Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2023.180056.2699 Volume 29, Issue 3, May 2023
Manuscript ID ZUMJ-2212-2699 (R1)
DOI 10.21608/zumj.2023.180056.2699

ORIGNAL ARTICLE

Surgical Management of Traumatic Acute Subdural Hematoma

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Submit Date 2022-12-10 22:56:24
Revise Date 2022-12-14 01:16:31
Accept Date 2023-01-01

ABSTRACT

Background: Traumatic acute subdural hematoma (ASDH) remains one of the most fatal traumatic brain injuries, despite recent advances in neurosurgical management. Nevertheless, the optimum surgical approach is still unclear. Hinged craniotomy (HC) and decompressive craniectomy (DC) are the most commonly adopted surgical techniques. The aim of this study is to evaluate the suitability, indications, outcome, and complications of HC and DC.

Methods: A retrospective case series including 45 patients with ASDH with Glasgow Coma Scale (GCS) 4-12. HC was performed (group I, 16 patients) when the brain was relaxed. If the brain was not relaxed significantly, DC was preferred in 29 patients (intermediate category, group II, 13 patients) or bulging brain (group III, 16 patients). The clinical, radiological, and surgical data and complications were analyzed, and Glasgow Outcome Scale (GOS) score was documented after 6 months of follow-up. The poor outcome included scores 1-3, while functional survivors included scores 4 and 5.

Results: The overall functional survivor rate was 11/45 (24.4%), while poor outcome and mortality rates were (75.6%) and (48.9%) respectively. The functional survivors across the 3 groups were comparable. There was no significant difference regarding the outcome between HC (5/16) and DC (6/29) ($p > 0.05$).

Conclusions: Both HC and DC were viable surgical options with no difference in the outcomes and complications following the evacuation of traumatic ASDH for relaxed and bulging brains respectively. Patients with intermediate brain condition are managed according to the surgeon's preference and facility equipment, especially the availability of ICP monitoring.

Keywords: Acute subdural hematoma; Decompressive craniectomy; Hinged craniotomy; Intracranial pressure; Massive brain odema

INTRODUCTION

Acute subdural hematoma (ASDH) represents about 11% of all traumatic brain injuries (TBI) and about 20-33% of severe TBI [1, 2]. The mechanism of ASDH is usually high-speed impact head injury causing brain tissue acceleration/ deceleration relative to the fixed dura and bony skull resulting in shearing of bridging/ cortical veins and polar contusions affecting the frontal, temporal and occipital lobes [3].

In the majority of cases, ASDH is associated with other severe intracranial injuries. Nevertheless, early brain computed tomography scan (CT) scan may underestimate the real associated parenchymal injury. Servadi et al. [4] reported a surge of parenchymal injury between the first and follow-up CT scan from 27% to 51% of cases of ASDH. The ASDH acts as an acute space-occupying lesion with associated co-existing parenchymal contusions, intracerebral hematoma, surrounding oedema and focal/ generalized ischemia resulting in increased intracranial pressure (ICP) [5, 6].

Traumatic ASDH may be managed conservatively or surgically. The goal of surgery is to prevent secondary brain injury cascade whenever there are manifestations of significant mass effect or intracranial hypertension resulting in progressive neurologic deterioration [1,2,5]. Multiple surgical strategies were advocated for management of ASDH including burr hole trephination, craniotomy, or hinged craniotomy (replacement of the bone flap securely or loosely respectively), subtemporal decompressive craniectomy or decompressive craniectomy (DC) involving

removal of extensive bone flap to increase the available space for edematous brain tissue expansion as a salvage procedure for uncontrollable ICP [1,5,6-9].

Because of its complex pathophysiology and commonly related intra and extracranial significant injuries, ASDH is still considered one of the most lethal TBI, despite recent advances in the patient transfer, diagnostic modalities, sophisticated neurointensive care and neurosurgical management [2,4,6]. Regrettably, the mortality and poor outcome rates did not differ significantly from the early CT published reports till up-to-date studies from 50-80%, while functional recovery ranged from 20-40% [1,2,6-13].

It is still unclear which surgical technique is the optimal treatment strategy for traumatic ASDH. The aim of this work is to assess the suitability, indications, outcome and complications of hinged craniotomy and DC, the most commonly used surgical techniques for ASDH in moderate and severe TBI.

