Original Research Article PREDICTORS OF MORTALITY IN TRAUMATIC BRAIN INJURY PATIENTS AFTER PRIMARY DECOMPRESSION CRANIOTOMY

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ABSTRACT

Introduction:Intracranial hypertension can cause secondary damage after a traumatic brain injury. Aggressive medical management might not be sufficient to alleviate the increasing intracranial pressure (ICP), and decompressive craniectomy (DC) can be considered. Decompressive craniectomy can be divided into categories, according to the timing and rationale for performing the procedure: primary (done at the time of mass lesion evacuation) and secondary craniectomy (done to treat refractory ICP). Most studies analyze primary and secondary DC together. The aim of this retrospective study is to predict the indicators of mortality after DC.

Methods: 30 cases in S.G.M.H, getting admitted through SOPD, casualty or transferred from other department diagnosed as severe Traumatic Brain Injury during the period of study were included in the study. Severity was defined according to Glasgow Comal Scale at admission. Patients with moderate to severe head injury requiring decompressive craniectomy considered for this trail, entry will be determined using the above inclusion and exclusion criteria after resuscitation, and data was entered in proforma.

Result: Age >55Years undergoing Primary decompression Craniotomy had 100% Mortality. Males constituted of about 83.33% of the studied cases as large number of males were admitted due to RTA(Road Traffic Accidents). In Mechanism of injury-RTA contributed for the maximum number of deaths (61.11%). Multiple seizure episodes show higher Mortality rate of 93.33%. LOC > 6hrs show higher Mortality rate of 88.88%. Cohort with ENT Bleed + had a mortality rate of 66.66% while the other cohort had mortality of 33.33%. Severe Head injury with GCS < 8 had the highest mortality of 90.90% when compared to that of Moderate

(63.63%) and Mild (12.5%) head injury. Mortality is highest in cohort with B/L Dilated Pupils on admission-88.88%. Mortality is highest in cohort with B/L Non reactive Pupils on admission-91.66%. Patients admitted wit Contusion had a mortality of 75% while that of SDH is 73.33%. Mortality is 100% in cohort of patients admitted with Midline shift \geq 10mm.Mortality is about 85.7% in Cohort of IntraOp Hypotension while it is 33.33% in cohort without IntraOp Hypotension. It showed a significant statistical relation. Mortality among Cohort with PostOp GCS at 24Hrs with GCS \leq 8 is 92.30% and that of PostOp GCS > 8 is 35.29%.Cohort of patients with Preop Hyperglycaemia had a mortality of 75%. Highest mortality is seen in patients with time lapse of >72hrs between trauma and surgery which is 80%.

Conclusion: In patients undergoing primary DC after traumatic brain injury, the predictors of mortality include Age \geq 70years, LOC > 6hrs, Multiple Seizure Episodes, ENT Bleed, Severe Head Injury(GCS \leq 8),Bilateral unreactive pupils, B/L Dilated Pupils, Contusion & Subdural hemorrhage, Midline shift \geq 10mm, Diagnosis of Contusion & SDH, intraoperative hypotension, Time Lapse of > 72hrs.

Keywords: Primary decompressive craniectomy; Predictors; Mortality; Traumatic brain injury, EDH, SDH, Contusion, Midline shift.

1. BACKGROUND:

Mortality and morbidity of patients with severe traumatic brain injury is high. Decompressive craniotomy increases oxygen delivery to cells by improving blood flow. It is still unclear if Decompression Craniotomy improves outcome in patients with severe Post Traumatic Brain Injury.

Despite the controversies over the efficacy and prognosis of decompressive craniectomy (DC), this technique plays an important role in traumatic brain injury (TBI) therapeutic protocols¹⁻³. DC is usually categorized into two types: primary DC is defined as prophylactic DC by removing a large bone flap after evacuating a mass lesion in an early post-injured phase (generally <24 hours)^{4,5}; secondary DC is performed for patients with intracranial hypertension who are unresponsive to medical treatment^{4,6}. The long-term results of DC after TBI have been well demonstrated in some published studies^{4,7,8}.

