RESEARCH ARTICLE

Factors affecting the development of hydrocephalus in patients with decompressive craniectomy

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Abstract

Objective: To explore the relationship between decompressive craniotomy and hydrocephalus formation and identify the various factors leading to post-traumatic hydrocephalus in patients with traumatic brain injury.

Methods: The prospective cohort study was conducted at the Department of Neurosurgery, Shifa International Hospital, Islamabad, Pakistan, from April 2023 to April 2024, and comprised patients regardless of age and gender who presented with traumatic brain injury to the emergency room and underwent a plain computed tomography of the brain to determine the management plan: conservative or decompressive craniotomy. Demographical, clinical, and radiological parameters were noted, including age, gender, Glasgow Coma Scale score on arrival, and midline shift on pre-operative scans. Data about the mean area/diameter of the bone flap, site of craniectomy, and medial midline margin of craniectomy on post-operative scans were also noted. An Evans ratio <0.3 was labelled as hydrocephalus on a post-operative scan done 6-8 weeks after decompressive craniotomy. Data was analysed using SPSS 21.

Results: Of the 80 patients, mean age of 47.67±9.34 years, 41(51.25%) were males and 39(48.75%) were females. Overall, 25(31.25%) patients developed hydrocephalus. There was a significant association of hydrocephalus with Glasgow Coma Scale score 11-13 on arrival (p=0.004), midline shift >25mm at the time of hydrocephalus diagnosis (p=0.000), area of the bone flap (p=0.001), bilateral craniectomy (p=0.029), and medial margin of craniectomy <25mm (p=0.032).

Conclusion: About one-third of traumatic brain injury patients undergoing decompressive craniotomy were found to have hydrocephalus, which had a significant association with Glasgow Coma Scale score on arrival, midline shift at the time of diagnosis, area of the bone flap, bilateral craniectomy, and medial margin of craniectomy.

Key Words: Decompressive craniectomy (DC), Hydrocephalus (HCP), Glasgow Coma Scale (GCS), Post-traumatic hydrocephalus (PTH), Traumatic brain injury (TBI).

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Introduction

Traumatic brain injury (TBI) is defined as an acute, accelerating, and decelerating injury to the head of the patient, either from blunt or penetrating forces, excluding congenital or degenerative problems.[1] To increase intracranial volume, a portion of cranial bone is removed in a surgical procedure known as decompressive craniectomy (DC). The Monro-Kellie doctrine explains that by providing more space for an oedematous traumatic brain, intracranial pressure (ICP) can be reduced. This principle was first applied by Kocher in 1909 to a refractory TBI that did not respond to medical therapy.[2]

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In TBI patients, DC has evidence of decreasing mortality as it is a lifesaving procedure to relieve intracranial hypertension (ICH)[2], providing better neurological recovery at 12 months and improved long-term prognosis.[3-6] Post-traumatic hydrocephalus (PTH) is a known complication observed in many patients undergoing DC. If ignored, this can result in detrimental effects.[7,8]

Although the contributing factors remain controversial[8], it is diagnostically challenging in patients with a history of trauma due to abnormal anatomy, and there are post-surgery challenges due to large craniotomy defects.

The incidence varies 10-36% in the literature in adults.[7, 9, 10], but a study with a large cohort of paediatric patients reported the incidence of PTH to be as low as 0.07% in patients aged <21.[11]

Various factors, such as age[9,10], larger craniectomy area/flap diameter[12,13], prolonged coma, bilateral DC[12], and medial margin distance from midline[6], can lead to the formation of PTH after a decompressive surgery in TBI

patients. Sequelae in such patients from HCP can include delayed recovery, memory loss, incontinence, psychomotor retardation, gait disturbances, emotional disorders, long-term neurological deterioration, and increased length and cost of hospitalization.[14-16] Early ventriculoperitoneal (VP) shunting is needed in most patients for good results in rehabilitative phases.[17]

Post-DC HCP is a treatable complication, and being aware of the contributing factors can help neurosurgeons improve patient outcomes and recovery. The current study was planned to explore the relationship between DC and HCP formation and to identify the various factors leading to PTH in patients with TBI.

Patients and Methods

The prospective cohort study was conducted at the Department of Neurosurgery, Shifa International Hospital, Islamabad, Pakistan, from April 2023 to April 2024. After approval from the institutional ethics review committee, the sample size was calculated using the World Health Organization (WHO) STEPS Sample calculator[18] with a confidence level of 95%, an anticipated population proportion of 29%[19], and an absolute precision of 10%. The sample was raised using a non-probability consecutive sampling technique. These included patients regardless of gender and age who presented with TBI to the emergency room (ER), having indications of raised ICP or diffuse brain injury, like obliteration of central cisterns, acute subdural hematoma>10mm on pre-operative scans, and undergoing DC. Patients with polytrauma, immediate brain death, pregnant females, massive subarachnoid haemorrhage (SAH), previous head surgery in the preceding 6 months or cranioplasty, and/or intraventricular haemorrhage (IVH) were excluded. Written informed consent was obtained from all the participants.