METHODS

A retrospective review of 45 patients with traumatic ASDH operated upon at the Departments of Neurosurgery, Cairo University and Beni Souf University during the period from March 2019- December 2021.

Patients (age ≥ 18 years) with GCS scores 4 to 12, who were operated on for a unilateral ASDH (thickness >10mm or midline shift > 5mm) within 1st 4 hours of trauma were included in this study. Patients with a unilateral dilated fixed pupil or presented with GCS (4-8) on admission with documented deterioration of ≥2 points were also included regardless of hematoma size. Patients younger than 18 years, vitally unstable, operated on after 4 hours from injury, GCS 3 or 13-15, bilateral hematomas were excluded.

Preoperative preparation

All patients were admitted to the emergency department and received primary care according to guidelines of advanced trauma life support (ATLS), and underwent urgent CT brain, ICU admission, rapid cerebral dehydrating measures and prepared for urgent surgical evacuating through first 4 hours.

All patients were examined for vital signs, consciousness level, pupils' size, equality and reactivity and signs of brain-stem dysfunction, and motor power. Detailed medical history including mode of trauma and time consumed before admission was documented.

Operative procedure:

Under general anesthesia, the patient was in supine position with ipsilateral shoulder support and head elevated and rested on a horseshoe head-holder and turned to the contralateral side, yet not compressing the contralateral internal jugular vein. A large question mark skin incision was designed (1 cm from midline), and the scalp and temporalis muscle were elevated as a single myocutaneous scalp flap and retracted anteriorly till the root of the zygoma was visible and the ipsilateral keyhole was also exposed.

A large frontotempro-parietal craniotomy flap was elevated. The bone flap should be at least 15 x 12 cm (anteroposterior craniocaudal directions) to ensure sufficient decompression to avoid brain herniation and squeezing of the cerebral cortex at the craniotomy edges. In addition, the squamous temporal bone was rongeured as close as possible to the floor of the middle fossa to accommodate the swollen temporal lobe. Five burr holes were created: one above the root of the zygoma, one at the keyhole, and one at the coronal suture approximately 2.5 cm lateral to midline, which was important for the placement of external ventricular drain (if required), one at the parietal boss and the last one along the floor of the middle fossa posterior to the petrous bone.

Then, the dura was opened, the hematoma was evacuated by irrigation and the bleeding points were controlled by surgicel and gel foam. When hemostasis was satisfactory, the dura was approximated loosely by the designed pericranium (Figure 1).

The bone flap was hinged loosely via sutures (after drilling 4-5 points) if the brain was relaxed significantly (HC group I) or removed (DC group II + III) and stored subcutaneously at the patient's abdominal wall or at the bone bank if the brain was gray zone condition (group II) or bulging (group III). An epidural or subgalial drain was placed for 48-72 hours.

Postoperative care:

Post-operative admission at neurosurgical ICU with complete sedation on barbiturate for 1st 24 - 48 hours after surgery with hyperventilation then gradual sedation withdrawal to assess GCS. Maximum dehydration (mannitol 20% (0.5 g/kg/4 hrs.), phenytoin for anticonvulsive treatment loading dose (15-20 mg. /kg/ on 100c saline for 15 min, then a maintenance dose of phenytoin 5mg /kg/ 24, fluid rate 100cc/h, FFP, antibiotic analgesics were given.

A follow-up CT brain after 24 hours were ordered for documentation of the hematoma evacuation and improvement of the midline shift, then routine follow-up CT scan every two-three days in the first two weeks.

All patients were monitored for 6 months after surgery. Outcomes were assessed using the 5-

point Glasgow Outcome Scale (GOS) [14]. In brief, 1 death; 2 Persistent vegetative state; 3 severe disability with a permanent need for help with daily living activities; 4 moderate disabilities without need for help in daily living activities and employment is possible but requires special equipment; 5 good recovery. Patients with grades 4 and 5 were defined as functional survivors, while patients with grades 1-3 were recorded as having poor outcomes.

Informed consent and ethics committee approval:

This clinical study was approved by the Research Ethics Committee (REC) of The Neurosurgery Department, Faculty of Medicine, Beni Souf University in January 2019. Informed consent for the procedure signed by all patients, first relative. All procedures involving humans were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments.

