

A Comparative Study To Analyse The Effectiveness Of Basal Cisternostomy Combined With Decompressive Craniectomy And Decompressive Craniotomy Alone, It's Impact On The Outcome Of Moderate And Severe Head Injury Patients

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Abstract

Introduction- Severe traumatic brain injury (TBI) is a life-threatening condition which is associated with substantial morbidity and mortality. Cisternostomy is a novel surgical technique that has been proposed to prevent the development of secondary brain injury and treat associated increase in intracranial pressure. Decompressive craniectomy has been shown to reduce ICP, but it actually provides an outlet for brain tissue to expand without reducing edema. Cisternostomy is associated with an improved outcome (both at early and long term), improved brain oxygenation, better control of ICP and shorter ICU stay when compared to standard decompressive craniectomy.

Aims and Objectives- to assess effectiveness of Basal cisternostomy surgery with Decompressive Craniotomy surgery, its impact and outcome post operative complication, morbidity, mortality in moderate and severe brain injury.

Material and methods- All enrolled patients were randomly assigned to 2 groups and assessed clinically and radiologically. TBI were categorised as mild, moderate and severe according to Marshall CT based score. Outcomes were assessed based on Glasgow coma outcome scale on follow up.

Results- Total of 50 patients were randomly assigned (25 patients in each group). Patients in cisternostomy group had decreased mean duration days of ventilator support and ICU stay significantly decreased in morbidity and low rate of complications in cisternostomy group.

Conclusions- Cisternostomy was effective in reducing mortality, morbidity and complications post operatively. Glasgow outcome scale and Marshall score had a significant prognostic impact in management of TBI.

Keywords: basal cisternostomy combined with decompressive craniectomy, decompressive craniotomy, moderate and severe head injury management, ICP, Recent trends in TBI

Introduction

Severe traumatic brain injury (TBI) is a life-threatening condition which is associated with substantial morbidity and mortality (1).

The pathogenesis of TBI includes a primary injury related to a physical injury to the brain and a delayed secondary injury caused by the subsequent molecular, chemical and inflammatory cascades that can result in brain oedema, ischemia and intracranial hypertension.

The burden of traumatic brain injury (TBI) is enormous and disproportionate. TBI causes 111 years of life lived with disability per 100 000, and 80% of its burden occurs in low- and middle-income countries (19). Moreover, there is a disparity in TBI research and innovation. Most TBI research, guidelines, and innovations are developed in high-income countries, where TBI management's epidemiology and resources are more favourable (20-22).

Cisternostomy in the context of severe TBI aims at opening the basal cisterns to atmospheric pressure and tackle the vicious process leading to posttraumatic brain swelling (23). There are two types of cisternostomy based on the mechanism of action: outflow (ventriculocisternostomy and cystocisternostomy) and inflow (cisternostomy proper) (24). Outflow cisternostomies were the first to be described in modern neurosurgery. Arne Torkildsen performed the first successful ventriculocisternostomy in 1937 for cerebrospinal fluid (CSF) diversion, and the intervention was the preferred treatment of noncommunicating hydrocephalus after World War II (25, 26). The idea of inflow cisternostomy was developed in the context of vascular neurosurgery and still represents a valuable microsurgical step routinely carried out during clipping of anterior circulation aneurysms (27). The first mention of inflow cisternostomy for the management of severe TBI was in 2012 by Dr. Cherian from Nepal (28). Cisternostomy is a novel surgical technique that has been proposed to prevent the development of secondary brain injury and treat associated increase in intracranial pressure (2, 3). Decompressive craniectomy is the time-tested and most commonly used neurosurgical procedure available to decrease ICP in TBI. Decompressive

craniectomy has been shown to reduce ICP, but it actually provides an outlet for brain tissue to expand without reducing edema (4)

A previous clinical study of one group (5) has showed that adjuvant cisternostomy is associated with an improved outcome (both at early and long term), improved brain oxygenation, better control of ICP and shorter ICU stay when compared to standard decompressive craniectomy (DC).

A recent randomized trial by Chandra et al.(6)has also confirmed the benefit of cisternostomy in terms of outcome and ICP control when compared to standard DC. Recently, a cerebrospinal fluid (CSF) circulation model has been reconsidered, and it has been stated that CSF can be produced and absorbed throughout the entire CSF system. Pericapillary Virchow-Robin spaces play a critical role in the CSF system.(7)

The glymphatic system has proven that CSF from the cisterns (and not from the ventricles) does communicate with the parenchyma through Virchow-Robin spaces.(8,9)It has been suggested that in TBI, there is a decrease in glymphatic removal of solutes from interstitial fluid, allowing CSF to be shifted from the cerebral cisterns to the brain following TBI(10). Mestre et al. (29) tracked CSF flow in mice after middle cerebral artery stroke and found evidence of CSF shift edema in the ipsilateral hemisphere. Therefore, the rationale of cisternostomy is to open and rinse the basal cisterns allowing a removal of blood products and addressing the altered gradient pressure between subarachnoid space and the brain parenchyma (23)

Cherian and Burhan(11)described cisternostomy for the control of ICP in TBI in 2009. Using this technique, CSF is released from basal cisterns, which reduces cerebral edema and relaxes the brain in acute and subacute settings, thus allowing replacement of bone flap in otherwise irreplaceable settings.