So far, only two multicenter, randomized controlled trials (RCTs) have been conducted regarding the outcomes of TBI patients with DC. Both RCTs focused on secondary DC, of these the DECRA showed a negative result but the RESCUE-icp demonstrated lower mortality in patients with traumatic brain injury and refractory intracranial hypertension. Notably, the recently started RESCUE-ASDH is an ongoing, multicenter, pragmatic RCT that aims to compare the outcomes between primary DC and craniectomy in adult TBI patients. Within these studies, most of the DC procedures were primary surgery and generally were associated with a high risk of unfavorable results^{2,10}.

OBJECTIVES:

The purpose of this study was to determine which of variables, like

1) Demographic Factors - Age, Gender, Mechanism of Injury,

2) Presenting symptoms - LOC, Vomitings, ENT Bleed, Seizures,

3) PreOp Clinical status - PreOp GCS, PreOp Pupillary

Abnormality, PreOp Pupillary Reaction,

4) IntraOp Clinical Status - IntraOp Hypotension,

5) PostOp Clinical status - PostOp GCS

6) Radiological Abnormalities - EDH, SDH, Contusion, Midline shift,

7) Time lag between injury and surgery,were predictive of mortality in patients with Severe TBI.

2. MATERIAL AND METHODS

30 cases in S.G.M.H, getting admitted through SOPD, casualty or transferred from other department diagnosed as severe Traumatic Brain Injury during the period of study were included in the study. Severity was defined according to Glasgow Comal Scale at admission. Hemodynamic stabilization and intubation was done where necessary and the post resuscitation GCS was noted. A CT scan was done as soon as possible. Preop and postop Clinical, Biochemical, Radiological findings were compared to derive conclusions about mortality. The postoperative GCS at 24hrs & at discharge were noted. Consent for surgery and study was obtained from next of kin after detail explanation about the study. Approval for the study was obtained from the Ethics committee. Data were entered in proforma.

Quantitative variables are presented as mean and standard deviation (SD) or median and interquartile range (IQR) in case of skewed data. For qualitative factors, absolute and relative frequencies are given. For mortality rates, 95 % confidence intervals (CI) were calculated for comparison of observed and predicted mortality by the RISC II score. Adjusted odds ratios with 95% CIs were calculated using a logistic regression model with hospital mortality as dependent variable.

Inclusion Criteria:

• All patients with severe Traumatic Brain Injury requiring Primary Cranial Decompression

Exclusion Criteria:

- Poly Trauma patients
- Patients with chronic Hepatic, Renal, Cardio pulmonary diseases.
- Nontraumatic causes like infarct, spontaneous ICH or aneurysmal bleed.
- Post resuscitation GCS 3.
- Absent brain stem reflexes.
- Devastating injury not expected to survive for 24 hrs.

3. **RESULTS:**

TABLE NO 1

RELATION BETWEEN IN DIFFERENT CHARACTERISTIC FEATURES OF PATIENT AND MORTALITY IN THE OPERATED CASE OF TBI

SL. No	AGE	No Of Cases In Specific Age Group	No Of Deaths In Specific Age Group	Age specific Mortality Rate	Total Number Of Deaths	% Of Deaths among Total Deaths
1.	<30	14	5	35.71		27.77
2.	30-55	11	8	72.72		44.44
3.	55-70	4	4	100	18	22.22
4.	>70	1	1	100		5.55
			Sex			
1.	Male	25	13	52	18	72.22
2.	Female	5	5	100	10	27.77
		Mech	anism Of Injı	ıry		
1.	RTA	18	11	61.11		61.11
2.	Fall	8	4	50	18	22.22
3.	Assault	4	3	75		16.66
		L	OC Duration			
1.	<30Min	7	1	14.28		5.55
2.	30Min-6hours	14	9	64.2	18	50.00
3.	>6 hour	9	8	88.88		44.44
		No of S	Seizure Episo	des		
1.	Nil	8	1	12.50		5.55
2.	1 Episode	7	3	42.85	18	16.66
3.	Multiple Episodes	15	14	93.33		77.77
		No of Ca	ses with ENT	Bleed		
1.	Nil	15	6	40		33.33

2.	ENT Bleed +	15	12	80		66.66	
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Significant Linear Trend Present.Chi Square- 7.055P Value - 0.0079Mean age ± SD - 34±17.8

Age >55 Years undergoing Primary decompression Craniotomy had 100% Mortality. The highest mortality is in the age group of 30-55 years due to large number of cases in that age group.