A trainee researcher conducted history-taking and physical examination, countersigned by consultant neurosurgeons. Initial demographic data, such as age, gender, mechanism of injury, Glasgow Coma Scale (GCS) score on arrival, and midline shift on initial imaging, were retrieved from medical records. The presence of intracranial pathologies, including intracranial bleed (ICH) and/or intraventricular haemorrhage (IVH) and subarachnoid haemorrhage (SAH), was confirmed on plain computed tomography (CT) of the head, while diffuse axonal injury (DAI) was confirmed on plain magnetic resonance imaging (MRI) of the brain. The Rotterdam CT Score[20] guided surgical decisions for the patients. Invasive neuromonitoring devices, such as the use of external ventricle drains (EVDs) and intraparenchymal intracranial pressure (ICP) monitors, were used as per hospital policy for TBI patients. Patients were managed conservatively until they had a refractory elevated ICP of >25mmHg, underwent surgery as per the Trauma Brain Foundation recent guidelines.[21]

Unilateral or bilateral DC was performed, and the size of the DC was measured on post-operative CT scans. The two-dimensional (2D) area of the bone flap was estimated on post-operative CT scan as area = largest AP diameter (D) x diameter perpendicular to D (d) x 7/4.[22]

The distance from the sagittal suture to the craniectomy site was calculated on post-operative CT scans for the medial craniectomy margin. HCP was determined based on the Evans ratio, calculated by dividing the ventricular width by the largest biparietal distance between the inner tables of the skull. An Evans ratio[23] of 0.30 or greater was defined as HCP. Patients were assessed 6-8 weeks after DC for HCP development.

Data was analysed using SPSS 21. Quantitative variables were reported as mean ± standard deviation (SD) or as median with interquartile range (IQR), depending on data normality. Frequencies and percentages were calculated for categorical variables. Factors affecting HCP, such as age, gender, initial GCS on arrival, midline shift on pre-operative CT head, bone flap area/diameter, craniectomy site, and medial craniectomy margin from the sagittal suture, were controlled by stratification, and chi-square tests were applied post-stratification. P≤0.05 was considered significant.

Results

Of the 80 patients with mean age 47.67±9.34 years, 41(51.25%) were males and 39(48.75%) were females females. Overall, 25 (31.25%) patients developed HCP.

The highest HCP frequency was observed in the elderly aged 45-60 years (15 [60%]) and in male patients (16 [64%]) (p=0.116).

Central cistern obliteration was present in 37 (46.25%) patients, as seen in Table 1, while 47 (58.75%) had intracranial bleed (ICB) and/or IVH, and SAH on CT head (plain), with 33 (41.25%) having confirmed DAI on MRI brain.

The descriptive statistics of key clinical and radiological parameters are summarized in Table 2. The mean GCS score on arrival was 7.25±2.14 (range: 4-15). GCS score 11-13 on arrival showed a significant association with HCP frequency (p=0.001).

The mean extent of midline shift was 22±5.95mm (range:

Table-1: Association Between Central Cistern Obliteration and Post-Surgical Hydrocephalus.

	Present	Absent	Total
CCentral Cistern obliteration	47 (58.75%)	33 (41.25%)	80
Post-Surgery HCP	25 (31.25%)	56 (68.75%)	80

HCP: Hydrocephalus.

Table-2: Descriptive Statistics of Clinical and Radiological Parameters.

	Mean	SD	Min	Max
Midline shift (mm)	20.38	7.57	10	35
GCS	7.25	2.14	4	11
Area of bone flap (mm2)	90.45	9.25	80	110
Craniotomy margins from midline (mm)	25.38	9.03	10	43
Post DC GCS	8.29	1.21	4	15

GCS: Glasgow Coma Scale, DC: Decompressive craniectomy, SD: Standard deviation.

Table-3: Descriptive Statistics of Clinical and Radiological Parameters.

Parameters	Division of parameters	Present (n=25)	Absent (n=55)	*P*-Value
Age (years)	16-30	6 (24%)	15 (27.27%)	0.927
	31-40	4 (16%)	10 (18.18%)	
	40-65	15 (60%)	30 (54.54%)	
Gender	Male	16 (64%)	25 (45.45%)	0.116
	Female	9 (36%)	30 (54.54%)	
Mean area of bone flap (mm^2)	80-90	19 (76%)	16 (29.09%)	0.000
	91-100	6 (24%)	27(49.09)	
	101-107	0 (0%)	7 (12.72%)	
Craniectomy site	Unilateral	20 (80%)	53 (96.36%)	0.029
	Bilateral	5 (20%)	02 (3.63%)	
GCS score on arrival	3-6	1 (4%)	23 (41.81%)	0.000
	7-10	2 (8%)	27 (49.09%)	
	11-13	22 (88%)	5 (9.09%)	
Extent of midline shift	< 25 mm	24 (96%)	27 (49.09%)	0.000

HCP: Hydrocephalus, GCS: Glasgow Coma Scale.

10-35mm). The extent of midline shift >25mm was significantly associated with the HCP formation (p=0.001).