TBI was studied in mice models by Plog et al. (30) who used horizontal cisternotomy to drain CSF from mice that had acute TBI continually. Of note, they found no evidence in favor of cisternostomy preventing the secondary cascade of TBI. The reason is that CSF drainage by the cisterna magna cisternotomy reduces the hydraulic pressure that drives fluid exchange between CSF and interstitial fluid (31). As a result, it inhibits glymphatic efflux, which alters TBI biomarkers' clearance and waste products. Unlike cisterna magna cisternotomy, cisternostomy exposes more cerebral cisterns (interoptic, optico-carotid, lateral carotid, interpeduncular, and prepontine) to atmospheric pressure and removes blood products from the subarachnoid space.

cisternostomy could in the future prove its effectiveness in all TBI cases, some of its effects could not be simply explained by the CSF shift edema theory alone and should perhaps be attributable to the reduced intracranial pressure and the overall optimization of the CSF flow as seen following decompressive craniectomy (32).

AIMS AND OBJECTIVES

To assess effectiveness of Basal cisternostomy surgery with Decompressive Craniotomy surgery, its impact and outcome postoperative complications, morbidity, mortality in moderate and severe brain injury

REVIEW OF LITERATURE

The pathogenesis of TBI includes a primary injury related to a physical injury to the brain and a delayed secondary injury caused by the subsequent molecular, chemical and inflammatory cascades that can result in brain oedema, ischemia and intracranial hypertension. Decompressive craniectomy is the time-tested and most commonly used neurosurgical procedure available to decrease ICP in TBI Recently, a cerebrospinal fluid (CSF) circulation model has been reconsidered, and it has been stated that CSF can be produced and absorbed throughout the entire CSF system

TBI generally results from mechanical impact or acceleration–deceleration insults. The trauma can lead to a spectrum of pathologic manifestations, including hemorrhagic contusion, intracerebral hemorrhage, subarachnoid hemorrhage, and widespread white matter damage. As a result, intracranial pressure increases, contributing to the pathogenesis of TBI. In an experimental study, a paravascular pathway was demonstrated that facilitates cerebrospinal fluid (CSF) flow from the subarachnoid space through the brain parenchyma and the clearance of interstitial solutes (37). This brain-wide network of paravascular channels, termed the “glymphatic” pathway, is structurally located between glial end-foot processes and vascular cells of arterioles, capillaries, and veins. The glymphatic pathway allows CSF influx along almost all penetrating arteries (through the so called Virchow-Robin spaces, which communicate to the aforementioned space from the cisterns), and

efflux along some large and deep veins. It has been reported that the glymphatic pathway reduces its activity by 60% after experimental TBI, thus contributing to the development of brain edema formation (37)

Cytotoxic brain edema is characterized by intracellular water accumulation of neurons, astrocytes, and microglia irrespective of the integrity of the vascular endothelial wall. This pathology is caused by increased cell membrane permeability for ions, ionic pump failure due to energy depletion, and cellular reabsorption of osmotically active solutes (38, 39). Water enters the central nervous system through aquaporin 4, which is located in perivascular astrocyte foot processes. Vasogenic brain edema is caused by mechanical or autodigestive disruption or functional breakdown of the endothelial cell layer of brain vessels. Disintegration of the cerebral vascular endothelial wall allows for uncontrolled ion and protein transfer from the intravascular to the extracellular/interstitial brain compartments with ensuring water accumulation (39,40).

Recent evidence suggests that edema formation also is associated with CSF entrance into the brain parenchyma via the low-resistance para-arterial space or decreased interstitial fluid efflux or a combination of the 2 processes (37).

Glymphatic removal of excess of interstitial fluid is likely decreased after injury or infarction. Accordingly, after TBI, CSF could be shifted from the cerebral cisterns to the brain, leading to a severe brain swelling. One of the reasons for this rapid shift may be the traumatic subarachnoid bleed, often associated with severe head trauma, causing a pressure gradient that is increase in the cisterns and lower in the brain Cheria and Burhan(11)described cisternostomy for the control of ICP in TBI in 2009. Using this technique, CSF is released from basal cisterns, which reduces cerebral edema and relaxes the brain in acute and subacute settings, thus allowing replacement of bone flap in otherwise irreplaceable settings.

Cisternostomy is always performed in conjunction with DC, and such approach represents the last resource in the treatment of medically refractory severe TBI [33,34,35]. Cheria et al. (36) reported lower mortality (15.6% in the cisternostomy group vs. 26.4% in the DC and cisternostomy group vs. 34.8% in the DC group), shorter mechanical ventilation times (2.4 days in the cisternostomy group vs. 3.2 days in the DC and cisternostomy group vs. 6.3 days in the DC group), and better Glasgow outcome scales at 6 weeks (3.9 in the cisternostomy group vs. 3.7 in the DC and cisternostomy group vs. 2.8 in DC group).