Statistical analysis

Analysis of data was performed using SPSS v. 25 (Statistical Package for Social science) for Windows, description of variables was presented for quantitative variables as mean ±standard deviation (SD), while for qualitative variables as numbers (No.) and percentage (%). Comparison between quantitative variables was performed by independent sample t-test and one-way ANOVA test for normally distributed variables. While comparison between qualitative variables was done by Qui-Square test. The significance of the results was assessed in the form of a P-value that was significant when P-value ≤ 0.05.

RESULTS

This study included 45 patients who underwent evacuation of traumatic ASDH. HC was performed in 16 patients (35.6%) with CT scan confirming the minimal parenchymal injury and intraoperative significant brain relaxation. DC was done in 29 patients (64.4%) when the brain was not relaxed significantly (either bulging beyond the inner table of the skull (group III, 16 patients) or intermediate category (group II, 13 patients) (Table 1). The bone flap was stored at the subcutaneous abdominal wall in 20 patients or the bone bank in 9 patients.

There was male predominance with male/ female = 2/1 (30/15). The mean age was 31.1± 11.4 years (mean ± SD) ranging from 18- 65 y. The most common mechanism of injury was motor vehicle accident in 31 patients (68.9%), followed by falls from heights in 10 patients (22.2%), and assaults in 4 patients (8.9 %). There was no significant difference in the age, sex, mechanisms of injury, pupillary condition, and GCS among the studied groups.

Regarding the CT findings, the mean ASDH thickness was higher in the DC category 12mm than in the HC category 9.25 mm (P= .016). Group III (bulging brain) had the greatest thickness among the 3 groups (P = 0.037). Although mean midline shift, complete basal cistern obliteration and associated parenchymal injury was higher in the DC group compared to the HC group, it was not statistically significant (p > 0.05).

The difference in operative duration for the two techniques was not statistically significant (183 ± 24 minutes in the HC group, 179 ± 26 minutes in the DC group; p > 0.05).

The overall functional survivor rate was 11/45 (24.4%), while poor outcome and mortality rates were (34/45 =75.6%) and (22/45=48.9% respectively. The functional survival rate was comparable between HC (31.3%) and DC (20.7%) groups (p > 0.05). Similarly, there was no difference among the functional survivors across the 3 groups (p > 0.05) (Figure 2).

Postoperative complications did not differ between HC (5/16= 31.3%) and DC (6/29= 20.7%) groups (p > 0.05). The intracranial complications included newly developed intracerebral hematoma 3 patients (HC 1/16, DC 2/29), intracerebral hematoma enlargement 5 patients (HC 2/16, DC 3/29), postoperative extradural hematoma 2 patients (HC 1/16, DC 1/29), and sinking of the skull bone (one patient after HC).

Table 1: The clinical and radiological features and the outcome of the patients with ASDH among study groups

	GI HC (relaxed brain) N=16	GII intermediate N=13	GIII Bulging brain N=16	GII+GIII (DC) N=29	P[a]	P[b]
Clinical data:						
Age	32 ±9.3	28.4±10.9	31±11.5	29.8±11.1	0.12	0.166
Mean GCS (range)	8.4 ± 2 (5-12)	8±1.9 (5-11)	7.3±1.6 (4-9)	7.6±1.7	0.197	0.268
Anisocoria	4 (25%)	7 (53.8%)	8 (50%)	15 (51.7%)	0.082	0.216

	GI HC (relaxed brain) N=16	GII intermediate N=13	GIII Bulging brain N=16	GII+GIII (DC) N=29	P^a	P^b
CT findings:						
Mean ASDH thickness mm	9.25±3.2	11.3±2.6	12.6±4.4	12.0±3.7	0.016*	0.037*
Mean midline shift mm	4.8±2	5.6±2.1	5.7±2.1	5.7±2.1	0.187	0.421
Basal cistern obliteration Mild Partial Complete	 6 (37.5%) 7 (43.8%) 3 (18.8%)	 3 (23.1%) 6 (46.2%) 4 (30.8%)	 3 (18.8%) 6 (37.5%) 7 (43.8%)	 6 (20.7%) 12 (41.4%) 11 (37.9%)	 0.313	 0.576
Associated ICH/contusion	3 (18.8%)	5 (38.5%)	8 (50%)	13 (44.8%)	0.176	0.08
GOS (Glasgow outcome scale):						
Functional survivors (GOS 4,5)	5 (31.3%)	3 (23.1%)	3 (18.8%)	6 (20.7%)	0.430	0.706
Poor outcome (GOS 1,2,3)	11 (68.8%)	10 (76.9%)	13 (81.3%)	23 (79.3%)	0.430	0.706
Death	7 (43.8%)	7 (53.8%)	8 (50.0%)	15 (51.7%)	0.608	0.859