Males constituted of about 83.33% of the studied cases as large number of males were admitted due to RTA. About 72.22% of total deaths are of males. But Gender specific death rate is highest (100%) in Females.

In Mechanism of injury-RTA contributed for the maximum number of deaths (61.11%). LOC>6Hours had the worst outcome with Mortality of 88.88% while that of LOC<30min is only 5.55%.

Multiple seizure episodes show Mortality rate of 93.33% while that of No seizure Episodes show Mortality of 12.50% which indicates that there is strong relation between Seizures and Mortality occurring post TBI.

Cohort with ENT Bleed + had a mortality rate of 66.66% while the other cohort had mortality of 33.33%. (**Table -1**)

TABLE NO 2

RELATION BETWEEN PRESENTING SYMPTOMS AND MORTALITY IN THE OPERATED CASES OF TBI

SL No	Symptoms	Total No of Cases	Total No of Deaths	Symptom Specific Death Rate	Total Number of Deaths	% of Deaths among total Deaths			
1.	LOC>30Min	23	17	73.91		94.44			
2.	Vomitings>1Episode	25	16	64	18	88.88			
3.	Seizures >1Episode	15	14	93.33	10	77.77			
4.	ENT Bleed	15	12	80		66.66			
		PreO	P GCS						
1.	Mild Head Injury (13-15)	8	1	12.5		5.5			
2.	Moderate Head Injury (9-12)	11	7	63.63	18	38.88			
3.	Severe Head Injury (3-8)	11	10	90.90		55.55			
	Pupillary Abnormalities								

1.	Uni Lateral Dilated/ Constricted Pupil	14	8	57.14		44.44			
2.	2. B/L Dilated/ Constricted Pupil		8	88.88	18	44.44			
3.	Normal Pupils	7	2	28.57		11.11			
		Intra	OP BP						
1.	SBP<90mm of Hg	14	12	85.7	18	66.66			
2.	$SBP \ge 90mm \text{ of } Hg$	16	6	37.5	10	33.33			
	Post OP GCS								
1.	$GCS \le 8$	13	12	92.30	18	66.66			
2.	GCS > 8	17	6	35.29	10	33.33			

Of the 4 clinical presentations studied (LOC, Vomitings, Seizures, ENT Bleed), Seizures >1episode had symptom specific mortality rate (93.33%) followed by ENT Bleed (80%), LOC>30Min (73.91%), Vomitings>1episode (64%).

Severe Head injury with GCS < 8 had the highest mortality of 90.90% when compared to that of Moderate (63.63%) and Mild (12.5%) head injury.

PreOp B/L Dilated Pupils cohort had a mortality of 88.88% and PreOp U/L Dilated Pupil cohort had 57.14% and that of PreOp Normal pupils being 28.57%. Mortality is about 85.7% in Cohort of IntraOp Hypotension while it is 33.33% in cohort without IntraOp Hypotension. Mortality among Cohort with PostOp GCS at 24Hrs with GCS≤8 is 92.30% and that of PostOp GCS > 8 is 35.29%.

TABLE NO 3 COMPARISON BETWEEN PREOP& POSTOP PUPILLARY ABNORMALITY TO MORTALITY

SL No		Total Preo p Case s	Total Preo P Deat hs	PreOp Pupillary Abnormal ity Specific Death Rate	Total Posto P Cases	Total Posto p Death s	PostOp Pupillary Abnormal ity Specific Death Rate
1.	Uni Lateral Abnormal Pupil	14	8	57.14	13	7	53.84
2.	Bi lateral Abnormal Pupil	9	8	88.88	9	8	88.88
3.	Normal Pupils	7	2	28.57	8	3	37.5

	PREOP& POSTOP PUPILLARY REACTION TO MORTALITY									
1.	Unilateral NonReactive Pupil	7	4	57.14	5	3	60			
2.	B/L Non Reactive Pupil	12	11	92.30	14	13	92.85			
3.	B/L Reactive Pupil	11	3	20	11	2	18.18			