Over the years, various studies and clinical trials have failed to evaluate the prognostic value of DHC. To date, the largest clinical trials, DECRA (41) and RESCUEicp (42). have proven DHC to be superior to medical therapy, but the rates of mortality remain a concern. The DECRA trial enrolled 155 patients from 3 countries (Australia, New Zealand, and Saudi Arabia) and showed that neuro protective bifrontal decompression is not helpful and mortality rates are similar with or without surgical treatment (41). Later, the RESCUEicp trial, with a much larger patient population (408 individuals from 20 countries), concluded that decompression for refractory intracranial hypertension and severe TBI reduce the mortality rate (26.9 vs. 48.9% in medical therapy) but is associated with higher rate of disability and vegetative state, particularly in severe head injury patients. the rising cost of health care, rehabilitation, and psycho-socio-economic instability calls for improvement in current standards of TBI management (43). In this context,they propose a novel technique, cisternostomy, which has rapidly gained popularity in the neurosurgical community and has been regarded as one of the surgical options for ICP reduction in moderate to severe brain injury by the Global Neurotrauma Outcome Study, funded by the US National Institutes of Health (44). The introduction of microsurgery into trauma is a much-needed practice considering the beneficial outcomes in all other neurosurgical practices.

Giammattei et al. presents the results of 40 patients with severe TBI (sTBI) treated at Centre Hospitalier Universitaire Vaudois in Lausanne between 2013 and 2018. This was a retrospective study. Of the 40 patients, 22 had a decompressive craniectomy (DC), while 18 had a DC with a cisternostomy. The decision as to whether a cisternostomy would be performed was *only* dependent on the availability of a neurosurgeon with vascular surgery expertise, rather than any clinical characteristics. This raises questions around the statement that “waiver of consent was granted because the procedure was part of our written algorithm for the management of sTBI”, given that the treatment algorithm would be different depending on who was the on-call neurosurgeon. This issue along with the fact that the published data on cisternostomy for TBI are very sparse indicates that the study should have happened prospectively in the context of a research protocol approved by an ethics committee (45). The very limited sample size and the retrospective nature are major sources of bias. With the “subgroup analysis” (primary vs secondary procedure), the sample size becomes even more limited. On this basis, all outcome data should be treated cautiously and, at best, as evidence that in the hands of experienced vascular neurosurgeons the addition of cisternostomy to DC does not seem to lead to worse outcomes.

Another concern is that these findings cannot be necessarily translated to a typical neurosurgical trauma service. Firstly, polytrauma patients were excluded. About one-third of sTBI patients have major extracranial injuries and these are independently associated with mortality (46). Secondly, the workload of the trauma service at Centre Hospitalier Universitaire Vaudois seems to be very limited, with only 50 operations for sTBI over 6 years—which is less than one per month.

Di Cristofori et al. have previously raised concerns as to whether a typical neurosurgical service would be able to offer cisternostomy routinely, assuming that its effectiveness is proven (47). Lausanne (48) have published a case report of stand-alone cisternostomy (i.e., without DC) and have postulated that cisternostomy could become a stand-alone treatment in the future, assuming that multi-centre clinical studies prove its effectiveness .

With the better knowledge of identification of patients in which cisternostomy may act as a damage-control surgery, and keeping decompression as a last tier for ischemic presentations, a number of centers in the world have started to adopt the strategic management plans for trauma.

The results of the initial phase of transformation from performing decompression to a combined cisternostomy with decompression, and, later, cisternostomy only with bone flap placement, could yield significant differences in the overall prognosis in TBI-affected patients.

Methods And Materials

All patients presenting to the Department of Neurosurgery at SRM Medical College Hospital and research centre, Chennai , India, with TBI who needed surgical management Written informed consent from each patient or his or her family member were obtained before the study All enrolled patients who gave consent to participate in the study were randomly assigned to a decompressive craniectomy group and a cisternostomy group.

The randomization sequence was generated before the start of the study Patients willing to participate in the study were exclude from the study Computed tomography (CT) of the skull was performed for every patient, as per institute protocol, to determine the type of injury, hematomas or contusions of brain, volume of hematomas, mass effect, midline shift All TBIs will classified as mild, moderate, and severe injuries based on the clinical findings, GCS and CT findings by Marshall CT-based score as shown in table 1 and table 2 respectively.

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; Moderate Brain Injury = 9-12 points; Severe Brain Injury = 3-8 points		

Table 1 (Glasgow coma score)

	MLS	CISTERNS	High or mixed density lesions	notes
I	none	present	none	No visible pathology
II	0-5 mm	present	none	

SCORE	DESCRIPTIONS
5	Good Recovery
4	Moderate Disability
3	Severe Disability
2	Vegetative State
1	Dead

III	0-5 mm	compressed	none	swelling
IV	>5 mm		none	
V	Any	Any	Any	Any lesion surgically evacuated
VI			>25 cm ³	Not surgically evacuated

Table 2 (marsh CT base score)

SURGERY METHODS

Decompressive Craniectomy:

In the decompressive craniectomy group, standard decompressive craniectomy with a large flap was done with placement of bone flap in the anterior abdominal wall.

Cisternostomy :

In the cisternostomy group, after craniotomy and dural opening, basal cisternostomy, including opening of the interoptic, opticocarotid, and lateral carotid cisterns was done. Duraplasty was done primarily or with a pericranial graft. The bone flap was replaced and fixed with miniplates and screws.