DC= Decompressive craniectomy / HC= Hinged craniotomy, GOS= Glasgow outcome scale / P^a value for HC vs DC /P^b value for HC vs Intermediate vs Bulging/*p value<0.05 is considered significant

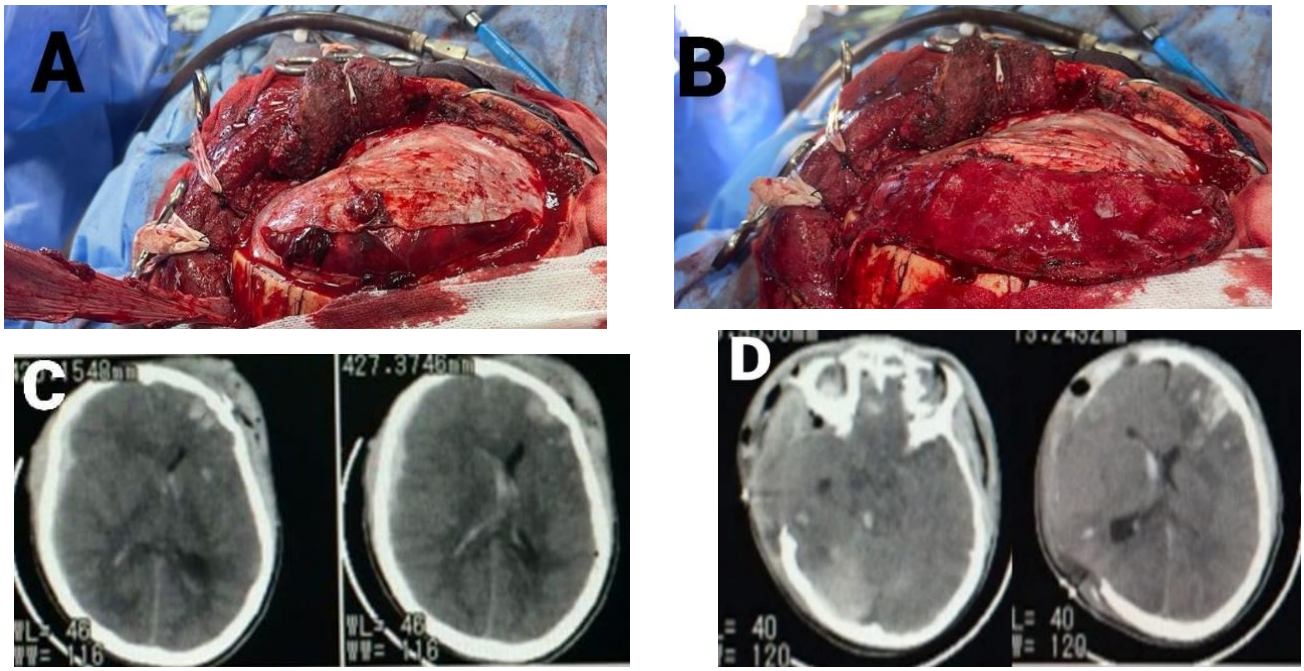

Figure 1: A) and B) Intraoperative photo of right fronto-parietal ASDH with bulging brain after evacuation of the hematoma and DC. Note the pericranium flap was used for augmentative duroplasty. C) preoperative CT scan of the brain, D) postoperative CT scan of the same patient

Figure 2:

