrhage (%)	1 (6.3%)	6 (15.4%)	
Intraparenchymal hematoma (%)	3 (18.6%)	5 (12.8%)	
Epidural hematoma (%)	5 (31.3%)	4 (10.3%)	
Laboratory			
INR on admission	1.52 ± 1.32	1.59 ± 1.19	0.677
No. of episodes with INR > 1.5	2.52 ± 3.87	3.75 ± 5.6	0.086
Platelet ($\times 10^9$/L)	226.3 ± 80.1	268.1 ± 96.4	0.144
Fibrinogen level (mg/dL)	133.01 ± 78.4	132.7 ± 66.7	0.920
< 150 mg/dL	9 (56.3%)	26 (66.6%)	0.751
≥150 mg/dL	7 (43.7%)	13 (33.4%)	

Table 5
Univariate analysis for determining the determinant factors of reoperation in 329 patients with traumatic brain injury undergoing primary decompressive craniectomy.

	No reoperation (n = 278)	Reoperation (n = 51)	p-value
Age (years)	36.87 ± 14.08	38.11 ± 16.54	0.173
Gender			
Men (%)	243 (87.4%)	46 (90.2%)	0.408
Women (%)	35 (12.6%)	5 (9.8%)	
Mechanism of injury			
Motor-vehicle accident (%)	214 (76.9%)	45 (88.2%)	0.082
Fall (%)	43 (15.4%)	4 (7.8%)	
Assault (%)	21 (7.7%)	2 (4.0%)	
Marshall Classification			
I (%)	1 (0.3%)	1 (1.9%)	0.099
II (%)	3 (1.1%)	2 (3.9%)	
III (%)	4 (1.4%)	1 (1.9%)	
IV (%)	6 (2.1%)	2 (3.9%)	
V (%)	237 (95.1%)	45 (88.4%)	
GCS	7.47 ± 2.64	7.51 ± 3.52	0.879
Severe (%)	219 (78.7%)	36 (70.5%)	0.342
Moderate (%)	46 (16.5%)	14 (27.4%)	
Mild (%)	13 (4.8%)	1 (2.1%)	
Pupil reactivity			
Reactive (%)	193 (69.4%)	25 (49.1%)	**0.002**
Anisocoric (%)	74 (26.6%)	8 (15.6%)	
Fixed (%)	11 (4.0%)	18 (35.3%)	
Laboratory			
INR on admission	1.37 ± 1.08	1.21 ± 1.65	0.207
No. of episodes with INR > 1.5	1.89 ± 4.26	2.96 ± 3.45	**0.037**
Platelet ($\times 10^9$/L)	225.1 ± 39.4	222.4 ± 79.8	0.189
Fibrinogen level (mg/dL)	146.7 ± 74.6	137.9 ± 81.5	0.079
< 150 mg/dL	154 (55.4%)	32 (62.7%)	0.215
≥150 mg/dL	124 (44.6%)	19 (37.3%)	

GCS: Glasgow Coma Scale; **INR:** International Normalized Ratio.

as lower outcome measures according to GOSE. The predictors of reoperation after DC included the primary DC, and higher Marshall grade of CT-scan. The predictors of outcome in those who underwent reoperation after DC for TBI were age, GCS and pupil reactivity. We found that number of episodes with INR above 1.5 was associated with reoperation in those with undergoing primary DC. The results of the current study demonstrate that the reoperation after DC cannot be predicted according to the laboratory markers of coagulation and only some clinical factors contribute to the need for reoperation. However, the reoperation declines the outcome significantly and is associated with higher hospital expenses according to longer hospital and ICU stay. The predictors of outcome are however, similar to those in patients with TBI undergoing DC which includes age, GCS and pupil reactivity. This is the largest series of the patients with reoperation after DC due to TBI and sheds light on importance of the predictors. According to the recently published randomized clinical trials on role of DC in patients with severe TBI [10,11,26], the secondary DC has limited efficacy on outcome of these patients while the primary DC is still the recommended methods of treatment of patients with intracranial evacuatable hematomas. In our series, most of the patients have undergone primary DC as most of the patients presented with intracranial hematomas.

The reoperation after DC in TBI has been less studied previously and thus the information accordingly is scarce. Recently, Lillemäe et al. [27] studied the occurrence of postoperative hematoma (POH) after neurosurgery according to procedure type. They found that the rate of overall POH was 0.6% after craniotomy, 0% after shunting procedure, 1.1% after spine surgery, and 0% after implantation of a spinal cord stimulator. Craniotomy types with higher POH incidence were decompressive craniectomy (7.9%), cranioplasty (3.6%), bypass surgery (1.7%), and EDH evacuation (1.6%). Hypertension was a significant factor for POH and the occurrence of POH was associated with poor outcome [27]. In 2003, Matsuno et al. [18] reported the rates and predictive factors of contralateral reoperation after DC in patients with TBI. Among 88 patients with acute SDH who were surgically treated, they encountered and studied 5 patients who developed contralateral acute EDH or SDH (5.7%). All the patients were men and suffered from severe TBI (GCS < 8). All patients underwent consecutive surgical procedures for acute SDH and contralateral SDH or EDH. Most of the patients (3 out of 5) had unfavorable outcome [18]. In the same way, we found that reoperation was associated with poor outcome and higher Marshall grading in initial CT scan.