Postoperatively, the patients were monitored for the number of days of ventilator support needed; number of days in the intensive care unit(ICU) , any new neurological deficits in the form of cognitive, motor, or sensory impairment postoperatively; number of days in the hospital; postoperative complications; and mortality and morbidity during follow-up after 3months with the Glasgow Outcome Scale (GOS) shown in table 3

Table 3 – Glasgow outcome scale

Study design

Interventional study design
Randomized controlled trials

Prospective study

Duration –December 2022 – December 2023 [1 year]
Place – SRM Medical college Hospital and research centre , Chennai

Sample Size

Patients will be randomly assigned into 2 groups each containing 25 patients
Cisternostomy - -25 patients
Decompressive Craniectomy -25 patients

Inclusion criteria

Age >18 years and <65 years,
Glasgow Coma Scale > 4,
Brain parenchymal contusions with mass effect and midline shift,
Acute subdural hematoma with mass effect and midline shift,
Traumatic subarachnoid hemorrhage with mass effect and midline shift,
Posttraumatic diffuse edema with mass effect and midline shift.

Exclusion criteria

age <18 years and age >65 years,
GCS score < 4,
extradural hemorrhage,
Nontraumatic subarachnoid hemorrhage,
Nontraumatic intraparenchymal bleed,
Acute infarcts with mass effect

Comparison between the 2 groups was done using Student t test, provided that the data were normally distributed. Statistical analysis was done using IBM SPSS Version 20.0 (IBM Corporation, Armonk, NewYork, USA).

Results

In our study, 50 patients who given consent to participate were randomly assigned to 2 groups with 25 patients each. Comparison between the 2 groups was done using Student t test, provided that the data were normally distributed. Statistical analysis was done using IBM SPSS Version 20.0 (IBM Corporation, Armonk, NewYork, USA).

Variable	Cisternostomy group	Decompressive craniotomy group	P value
Age , years	32.88 +_ 10.89	37.72 +_ 12.27	0.443
18-30	13 (52%)	07 (28%)	
31-40	06 (24%)	07 (28%)	
41-50	03 (12%)	06 (24%)	
>50	03 (12%)	05 (20%)	

The mean age of the patients was 32.88 +_ 10.89 years in the cisternostomy group and 37.72 +_ 12.27 in the decompressive craniectomy group. There were 06 (24%) patients in the cisternostomy group and 11(44%)patients in the decompressive craniectomy group >40 years old.

GCS	7.96 +_ 3.12	8.44 +_ 3.12	0.694
Mild (14-15)	0	0	
Moderate (9-13)	09 (36%)	12 (48%)	
Severe (< 9)	16 (64%)	13 (52%)	

preoperative GCS score was 7.96 +_ 3.12 in the cisternostomy group and 8.44 +_ 3.12 in the decompressive craniectomy group. There were 16 (64%) patients in the cisternostomy group and 13 (52%) patients in the decompressive craniectomy group with severe head injury with GCS score < 9 at the time of presentation.

Time from trauma to surgery, hours	16.08 +_ 12.96	12.52 +_ 5.72	0.405
< 12 hours	10 (40%)	13 (52%)	
2-24 ours	13 (52%)	10 (40%)	
>24 hours	02 (08%)	02 (08%)	

The mean time from trauma to surgery was 16.08 +_ 12.96 hours in the cisternostomy group and 12.52 +_ 5.72 hours in the decompressive craniectomy group. (N.B. - As our institute is a tertiary care center in our region, many cases were referred to here from peripheral centers, so transportation of the patients took some time.)

Intra operative period	Cisternostomy group	Decompressive craniotomy group	
Duration of surgery	2.24 +_ 0.833	2.16 +_ 0.85	

Mean duration of surgery was 2.24 ± 0.833 hours in the cisternostomy group and 2.16 ± 0.85 hours in the decompressive craniectomy group

Marshall CT score	4.28 \pm 1.27	4.20 \pm 1.25	0.570
1	00 (0%)	00 (0%)	
2	02 (8%)	02 (8%)	
3	04(16%)	05 (20%)	
4	11 (44%)	10 (40%)	
5	01 (4%)	02 (8%)	
6	07 (28%)	06(24%)	

The mean preoperative Marshall CT score was 4.28 ± 1.27 in the cisternostomy group and 4.20 ± 1.25 in the decompressive craniectomy group.

Post operative period	Cisternostomy group	Decompressive craniectomy group	P value
M V support	4.40 \pm 1.00	5.00 \pm 0.95	0.088
Duration of ICU stay	5.88 \pm 0.92	6.68 \pm 0.90	0.143
Total duration of hospital stay	8.60 \pm 1.00	9.92 \pm 1.151	0.582

The mean duration of mechanical ventilation support was 4.40 ± 1.00 days in the cisternostomy group and 5.00 ± 0.95 days in the decompressive craniectomy group. The mean duration of ICU stay was 5.88 ± 0.92 days in the cisternostomy group and 6.68 ± 0.90 days in the decompressive craniectomy group. The mean duration of hospital stay was 8.60 ± 1.00 days in the cisternostomy group and 9.92 ± 1.151 days in the decompressive craniectomy group.