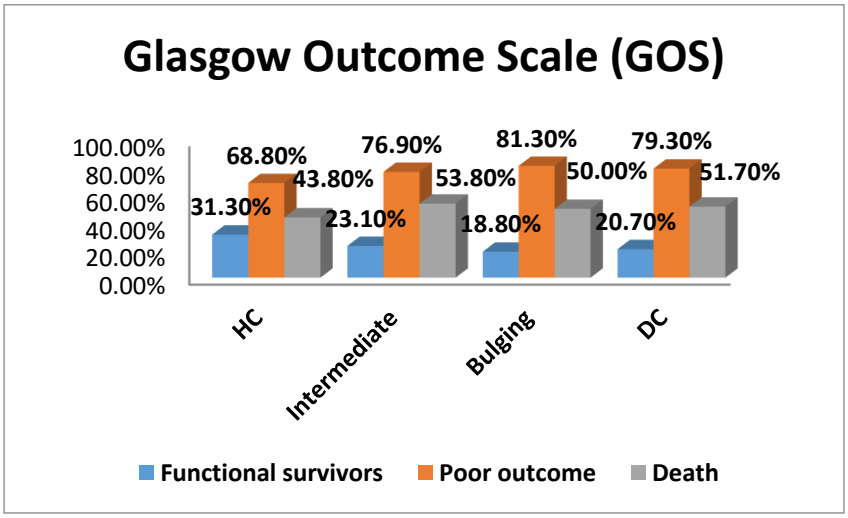

Figure 2: The outcome among the study groups

DISCUSSION

TBI is a major international health and socioeconomic problem impacting about 50-70 million individuals annually. However, the actual burden of TBI may be underestimated especially in low- and middle-income countries which may encounter almost three times more cases of TBI than high-income countries [15]. ASDH represents about one-third of severe TBI with grim prognosis and unfavorable outcomes despite advances in neurotraumatology management.

Significant primary brain injury and associated substantial intra and extracranial injuries are common in ASDH. Moreover, subsequent complex secondary brain injury mechanisms predominate after trauma including ischemia, reactive hyperemia, coagulopathy and delayed intracerebral contusion/hematomas resulting in intracranial hypertension and herniation syndromes [5]. The post-traumatic cellular cascade involves excitotoxicity, mitochondrial dysfunction and neuroinflammation causing axon degeneration and ultimately cell death. Such molecular mechanisms are the target for future therapeutics for TBI [16].

The mainstay of surgical management of ASDH is HC and DC, which allows for edematous brain tissue expansion to decrease intracranial hypertension [2, 5, 12]. Nevertheless, there is no high-quality evidence regarding the optimum technique for ASDH evacuation.

DC provides the most aggressive method for brain decompression and may be the only available solution for lowering ICP with brain bulging after ASDH evacuation. Nonetheless, concomitant parenchymal contusion may increase secondary to unrestrained brain expansion after DC, which may indicate a graver neurological outcome as reported by Flint et al [17]. Likewise, brain edema, venous congestion, and hemorrhagic infarction may occur along the cranial defect boundaries after brain herniation [18]. In addition, DC necessitates additional effort or incision for bone flap storage and second surgery for cranioplasty. Moreover, delayed postoperative seizures (3.5%) and hydrocephalus (11%) have been reported [17]. Furthermore, a higher incidence of bone flap infection was reported after DC autologous cranioplasty (4.5-16%) compared to (0-4%) after HC [19-21].

On the other hand, HC permits adequate brain expansion (except in brain bulging beyond the craniotomy edge) and avoids a second incision for bone flap storage or second surgery for cranioplasty [2, 20-24]. Kenning et al. [22] confirmed that ICP control was effective and equivalent after (HC 12.1 ± 2.6mmHg) compared to DC (15.0 ± 6.3mmHg), despite the lesser volume of expansion with HC (77.5 ± 54.1 ml) than DC (105.1 ± 65.1 ml), which was not statistically Significant. In fact, there may be a (3.2%) failure rate requiring subsequent DC due to significant ICP elevation [21]. Additionally, bone flap depression may develop requiring reoperation to fix the craniotomy flap.

The rationale of selecting the surgical technique for ASDH in the current study was consistent with the recommendations of Hutchinson et al. [25]. HC was preferred in patients with lax brain to avoid second surgery for bone flap placement with minimal risk of ICP elevation postoperatively. While patients with brain bulging

preventing safe replacement of the bone flap underwent DC to allow maximum room for brain expansion and decrease the ICP effectively.

Regarding the gray zone/ intermediate brain condition, DC was adopted due to the unavailability of ICP monitoring, which may result in late detection of intracranial hypertension resistant to HC and potential permanent neurological deterioration. This means several patients had a "prophylactic" DC and HC might have been appropriate. Such patients are candidates for an ongoing randomized controlled clinical trial [26].

Unfortunately, the unavailability of ICP monitoring is a prevalent scenario in most low and middle-income countries [27]. ICP monitoring is recommended in the recent management of severe TBI to control intracranial hypertension and ensure adequate cerebral perfusion pressure [28], both of which have been demonstrated to decrease mortality significantly [29,30].