No Significant difference is observed in between Mortality Ratios of PreOp B/L Abnormal Pupils and PostOp B/L Abnormal Pupils (88.88% vs 88.88%) and also in PreOp and PostOp U/L Abnormal pupils (57.14% vs 53.84%). No Significance difference in between Mortality Ratios of PreOp and PostOp U/L Non Reactive Pupils (57.14% vs 60%) is observed. No Significance difference in between Mortality Ratios of PreOp and PostOp B/L Reactive Pupils(20% vs 18.18%) is observed. (Table -3)

OPERATED CASES OF TBI										
SL No	NCCT Head Finding	Total No Of Cases	Total No Of Deaths	Case Specific Death Rate	Total Number Of Deaths	% Of Deaths among total Deaths(18)				
1.	EDH	7	1	14.28		5.55				
2.	SDH	15	11	73.33	18	61.11				
3.	Contusion	8	6	75		33.33				
MI	MIDLINE SHIFT AS PER NCCT HEAD REPORT AND MORTALITY IN THE OPERATED CASES OF TBI									
1.	<5mm	8	2	25		11.11				
2.	5mm-10mm	19	13	68.42	18	72.22				
3.	\geq 10mm	3	3	100		16.66				
TIM	TIME GAP BETWEEN TIME OF INJURY TO OPERATIVE PROCEDURE AND MORTALITY IN THE OPERATED CASES OF TBI									
1.	<6Hours	3	1	33.33	18	5.55				
2.	6-24Hours	10	4	40		22.22				
3.	24-72Hours	8	6	75		33.33				
4.	>72 Hours	10	8	80		44.44				

TABLE NO 4 RELATION BETWEEN NCCT HEAD FINDINGS AND MORTALITY IN THE OPERATED CASES OF TBI

Mortality among contusion cases is significantly higher at 75% followed by SDH cases at 73.33% followed by EDH at 14.26%. It shows best prognosis for EDH Cases.

Mortality among Cohort with Mid line shift \geq 10mm is 100% and that of 5-10mm is about 68.42% and that of <5mm is 25%.

Mortality in cohort with PreOp Hyperglycaemia is about 75% while that of no PreOp hyperglycaemia is about 30%.

Time Lapse between Time of Injury to Operative Procedure showed a significant Linear trend association with Mortality with maximum at >72 Hours (80%) followed by 24 hours (62.50%), 6Hrs-24Hrs (50%), <6Hrs (33%). (Table-4)

4. **DISCUSSION**

In patients after sTBI, neuronal injury and neural death secondary to the primary insult are irreversible. Raised ICP and cerebral edema causes secondary injury of Brain. This

secondary injury can be potentially dangerous and can be prevented. Proper initial management provides an opportunity to salvage neurons and improve functional outcome post DC. DC can be performed as an early and emergency intervention as part of a multi-tiered management protocol of sTBI. It can also be considered as a last-tier intervention to reduce intractable ICP in patients for whom all other medical therapeutic methods have failed.

AGE

Our study yielded results Similar to the results obtained by **Rozzelle CJ etal**¹²stating that Mortality rates in adults with severe TBI aged 55 and older range from 80%, significantly higher than in younger patients. **Harris C et al**¹³ reported that the likelihood of death was maximal after age 71.

Factors increasing mortality in old age-

1. Dura becoming more adherent to the skull.

2. More older adults receiving aspirin and anticoagulant therapies.

3.Normal aging changes include cerebrovascular atherosclerosis and decreased free radical clearance

GENDER

Our results are similar to study by **Nelson Saade etal**¹¹stating 83.9% Mortality among Males & **AmitAgrawaletal**¹⁴stating 18% Mortality among female patients and 82% among male patients.

Various studies in animals and healthy human brains suggest inherent gender differences in brain metabolism. It is found that higher mean regional and hemispheric cerebral blood flow was present in females as compared with males. Although mean cortical thickness of loci were equal in males and females, significantly higher neuronal densities were demonstrated in males. Studies indicate that higher mortality rates are seen in female rats following induced fluid percussion injury.