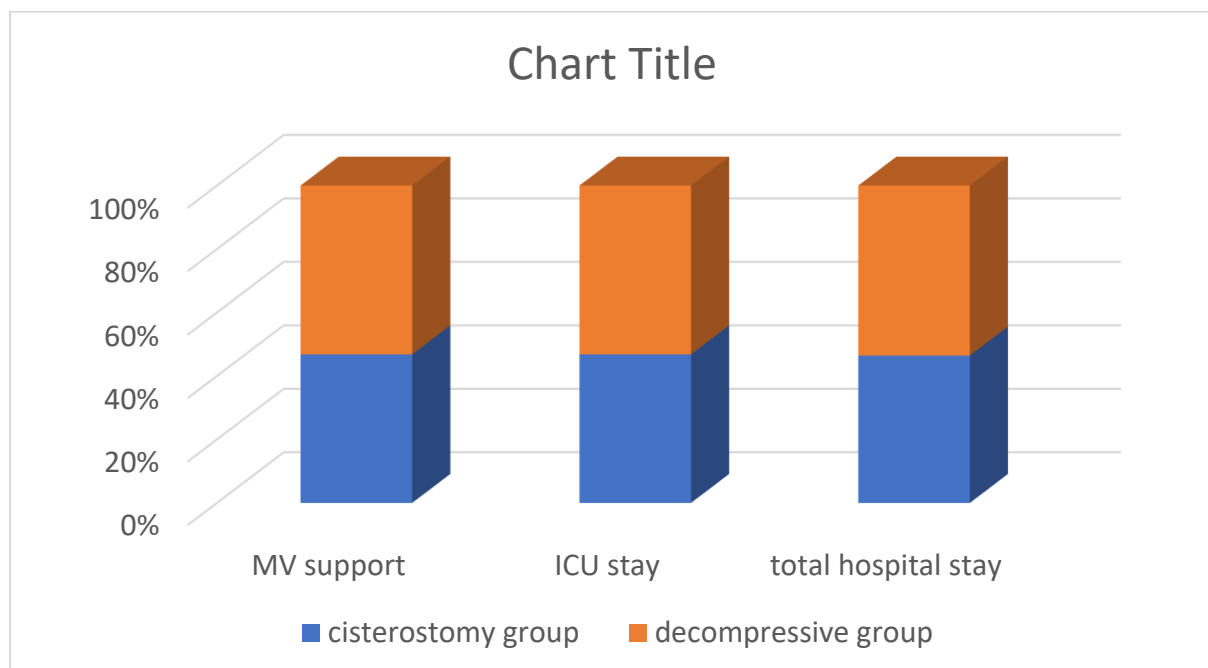


Fig 1 - Bar chart for Post operative period

GOS (after 3 months follow up)

GOS	3.44 \pm 1.50	2.68 \pm 1.72	0.010
5	08 (32%)	06 (24%)	
4	06 (24%)	04 (16%)	
3	05 (20%)	02 (08%)	
2	01 (04%)	02 (08%)	
1	05 (20%)	11 (44%)	

The mortality rate in this study was 20% (n = 5 patients) in the craniostomy group and 44% (n = 11 patients) in the decompressive craniectomy group. These were assigned a GOS score of 1. In this study, mean GOS was 3.44 ± 1.50 in craniostomy group and 2.68 ± 1.72 in decompressive craniectomy group. ; this was statistically significant (P = 0.010).