The results of the current study suggest no significant difference regarding the outcome between HC (5/16) and DC (6/29) ($p > 0.05$). These results are consistent with Kenning et al. [22] who demonstrated that the outcome of both HC and DC were comparable. Similarly, Peethambaran et al. [31] reported no significant difference between HC and DC with regard to the duration of surgery, length of ICU stay and survival. Moreover, Woertgen et al. [2] and Chen et al. [8] found DC had a higher mortality rate than HC, however, there was no significant difference regarding the outcome between the 2 groups.

Regarding the effectiveness of secondary DC after failure of first- and second-tier medical therapies to control sustained and refractory intracranial hypertension in TBI patients; the randomized clinical trial (RESCUEicp) confirmed that DC significantly decreased the ICP and resulted in lower mortality and higher rates of vegetative state and severe disability than medical care. However, the rates of moderate disability and good recovery were comparable in the two groups [32].

The limitations of the current study include the retrospective study design, limited patient number and the unavailability of ICP monitoring. Nevertheless, it evaluated the suitability, indications, outcome and complications of HC and DC, the main surgical management of ASDH according to the intraoperative brain condition.

In summary, in this study, both HC and DC were viable surgical options with no difference in the outcomes and complications following evacuation of traumatic ASDH for relaxed and bulging brains respectively. Patients with intermediate brain condition are managed according to the surgeon's preference and facility equipment, especially availability of ICP monitoring. Future prospective randomized studies with larger sample size are necessary.

Conflicts of interest: we have no conflict of interest.

Financial Disclosures: Nil.

REFERENCES

1-Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW et al. Surgical Management of Traumatic Brain Injury Author Group. Surgical management of acute subdural hematomas. Neurosurgery. 2006; 58(3 Suppl): S16-24; discussion Si-iv.

2-Woertgen C, Rothoerl RD, Schebesch KM, Albert R. Comparison of craniotomy and craniectomy in patients with acute subdural haematoma. Journal of Clinical Neuroscience. 2006; 13:718-721.

3-Graham DI, Gennarelli TA, McIntosh TK. Trauma. In: Graham DI, Lantos PL, editors. Greenfield's Neuropathology. Arnold; London: 2002; 823–898

4-Servadei F, Nasi MT, Giuliani G, Cremonini AM, Cenni P, Zappi D et al. CT prognostic factors in acute subdural haematomas: the value of the 'worst' CT scan. Br J Neurosurg. 2000; 14(2):110-6.

5-Karibe H, Hayashi T, Hirano T, Kameyama M, Nakagawa A, Tominaga T. Surgical management of traumatic acute subdural hematoma in adults: a review. Neurol Med Chir (Tokyo). 2014; 54(11):887-94.

6-Jiang JY, Xu W, Li WP, Xu WH, Zhang J, Bao YH et al. Efficacy of standard trauma craniectomy for refractory intracranial hypertension with severe traumatic brain injury: a multicenter, prospective, randomized controlled study. J Neurotrauma. 2005; 22(6):623-8.

7-Phan K, Moore JM, Griessenauer C, Dmytriw AA, Scherman DB, Sheik-Ali S, Adeeb N, Ogilvy CS, Thomas A, Rosenfeld JV. Craniotomy Versus Decompressive Craniectomy for Acute Subdural Hematoma: Systematic Review and Meta-Analysis. World Neurosurg. 2017; 101:677-685.e2.

8-Chen SH, Chen Y, Fang WK, Huang DW, Huang KC, Tseng SH. Comparison of craniotomy and decompressive craniectomy in severely head-injured patients with acute subdural hematoma. J Trauma. 2011; 71(6):1632-6.

9-Massaro F, Lanotte M, Faccani G, Triolo C. One hundred and twenty-seven cases of acute subdural haematoma operated on. Correlation between CT scan findings and outcome. Acta Neurochir (Wien) 1996; 138:185- 91.