MECHANISM OF INJURY

In our Study of 30 Patients, Mortality is high in Patients with Mechanism of Injury being RTA which is 61.11% followed by Fall - 22.22% followed by Assault which is 16.66%. This is because of the large number of patients being admitted due to RTA followed by fall & Assault. Our study states that there is no significant relation between Mechanism of Injury and Mortality rate.

This is similar to results obtained by **G Gururajetal**¹⁵ which shows that Road traffic injuries are the leading cause (60%) of TBIs followed by falls (20%-25%) and violence (10%). This is also similar to results obtained by **Abdul Rehmanetal**¹⁶ which shows that mortality is high in cohort of RTA which is 56% followed by Fall with 8% followed by Assault which is 16.66%.

SEIZURE EPISODES

In our Study of 30 Patients, Most of the deaths occurred in Patients with Multiple Episodes Of seizures (77.77%), followed by 1 Episode of seizure (16.66%), followed by no Episodes of seizures (5.55% among total deaths).

The seizure usually occurs when there is a scar tissue in the brain as a consequence following TBI. During an episode of seizure there is a sudden abnormal electrical disturbance that occurs in the brain. Seizure episode is known to decrease blood supply to the brain momentarily decreasing the EVM of the patient leading to Aspiration and Hypoxia. Drugs and alcohol lower the threshold for seizure regardless of prior brain injury.

ENT BLEED

Our results can be compared to study by **MadhusudhanNageshetal**¹⁹show Mortality of about 45.5% among the cases with ENT bleed.

ENT Bleed can occurs due to direct temporo squamous bone fracture or due to tympanic membrane perforation in traumatic brain Injury. ENT bleed significantly decides the morbidity and mortality of patient. ENT bleed may also mask CSF Otorrhea which significantly increases the risk of Meningitis increasing the mortality. Studies by **MadhusudhanNageshetal**¹⁹show Mortality of about 45.5% among the cases with ENT bleed.

SEVERITY OF HEAD INJURY

Our results were similar to results obtained by **Herbert Ariaka et al**²⁰ stating Mortality among Severe head injury being 65.2%, followed by Moderate head injury being 30.4% and Mild head injury being 4.4% among total deaths. As an isolated predictor, GCS is an important determinant of mortality in both TBI and non-TBI patients but GCS is a more powerful predictor of death in TBI patients.

PREOPERATIVE PUPILS SIZE

Our results are similar to the results obtained by **Seyed Reza Bagheri et al**²² stating that mortality in Patients with PreOp B/L Pupillary abnormality(42.9%), followed by PreOp U/L Pupillary abnormality (35.7% among total deaths).

It is also similar study of **GaétaneGouello et al²³**stating that most of the deaths occurred in Patients with PreOp B/L Pupillary abnormality(47.05%), followed by PreOp U/L Pupillary abnormality(35.29% among total deaths).

The widely accepted theory for the pupillary dilation is that an intracranial mass lesion traps the lllrd cranial nerve at the tentorial edge as the uncus is herniated downward and medially. Continued compression of the medial temporal lobe into the brain stem results in loss of consciousness, decerebrate posturing, and cardiovascular collapse. An alternative view holds that compromise of brain stem circulation is a major contributing factor to pupillary dilation. It states that central brain stem ischemia, and not direct peripheral third nerve compression, is responsible for pupil dilation.

PREOPERATIVE REACTION OF PUPILS

Our results are similar to results obtained by **Zhiji Tang et al²⁴** stating that motality is high in Patients with PreOpB/L Non-Reactive Pupil (78.68%), followed by PreOpUnilateral Non-Reactive Pupil (16.39 %) followed by Preop B/L Reactive Pupil (4.9 % among total deaths).

About 91.66% of cases with B/L Non-Reactive Pupils have expired. A sluggish or slow pupillary response may indicate increased ICP, and nonreactive pupils are often associated with severe increases in ICP and/or severe brain damage.

INTRA OPERATIVE BP

Studies by **Miller etal.**²⁶showed that hypoxia and hypotension and hypothermia are statistically related to unfavourable outcome following TBI.The causes maybe:

1)Development of progressive treatment-refractory intraoperative hypotension as a result of the intraoperative blood loss and a deep anesthetic state;

2)Development of vasoparalysis, due to sudden decompression while opening dura.