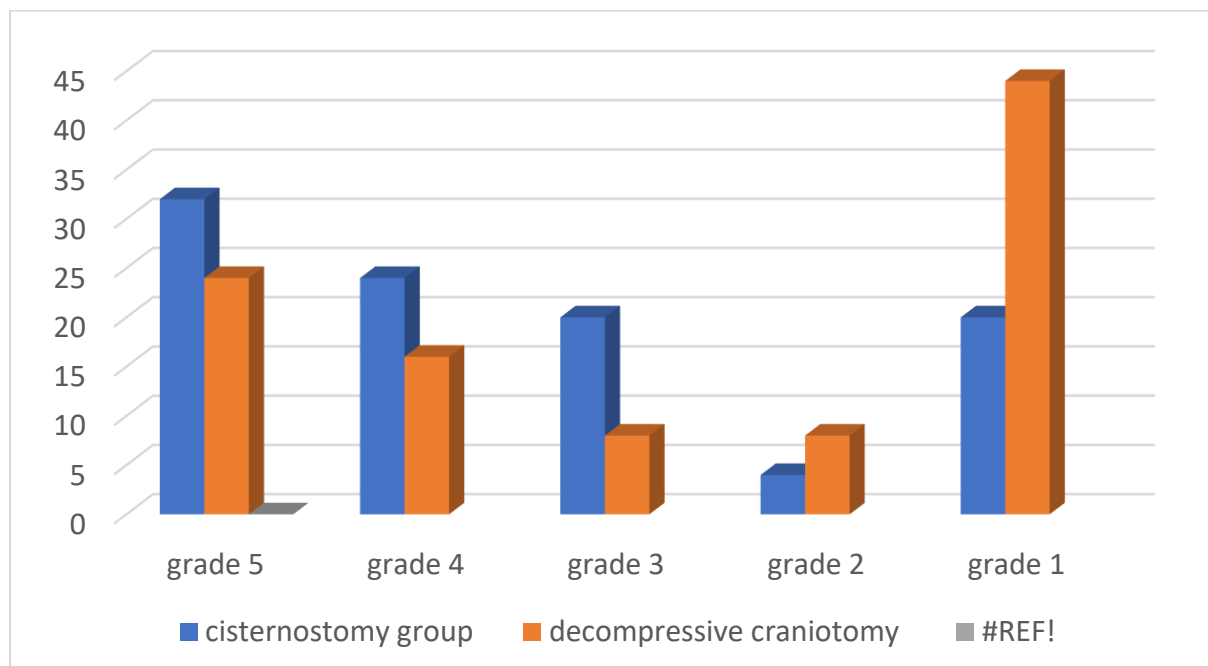
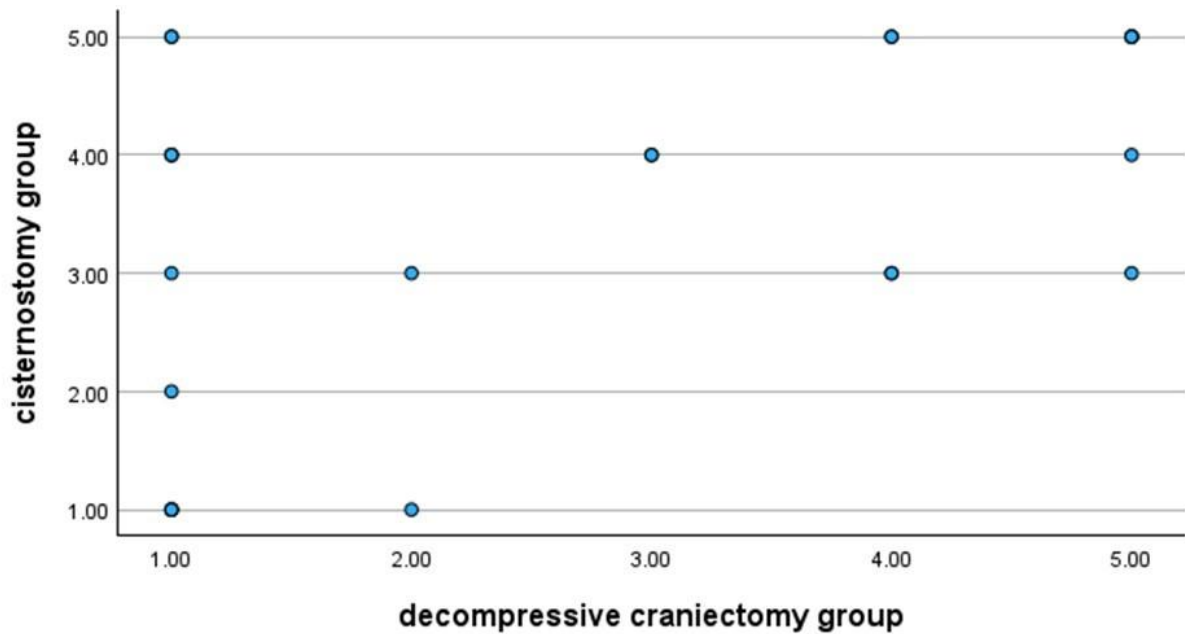


Fig 2 – Graph for GOS (after 3 months follow up)

Relation of prognostic factor of GCS to GOS

Prognostic factor	Craniostomy group	Decompressive craniotomy group	P value

According to Cherian et al.,(21) the average time for cisternostomy from dural opening is approximately 20 minutes with extra time needed in the case of posterior clinoid drilling or any other additional unforeseen circumstances associated with severe head injuries. In our study, Mean duration of surgery was 2.24 ± 0.833 hours in the cisternostomy group and 2.16 ± 0.85 hours in the decompressive craniectomy group. In the study by Cherian et al.,(21) the mortality rate 13.8% for cisternostomy and 34.8% for decompressive hemicraniectomy (DHC), and in our study, the mortality rate was 20% in the cisternostomy group and 44% in the DHC group. Even though the mortality rate was high in our study, it was less in the cisternostomy group. The mean duration on ventilator support and ICU care in this study was lower in the cisternostomy group compared with the decompressive craniectomy group.

Glasgow Outcome Scale

According to Cherian et al.,(21) the mean GOS score was 2.8 for patients treated with DHC and 3.9 for patients treated with cisternostomy. Our study results with a mean GOS score of 2.68 ± 1.72 in the DHC group and 3.44 ± 1.50 in the cisternostomy group with statistically significant (P value = 0.010).

These results were also supported by Giammattei et al.(23) in a retrospective series of 40 patients who underwent either basal cisternostomy or decompressive craniotomy alone. The GOS scores were also significantly better for basal cisternostomy patients at 6 months (61% for basal cisternostomy vs. 35% for decompressive craniotomy).

In a study by Parthiban et al.,(24) basal cisternostomy alone had a favorable GOS score compared with basal cisternostomy combined with decompressive craniotomy (82% vs. 62%).

Prognostic factor of GCS to GOS

Patients with severe head injury (presenting GCS < 9) showed better outcome in the cisternostomy group, which was statistically significant compared with the decompressive craniectomy group (P = 0.02).

Limitations

Our study was limited because it was a single-center study.

Another limitation was the small number of patients, which was due to a smaller number of trauma cases.

Conclusions

Patients have a good GOS score in the postoperative period following cisternostomy. Cisternostomy decreases the number of days of ventilator support, the length of ICU stay and total duration of hospital stays.