Other studies in the field include small series or case reports [14–17]. Shen et al. [15] demonstrated that lower preoperative GCS score is an independent risk factor for prognosis of contralateral acute EDH after acute SDH. These results are in line with ours that the GCS is an independent factors of outcome in reoperation after DC for TBI. We also found pupil reactivity and age to be other independent prognosticators of outcome. Tomycz et al. [17] reported a case of acute SDH after evacuation of the same pathology in an old age lady. They believed that early decompression and evacuation of hematoma was responsible for the hematoma [17]. Two other case reports, described intraoperative acute and severe swelling of the brain after DC in severe TBI and immediate CT-scan revealed contralateral SDH which was treated by the contralateral DC [16,19].

Several factors are responsible for hematoma formation and reoperation after DC in patients with TBI. The most common cause of reoperation which was found in our series is the technical problems in hemostasis of bone, muscle, soft tissue and scalp which leads to formation of the EDH and less commonly SDH. Inappropriate hemostasis of the extradural structures such as scalp, temporalis muscle, bone edges and the dura will led to formation of EDH while inappropriate hemostasis of the cortical contusions or ruptures vessels leads to formation of SDH [28]. This is evident by the fact that most of the patients who underwent primary DC developed postoperative hematomas requiring surgical evacuation. Those undergoing secondary DC due to malignant intracranial hypertension had lower rates of reoperation probably due to tamponade effects of the brain on the surrounding tissue. Thus, we have analyzed the primary and secondary DC separately and came to the decision that reoperation in primary DC was associated with pupil reactivity and number of episodes with INR above 1.5. We found that EDH was the most common postoperative hematoma requiring surgical evacuation and ipsilateral EDH was the most common type probably due to inappropriate hemostasis. The second most common cause of new hematomas after DC in TBI is the hemorrhage from the skull fractures of injured contralateral middle meningeal artery [14,15,29]. This probably due to compression of the injured contralateral middle meningeal artery with the pressure of the ipsilateral hematoma. As soon as the hematoma was evacuated and the compression is resolved, the artery begins to bleed into the contralateral epidural space [15,29]. The other mechanism for formation of postoperative SDH could be the cortical venous injury or sagging. We recorded 8 cases of contralateral SDH immediately after the operation. The immediate and rapid evacuation of the ipsilateral hematoma can result in sagging and cortical venous injury of the contralateral side leading to formation of SDH [15–17,19]. This has been previously described elsewhere and it is recommended that early decompression of the SDH be performed with a burr-hole craniotomy, immediately followed by a decompressive craniectomy [14,18]. This strategy provides gradual decompression, while advancing the initial surgical time and preventing the sudden decreased tamponade effect. As such, it may help decrease the risk of contralateral SDH associated with decompression. The last mechanism of hematoma formation is considered to be expansion of the deep seated contusions after decompression leading to formation of IPH. We have recorded 9 cases of IPH after DC in TBI. Juralti et al. [12] demonstrated that hemorrhagic progression of brain contusions (HPC)occurred the first 6 h was in 43.5% of the patients. They reported that elevated D-dimer level (≥10,000 μg/L), HPC, and initial brain contusions ≥3 cm were associated with poor outcome. They also found that early HPC was associated with coagulopathy measured by fibrinogen level and D-dimer [12]. Although we did not find any relation between the initial markers of coagulopathy (fibrinogen level, INR and platelet count) and the reoperation, several other studies have emphasized on the issue [20,21,30].

We note some limitations to our study. First, the retrospective nature of the study limited us from recording several important factors such as the initial Rotterdam scores, the parametric amount of midline shift, and the skull fracture frequency and types. These factors could also affect the rate of reoperation in different ways. For example, the contralateral or posterior fossa fractures could be responsible for the EDHs far from the primary site of DC [29]. In addition, we could not comment on the role of infection and rate of meningitis and ventriculitis in these series because retrospective records limited us from obtaining the exact result of the cultures and antibiograms. These factors can affect the outcome significantly [31]. The other limitation was that we could not retrieve and record the exact parametric values for serial measurements of fibrinogen level, INR and platelet counts. All the reported values are on admission ones. The cerebral coagulopathy due to TBI occurs hours after the injury [32], while most of our patients were transferred within 4 h of the injury. Thus, the initial laboratory markers of the coagulation would not be a reliable marker. We have also excluded those who died within 48 h of injury due to the fact that the main cause of death during first 48 h of trauma is extra-cranial injuries which would have biased the evaluation of DC patients. In addition, we did not want to evaluate the prognosis of DC in our study and the main goal was to determine the factors contributing to reoperation in patients with DC. Taking all these together, this is the largest series of patients with reoperation after DC for TBI and sheds light on important aspects of the issue. Further studies are recommended by taking into consideration more clinical and laboratory parameters.

In conclusion, reoperation after DC for TBI is associated with primary DC and Marshall grade. The reoperation after DC is associated with worse outcome and longer ICU and hospital stay. The age, GCS and pupil reactivity are the main predictors of outcome in those with reoperation after DC for TBI. The laboratory markers of coagulation are nor associated with the outcome, neither with the need for reoperation in these patients.

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