POST OPERATIVE GCS AT 24 HOURS

Patients presented with a lower GCS scores are associated with a poor outcome. Our study shows similar results to study by **Reddy etal.**²⁷ reported survival rate of 88% among the patients who had a preoperative GCS of eight and above, and survival of 27% among those with GCS less than eight.

MIDLINE SHIFT

Our results are similar to results obtained by **Zhiji Tanget al**²⁴ which states 12.29% (15/122) death rate of cases with Midline Shift <5mm among total deaths and 87.77% (107/122) death rate of cases with Midline Shift >5mm. Though it follows the same trend, the rates are different and high in our study as the total mortality rates are higher in our study because of Increased Time gap between time of incident and time of surgery due to delay in referral from lower to higher centre.

Following traumatic brain injury (TBI), midline shift of the brain at the level of the septum pellucidum is often caused by unilateral space-occupying lesions and is associated with increased intracranial pressure and worsened morbidity and mortality. In fact, midline shift is a measure of ICP; presence of the former is an indication of the latter. Immediate surgery may be indicated when there is a midline shift of over 5 mm.

TIME LAG BETWEEN INJURY AND SURGERY

Our study yielded very similar results to that of a study by **Seeligetal.** showing that if the surgery was performed within 4 hours, the mortality rate is 30%, whereas after 4 h of injury - rate of mortality increased over 90%. The studies, trails and medical literature regarding early DC is very conflicting. There are studies supporting early DC while few oppose early DC. **Faleiroetal.**³² dichotomized 89 patients into 6hours, 6-24 hours and >24hours for DC in his studies. His study found that patients who were operated early had 59% mortality when compared to that of 53% who had the surgery later.

The relationship between time to surgery from injury and outcome is still controversial.

5. CONCLUSION

According to the results yielded by our study,

In patients undergoing primary DC after traumatic brain injury, the predictors of mortality include Age ≥70years, LOC > 6hrs, Multiple Seizure Episodes, ENT Bleed, Severe Head Injury(GCS≤8), Bilateral unreactive pupils, B/L Dilated Pupils, Contusion & Subdural hemorrhage, Midline shift ≥10mm, intraoperative hypotension, Time Lapse of > 72hrs.

6. REFERENCES

- 1. WettervikTS, Lenell S, Nyholm L, Howells T, Lewén A, Enblad P. Decompressive craniectomy in traumatic brain injury: usage and clinical outcome in a single centre. Actaneurochirurgica. 2018/02/01 2018;160(2):229-237.
- 2. Kramer AH, Deis N, Ruddell S, et al. Decompressive Craniectomy in Patients withTraumatic Brain Injury: Are the Usual Indications Congruent with Those Evaluated in Clinical Trials? Neurocritical care. Aug 2016;25(1):10-19.
- 3. Picetti E, Caspani M, Iaccarino C, et al. Intracranial pressure monitoring after primary decompressive craniectomy in traumatic brain injury: a clinical study. Actaneurochirurgica. 2017;159(4):615-622.
- 4. Nambiar M, MacIsaac C, Grabinski R, Liew D, Kavar B. Outcomes of decompressive craniectomy in patients after traumatic brain injury. Critical care and resuscitation :journal of the Australasian Academy of Critical Care Medicine. Jun 2015;17(2):67-72.
- 5. Kolias AG, Adams H, Timofeev I, et al. Decompressive craniectomy following traumatic brain injury: developing the evidence base. British journal of neurosurgery.2016;30(2):246-250.
- 6. Josan V, Sgouros S. Early decompressive craniectomy may be effective in the treatment of refractory intracranial hypertension after traumatic brain injury. Vol222006.
- 7. Gouello G, Hamel O, Asehnoune K, Bord E, Robert R, Buffenoir K. Study of the longterm results of decompressive craniectomy after severe traumatic brain injury based on a series of 60 consecutive cases. TheScientificWorldJournal. 2014;2014:207585.
- 8. Vilcinis R, Bunevicius A, Tamasauskas A. The Association of Surgical Method with Outcomes of Acute Subdural Hematoma Patients: Experience with 643 Consecutive Patients. Vol 1012017.
- 9. Huang YH, Lee TC, Lee TH, Liao CC, Sheehan J, Kwan AL. Thirty-day mortality in traumatically brain-injured patients undergoing decompressive craniectomy. Journal of neurosurgery. Jun 2013;118(6):1329-1335.
- 10. Tapper J, Skrifvars M, Kivisaari R, Siironen J, Raj R. Primary decompressive craniectomy is associated with worse neurological outcome in patients with traumatic brain injury requiring acute surgeryDecompressive craniectomy in the treatment of

severe traumatic brain injury: The question of quality of survivalComment on results of decompressive craniotomy for trauma. Vol 82017.