Therefore, basal cisternostomy seems like a promising procedure, but performing cisternostomy in TBI is challenging which requires expertise of the surgeon in skull base surgeries and availability of a microscope. With this single randomized controlled trial, we cannot state that it is an alternative procedure for decompressive craniectomy to treat patients with TBI. More large multicenter randomized trials are needed to establish the effectiveness of cisternostomy in the management of TBI.

References

1. Peeters W, van den Brande R, Polinder S, Brazinova A, Steyerberg EW, Lingsma HF, et al. Epidemiology of traumatic brain injury in Europe. *Acta Neurochir (Wien)*. (2015) 157(10):1683–96. doi: 10.1007/s00701-015-2512-7
2. Cherian I, Yi G, Munakomi S. Cisternostomy: replacing the age old decompressive hemicraniectomy? *Asian J Neurosurg*. (2013) 8(3):132–8. doi: 10.4103/1793-5482.121684
3. Giammattei L, Messerer M, Oddo M, Borsotti F, Levivier M, Daniel RT. Cisternostomy for refractory posttraumatic intracranial hypertension. *World Neurosurg*. (2018) 109:460–3. doi: 10.1016/j.wneu.2017.10.085
4. Cooper JD, Rosenfeld VJ, Murray L, et al. Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med*. 2011;364:1493-1502
5. Giammattei L, Starnoni D, Maduri R, Bernini A, Abed-Maillard S, Rocca A, et al. Implementation of cisternostomy as adjuvant to decompressive craniectomy for the management of severe brain trauma. *Acta Neurochir (Wien)*. (2020) 162(3):469–79. doi: 10.1007/s00701-020-04222-y
6. Ramesh Chandra VV, Bodapati Chandra Mowliswara P, Banavath HN, Kalakoti CSR. Cisternostomy vs decompressive craniectomy for the management of traumatic brain injury: a randomized controlled trial. *World Neurosurg*. (2022) 19:S1878-8750(22)00214-5. doi: 10.1016/j.wneu.2022.02.067
7. Oreskovic D, Klarica M. The formation of cerebrospinal fluid: nearly a hundred years of interpretations and misinterpretations. *Brain Res Rev*. 2010;64:241-262.

8. Yang L, Kress BT, Weber HJ, et al. Evaluating glymphatic pathway function utilizing clinically relevant intrathecal infusion of CSF tracer. *J Transl Med.* 2013;11:107erences
9. Iliff JJ, Wang M, Liao Y, et al. A paravascular pathway facilitates CSF flow through the brain parenchyma and the clearance of interstitial solutes, including amyloid B. *Sci Transl Med.* 2012;4:147ra111.
10. Iliff JJ, Chen MJ, Plog BA, et al. Impairment of glymphatic pathway function promotes tau pathology after traumatic brain injury. *J Neurosci.* 2014;34:16180-16193.
11. Cherian I, Burhan H. Outcomes of severe head injury patients undergoing cisternostomy at tertiary care hospital in Nepal. *IJN.* 2019;2:55-59.
12. Cherian I, Beltran M, Landi A, Alafaci C, Torregrossa F, Grasso G. Introducing the concept of “CSF-shift edema” in traumatic brain injury. *J Neurosci Res* 2018;96:744-52.
13. Bulat M, Klarica M. Recent insights into a new hydrodynamics of the cerebrospinal fluid. *Brain Res Rev* 2011;65:99–112.
14. Oreskovic D, Klarica M. The formation of cerebrospinal fluid: Nearly a hundred years of interpretations and misinterpretations. *Brain Res Rev* 2010;64:241-62.
15. Iliff JJ, Wang M, Liao Y, Plogg BA, Peng W, Gundersen GA, et al. A paravascular pathway facilitates CSF flow through the brain parenchyma and the clearance of interstitial solutes, including amyloid beta. *Sci Transl Med* 2012;4:147ra111. doi: 10.1126/scitranslmed.3003748.
16. Iliff JJ, Chen MJ, Plog BA, Zeppenfeld DM, Soltero M, Yang L, et al. Impairment of glymphatic pathway function promotes tau pathology after traumatic brain injury. *J Neurosci* 2014;34:16180-93
17. Hutchinson P, Kolias A, Timofeev I, Corteen E, Czosnyka M, Timothy J, et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. *N Eng J Med* 2016;375:1119-30.
18. 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;80:6-15.
19. James S. L., Theadom A., Ellenbogen R. G. Global, regional, and national burden of traumatic brain injury and spinal cord injury, 1990–2016: a systematic analysis for the global burden of disease study 2016. *Lancet Neurology.* 2019;18:56–87.
20. Carney N., Totten A. M., O'Reilly C., et al. Guidelines for the management of severe traumatic brain injury, fourth edition. *Neurosurgery.* 2017;80(1):6–15
21. Hawryluk G. W. J., Rubiano A. M., Totten A. M., et al. Guidelines for the management of severe traumatic brain injury: 2020 update of the decompressive craniectomy recommendations. *Neurosurgery.* 2020;87(3):427–434.
22. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. Introduction. *Journal of Neurotrauma.* 2007;24(1):S1–S2
23. Cherian I., Grasso G., Bernardo A., Munakomi S. Anatomy and physiology of cisternostomy. *Chinese Journal of Traumatology.* 2016;19(1):7–10
24. Hoz S. S., Alramadan A. H., Hadi A. Q., Salazar L. R. M. Cisternostomy in neurosurgery: a new proposed general classification based on mechanism and indications of the cisternostomy proper. *Journal of Neurosciences in Rural Practice.* 2018;09(04):650–652
25. Torkildsen A. Ventriculo-cisternostomy: a post operative study. *Acta Chirurgica Scandinavica.* 1941;85:p. 254
26. Eide P. K., Lundar T. Arne Torkildsen and the ventriculocisternal shunt: the first clinically successful shunt for hydrocephalus. *Journal of Neurosurgery.* 2016;124(5):1421–1428. doi: 10.3171/2015.1.JNS142659
27. Hernesniemi J., Dashti R., Lehecka M., et al. Microneurosurgical management of anterior communicating artery aneurysms. *Surgical Neurology.* 2008;70(1):8–28.
28. Cherian I. Basal cisternostomy-is it a panacea for traumatic brain swelling? *Journal of College of Medical Sciences-Nepal.* 2012;8(1):1–6
29. Mestre H., Du T., Sweeney A. M., et al. Cerebrospinal fluid influx drives acute ischemic tissue swelling. *Science.* 2020;367(6483):p. eaax7171.
30. Plog B. A., Dashnaw M. L., Hitomi E., et al. Biomarkers of traumatic injury are transported from brain to blood via the glymphatic system. *The Journal of Neuroscience.* 2015;35(2):518–526.
31. . Jessen N. A., Munk A. S. F., Lundgaard I., Nedergaard M. The glymphatic system: a beginner's guide. *Neurochemical Research.* 2015;40(12):2583–2599.
32. Láng J., Ganau M., Prisco L., Bozsik K., Banczerowski P. Syndrome of trephined-underestimated and poorly understood complication after decompressive craniectomy. *Ideggyógyászati Szemle.* 2016;69(7-8):227–232.