Of the total, 11 patients underwent ICP monitoring, while 27 patients had EVD as a measure of ICP monitoring. The method of ICP monitoring had no statistical relevance to the outcome of the patients (p>0.05).

Mean bone flap area was 90.45±9.25mm^2 (range: 80-110mm^2). Patients with a smaller bone flap (80-90mm2) ended up developing PTH (p=0.001).

The mean medial craniectomy margin from the midline distance was 25.38±9.035mm (range: 10-43mm). Patients with medial craniectomy margins from the midline distance of <25mm ended up developing PTH (p=0.032).

In 73(91.25%) patients, the craniectomy site was unilateral, while 7(8.75%) patients had bilateral decompression. The frequency of HCP was lower with a unilateral craniectomy site compared to a bilateral DC (p=0.029), as seen in Table 3. The mean GCS score post-DC was 8.29±1.21.

Discussion

TBI is a leading cause of traumatic deaths and morbidity. Measures to save high-risk patients from long-term complications should be meticulously covered. A common complication observed after DC for TBI is subdural hygroma and HCP, although the exact cause remains controversial.[7] Subdural hygroma was first defined in 1932 as a collection of cerebrospinal fluid (CSF) post-DC seen on imaging after head trauma, which can, in turn, lead to the formation of communicating HCP, eventually needing a CSF diversion procedure.[7]

Clinicians worldwide use different methods to diagnose HCP, resulting in an incidence of PTH ranging from 10-36% in adults.[7, 9,10] However, the incidence of PTH in the paediatric population is smaller.[11] PTH can disrupt brain function/metabolism and delay recovery if missed during the post-operative period. However, there is significant heterogeneity in the literature regarding the factors leading to PTH in TBI patients due to differences in enrolment criteria and evaluation methods.

DC done to relieve ICH has been linked to the formation of PTH[6], and similar evidence was noted in the current study, with 31.25% patients developing HCP in the cohort. However, decompressive surgery for TBI is not an isolated factor for PTH.[24] Factors leading to HCP and subdural hygroma following a DC and its association with the type of HCP were shown in a study[25] in which 26.09% patients developed PTH.

Younger age has been associated with the formation of PTH[7,26], but in the current study, HCP was seen developing more in the elderly population.

Most local series of TBI patients have reported a predominance of male subjects, which aligns with the current results.[27] However, in studies done in Pakistan, the number of females is typically lower than that noticed in the current study.[28] This could be due to differences in regional, demographic, and sociocultural factors across the samples.

GCS at arrival as a risk factor for PTH formation following a DC in traumatic head injury has been controversial, as some studies included it as a risk factor for HCP formation, while others disregarded the theory.[8,9] However, in the current study, GCS at arrival was inversely associated with HCP formation post-operatively; that is, patients who

underwent early decompression with a good GCS ended up developing PTH. A recent study[29] with a huge sample size showed the correlation between GCS >13 as a protective marker for shunting.

Multiple factors, like a large craniotomy defect, subdural hygroma, SAH with thick distribution, midline shift of <25mm, re-do surgery, low temporal lobe perfusion, and increased duration of coma, have been recognized to influence PTH association with decompression.[2,10,13,25,30]

Midline shift on initial CT head plain has been associated with formation of post-operative HCP.[20] The current study reported similar findings.

An ideal decompression should be of at least 12cm, and complications have been reported by removing a small or larger bone flap.[13,25], including the formation of post-operative communicating HCP in decompressive surgery patients suffering from TBI. These results are in line with those of the current study.

If DC margins are close to the midline, this can cause increased venous outflow to sinuses due to external force on the bridging veins. Ventricular enlargement is caused by extracellular fluid absorption and a decrease in parenchymal brain volume[3], and this was seen in the current study as well.

Also, the current patients who underwent bilateral DC ended up developing PTH, which was consistent with an earlier study.[26]

One interesting factor to avoid PTH formation is to perform an early cranioplasty within 2 months of decompression.[31] A correct and timely diagnosis of PTH is needed to identify patients who would benefit from CSF diversion procedures, including VP shunting or endoscopic third ventriculostomy (ETV).[32]

Literature reviews have shown a relationship between higher injury severity score (ISS)[7], nature of injury ICB/DAI[9], SAH[10], delayed cranioplasty[10], and HCP, along with various relevant factors, but these factors were beyond the scope of the current study, which is the major limitation of the present study. The relationship of various risk factors to PTH development in the paediatric population is also very controversial[11], but beyond the scope of the study, as it did not have any patients aged <13 years. Also among the limitations of the current study are its short duration, a smaller sample size, and single-centre data. Removing these limitations would lead to more informed data regarding gender distribution in patients with TBI.

Conclusion

About one-third of TBI patients undergoing DC were found to have HCP, which had a significant association with GCS score on arrival, midline shift at the time of diagnosis, area of the bone flap, bilateral craniectomy, and medial margin of craniectomy. Patients with TBI and a history of DC should be monitored during the recovery phase for early detection and timely management by HCP to aid in better recovery.

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AUTHOR'S CONTRIBUTION:

MA & SVH: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.

HM: Data collection and statistical analysis.