- Nelson Saade ; José Carlos EstevesVeiga, Luiz Fernando Cannoni ; Luciano Haddad ; JoãoLuizVitorinoAraújo . Evaluation of prognostic factors of decompressive craniectomy in the treatment of severe traumatic brain injury. Rev. Col. Bras. Cir. 2014; 41(4): 256-262
- Curtis J. Rozzelle, James L. Wofford MD, Charles L. Branch MD.Predictors of Hospital Mortality in Older Patients with Subdural Hematoma. J Am GeriatrSoc 43: 240–244, 1995.
- 13. Harrison-Felix, C., Whiteneck, G., Devivo, M.J., Hammond, F.M. and Jha, A. (2006). Causes of death following 1 year post injury among individuals with traumatic brain injury. J Head Trauma Rehabil 21, 22-33.
- Agrawal A, Munivenkatappa A, Rustagi N, Mohan P R, Subrahmanyan B V. Epidemiological characteristics affecting outcome in traumatic brain injury. J Med Soc 2017;31:28-31.
- 15. Gururaj G, KolluriS.V.R, ChandramouliB.A, SubbakrishnaD.K and Kraus JF, "Traumatic Brain Injury", National Institute of Mental Health &Neuro Sciences, Publication no. 61.
- 16. Abdul RahmanShour, Benjamin Holmes , Emmanuel AdoyiAmeh, OluwoleOlayemiOlaomi, Ronald Anguzu, Laura Dawn Cassidy. Motor vehicle accident is a risk factor for traumatic head injury among children in Abuja: analysis of the first trauma registry in Nigeria. Pan African Medical Journal- 2019;33:215;– ISSN: 1937-8688.
- 17. Ziaeirad M, Alimohammadi N, Irajpour A, Aminmansour B. Association between Outcome of Severe Traumatic Brain Injury and Demographic, Clinical, Injury-related Variables of Patients. Iran J Nurs Midwifery Res. 2018 May-Jun;23(3):211-216.
- 18. MarekMajdan, Walter Mauritz, Ingrid Wilbacher, Ivan Janciak, Alexandra Brazinova, Martin Rusnak, Johannes Leitgeb, Traumatic brain injuries caused by traffic accidents in five European countries: outcome and public health consequences, *European Journal of Public Health*, Volume 23, Issue 4, August 2013, Pages 682–687.
- 19. MadhusudhanNagesh, KautilyaRajendrakumar Patel, Ajit Mishra, UjwalYeole, Andiperumal R. Prabhuraj. Role of repeat CT in mild to moderate head injury: an institutional study. Neurosurg Focus 47 (5):E2, 2019.
- 20. Ariaka, Herbert &Kiryabwire, Joel &Ssenyonjo, Hussein &Ogwal, Alfred &Nkonge, Emmanuel &Oyania, Felix. (2020). A Comparison of the Predictive Value of the Glasgow Coma Scale and the Kampala Trauma Score for Mortality and Length of Hospital Stay in Head Injury Patients at a Tertiary Hospital in Uganda: A Diagnostic Prospective Study. Surgery Research and Practice. 2020(7641):1-9. 10.1155/2020/1362741.
- 21. Khan, F., Villani, A., Rehman, A., Bari, M. (2018). Factors affecting functional outcome after decompressive craniectomy performed for traumatic brain injury: A retrospective, cross sectional study. Asian Journal of Neurosurgery, 13(3), 730-736.