33. Carney N., Totten A. M., O'Reilly C., et al. Guidelines for the management of severe traumatic brain injury, fourth edition. *Neurosurgery*. 2017;80(1):6–15. doi: 10.1227/NEU.0000000000001432
34. Hutchinson P. J., Kolias A. G., Timofeev I. S., et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. *New England Journal of Medicine*. 2016;375(12):1119–1130.
35. Ganau M., Prisco L. Comment on “neuromonitoring in traumatic brain injury. *Minerva Anestesiologica*. 2013;79:310–311
36. Cherian I., Yi G., Munakomi S. Cisternostomy: replacing the age old decompressive hemicraniectomy? *Asian Journal of Neurosurgery*. 2013;8(3):132–138. doi: 10.4103/1793-5482.121684
37. Iliff J.J., Wang M., Liao Y., Plogg B.A., Peng W., Gundersen G.A., et. al.: A paravascular pathway facilitates CSF flow through the brain parenchyma and the clearance of interstitial solutes, including amyloid beta. *Sci Transl Med* 2012; 4: pp. 147ra111
38. Stiefel M.F., Tomita Y., Marmarou A.: Secondary ischemia impairing the restoration of ion homeostasis following traumatic brain injury. *J Neurosurg* 2005; 103: pp. 707-714.
39. Unterberg A.W., Stover J., Kress B., Kiening K.L.: Edema and brain trauma. *Neuroscience* 2004; 129: pp. 1021-1029.
40. DeWitt D.S., Prough D.S.: Traumatic cerebral vascular injury: the effects of concussive brain injury on the cerebral vasculature. *J Neurotrauma* 2003; 20: pp. 795-825.
41. Cooper D.J., Rosenfeld J.V., Murray L., et. al.: Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med* 2011; 364: pp. 1493-1502.
42. Hutchinson P.J., Kolias A.G., Timofeev I.S., et. al.: Craniectomy for traumatic intracranial hypertension. *N Engl J Med* 2016; 375: pp. 1119-1130.
43. Dismuke C.E., Walker R.J., Egede L.E.: Utilization and cost of health services in individuals with traumatic brain injury. *Glob J Health Sci* 2015; 7: pp. 156-169.
44. Panero Perez I., Castano Leon A.M., Gandia Gonzalez M.L., Kolias A.: Call to participate in the international study of traumatic brain injury results (Global Neurotrauma Outcomes Study). *Neurocirugia (Astur)* 2019; 30: pp. 77-80.
45. Hirst A, Philippou Y, Blazeby J et al (2019) No surgical innovation without evaluation. *Ann Surg* 269(2):211–220
46. van Leeuwen N, Lingsma HF, Perel P et al (2012) Prognostic value of major extracranial injury in traumatic brain injury: an individual patient data meta-analysis in 39,274 patients. *Neurosurgery* 70(4):811–818 discussion 818
47. Di Cristofori A, Gerosa A, Panzarasa G (2018) Is neurosurgery ready for cisternostomy in traumatic brain injuries? *World Neurosurg* 111:427
48. Giammattei L, Messerer M, Oddo M, Borsotti F, Levivier M, Daniel RT (2018) Cisternostomy for refractory posttraumatic intracranial hypertension. *World Neurosurg* 109:460–463