10-Pastor IS, Dumbravă LP, Siserman C, Stan H, Para I, Florian IȘ. Predictive factors of 30-day mortality in patients with traumatic subdural hematoma. Exp Ther Med. 2021; 22(1):757

11-Tanizaki S, Toma Y, Miyashita K, Maeda S. The Characteristics of Withdrawal or Withholding of

Life-Sustaining Treatment in Severe Traumatic Brain Injury: A Single Japanese Institutional Study. World Neurosurg X. 2022; 4(17):100144.
12-Kolias AG, Belli A, Li LM, Timofeev I, Corteen EA, Santarius T et al. Primary decompressive craniectomy for acute subdural haematomas: results of an international survey. Acta Neurochir (Wien). 2012; 154(9):1563-5.
13-Jost JN. Primary Decompressive Craniectomy After Traumatic Brain Injury: A Literature Review. Cureus. 2022; 4;14(10):e29894.
14-Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet 1975; 1;1(7905):480-4.
15-Roozenbeek B, Maas A, Menon D. Changing patterns in the epidemiology of traumatic brain injury. Nat Rev Neurol. 2013; 9:231-6.
16-Ladak AA, Enam SA, Ibrahim MT. A Review of the Molecular Mechanisms of Traumatic Brain Injury. World Neurosurg. 2019; 131:126-132.
17-Flint AC, Manley GT, Gean AD, Hemphill JC, Rosenthal G. Post-operative expansion of hemorrhagic contusions after unilateral decompressive hemicraniectomy in severe traumatic brain injury. J Neurotrauma. 2008; 25:503–512
18-Simard JM, Kent TA, Chen M, Tarasov KV, Gerzanich V. Brain oedema in focal ischaemia: molecular pathophysiology and theoretical implications. Lancet Neurol. 2007; 6:258–268
19-Ban SP, Son YJ, Yang HJ, Chung YS, Lee SH, Han DH. Analysis of complications following decompressive craniectomy for traumatic brain injury. J Korean Neurosurg Soc. 2010; 48(3):244-50
20-Kano T, Kurosaki S, Wada H. Retrospective analysis of hinge technique for head trauma or stroke. Neurol Med-Chir. 2012; 52:816–821
21-Layard Horsfall H, Mohan M, Devi BI, Adeleye AO, Shukla DP, Bhat D et al. Hinge/floating craniotomy as an alternative technique for cerebral decompression: a scoping review. Neurosurg Rev. 2020; 43(6):1493-1507.
22-Kenning TJ, Gandhi RH, German JW. A comparison of hinge craniotomy and decompressive craniectomy for the treatment of malignant intracranial hypertension: early clinical and radiographic analysis. Neurosurg Focus. 2009; 26:E6
23-Ko K, Segan S. In situ hinge craniectomy. Oper Neurosurg. 2007; 60:ONS-255–ONS-259
24-Adeleye AO. Clinical and radiologic outcome of a less invasive, low-cost surgical technique of osteoplastic decompressive craniectomy. J Neurol Surg Part A: Central European Neurosurg. 2016; 77:167–175
25- Hutchinson PJ, Kolias AG, Tajsic T, Adeleye A, Aklilu AT, Apriawan T et al. Consensus statement from the International Consensus Meeting on the Role of Decompressive Craniectomy in the Management of Traumatic Brain Injury. Acta Neurochir. 2019; 161, 1261–1274
26- RESCUE-ASDH trial official website. Available at: http://www.
rescueasdh.org/
27-Smart LR, Mangat HS, Issarow B, McClelland P, Mayaya G, Kanumba E et al. Severe traumatic brain injury at a tertiary referral Center in Tanzania: epidemiology and adherence to brain Trauma Foundation guidelines. World Neurosurg. 2017; 105:238–248
28-Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery. 2017; 80(1):6–15.
29-Gerber LM, Chiu YL, Carney N, Hartl R, Ghajar J. Marked reduction in mortality in patients with severe traumatic brain injury. J Neurosurg. 2013; 119:1583–1590.
30-Rubiano AM, Carney N, Chesnut R, Puyana JC. Global neurotrauma research challenges and opportunities. Nature. 2015; 527:S183–S197
31-Peethambaran AK, Gopal VV, Valsalamony J. Four-quadrant osteoplastic decompressive craniotomy: A novel technique for refractory intracranial hypertension - A pilot study. Neurol India. 2015; 63(6):895-902.
32-Hutchinson PJ, Kolias AG, Timofeev I, Corteen EA, Czosnyka M, Timothy I, et al. Trial of decompressive Craniectomy for traumatic intracranial hypertension. N Engl J Med. 2016; 375:1119–1130.

To cite:
ElGaidi, M., Shaban, M. Surgical Management of Traumatic Acute Subdural Hematoma. *Zagazig University Medical Journal*, 2023; (852-858): -. doi: 10.21608/zumj.2023.180056.2699