- 22. Bagheri SR, Alimohammadi E, Saeidi H, Fatahian R, Soleimani P, Sepehri P, Abdi A, Beiki O. Decompressive Craniectomy in Traumatic Brain Injury: Factors Influencing Prognosis and Outcome. Iran J Neurosurg. 2017;3(1):21-26.
- 23. GaétaneGouello, Olivier Hamel, KarimAsehnoune, Eric Bord, Roger Robert, and Kevin Buffenoir1. Study of the Long-Term Results of Decompressive Craniectomy after Severe Traumatic Brain Injury Based on a Series of 60 Consecutive Cases. The Scientific World Journal Volume 2014, Article ID 207585, 10 pages.
- 24. Tang Z, Yang K, Zhong M, Yang R, Zhang J, Jiang Q, Liu H, Predictors of 30-Day Mortality in Traumatic Brain-Injured Patients after Primary Decompressive Craniectomy, *World Neurosurgery*. 2019;19:32671-3.
- 25. PedramEmami, Patrick Czorlich, Friederike S. Fritzsche, Manfred Westphal. Impact of Glasgow Coma Scale score and pupil parameters on mortality rate and outcome in pediatric and adult severe traumatic brain injury: a retrospective, multicenter cohort study. J Neurosurg 126:760–767, 2017.
- Miller JD, Becker DP, Ward JD, Sullivan HG, Adams WE, RosnerMJ (1977) Significance of intracranial hypertension in severe head injury. J Neurosurg 47:503– 516.
- Reddy AK, Saradhi V, Panigrahi M, Rao TN, Tripathi P, Meena AK. Decompressive craniectomy for stroke: indications and results. Neurol India. 2002 Dec;50(Suppl 1):66-9.
- Kyu-Hong Kim. Predictors for Functional Recovery and Mortality of Surgically Treated Traumatic Acute Subdural Hematomas in 256 Patients. J Korean NeurosurgSoc 45: 143-150, 2009.
- 29. Kafaki et al. Hyperglycemia: A Predictor of Death in Severe Head Injury Patients. Clinical Medicine Insights: Endocrinology and Diabetes 2016:9 43–46.
- 30. SeeligJM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC :Traumatic acute subdural hematoma : major mortality reduction in comatose patients treated within four hours. New Engl J Med 304 :1511-1518, 1981.
- Akyuz, Mahmut&Ucar, Tanju&Acikbas, Cem& Kazan, Saim&Yilmaz, Murat &Tuncer, Recai. (2010). Effect of Early Bilateral Decompressive Craniectomy on Outcome for Severe Traumatic Brain Injury. Turkish Neurosurgery 2010, Vol: 20, No: 3, 382-389.
- Rodrigo Moreira Faleiro, Luiz Carlos Mendes Faleiro, Elisa Caetano, Isabella Gomide. Decompression craniotomy- Prognostic factors and complications in 89 patients. ArqNeuropsiquiatr 2008;66(2-B):369-373.
- 33. Al-Jishi, Ahmed &Saluja, Rajeet& Al-Jehani, Hosam&Lamoureux, Julie &Maleki, Mohammad &Marcoux, Judith. (2011). Primary or Secondary Decompressive Craniectomy: Different Indication and Outcome. The Canadian journal of neurological sciences. Can. J. Neurol. Sci. 2011; 38: 612-620.
- Angelos G. Kolias, Hadie Adams, Ivan Timofeev, MarekCzosnyka, Elizabeth A. Corteen, John D. Pickard, Carole Turner, Barbara A. Gregson, Peter J. Kirkpatrick, Gordon D. Murray, David K. Menon& Peter J. Hutchinson. Decompressive

craniectomy following traumatic brain injury: developing the evidence base, British Journal of Neurosurgery, 2016;30:2,246-250.

- 35. Hartings JA, Vidgeon S, Strong AJ, et al. Surgical management of traumatic brain injury,: a comparative-effectiveness study of 2 centers. *J Neurosurg*. 2014;120:434–446.
- 36. Haselsberger K, Pucher R, Auer LM. Prognosis after acute subdural or epidural haemorrage. *ActaNeurochir (Wien)* 1988;90:111–116.
- 37. Stone JL, Rifai MH, Sugar O, Lang RG, OldershawJB, Moody RA. Subdural hematomas. I. Acute subdural hematoma: progress in definition, clinical pathology, and therapy. *Surg Neurol.* 1983;19:216–231.