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RESEARCH

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Modified frontal horn index: a novel risk predictor for sunken flap syndrome in the patients undergoing shunt procedures for post-decompressive craniectomy hydrocephalus

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Abstract

Background Decompressive craniectomy (DC) is a neurosurgical procedure, frequently used in lowering the refractory intracranial pressure (ICP) following traumatic brain injuries. Post-traumatic hydrocephalus (PTH), a debilitating complication in the patients with traumatic brain injuries, occurs in 11.9–36% patients undergoing DCs. Sunken flap syndrome (SFS) is a rare entity, following DCs or cerebrospinal fluid (CSF) diversion procedures for PTH after DCs and leads to neurological deterioration of the patients. Literature regarding risk factors associated with SFS in the patients undergoing ventriculoperitoneal shunt procedures for hydrocephalus following DCs is scarce. The aim of this study is to determine the incidence of SFS and to establish a relationship between several clinico-radiological features and SFS in patients undergoing shunt procedures for PTH.

Results This retrospective study was conducted in a tertiary care trauma centre upon 60 patients who underwent shunt procedures for PTH. Intraventricular haemorrhage ($P < 0.0001$), communicating-type hydrocephalus ($P = 0.0006$), and modified frontal horn index ($P < 0.0001$) were significantly associated with development of SFS. MFHI > 43 was a significant risk factor in development of SFS.

Conclusions SFS is the common complication following shunt procedures for PTH after DCs. MFHI is significant risk predictor for SFS. MFHI > 43 is associated with higher chances of developing SFS following shunt insertion in PTH. Early cranioplasty following DCs might prevent development of SFS.

Keywords Hydrocephalus, Decompressive craniectomy, Sunken flap syndrome, Modified frontal horn index, Ventriculoperitoneal shunt

Background

Decompressive craniectomy (DC) is a neurosurgical procedure in which a large part of the skull bone is removed and the duramater is opened. It is used to reduce refractory intracranial pressure (ICP) after traumatic brain injuries (TBIs) [1]. Post-traumatic hydrocephalus (PTH) is a debilitating complication in patients with traumatic brain injuries, occurring

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in 11.9–36% of patients after DCs [2]. PTH is defined by several criteria which including modified frontal horn index (MFHI, the greatest width of the frontal horns divided by the bicortical distance in the same plane) > 33% (Fig. 1), enlargement of the temporal horns, lateral ventricles and third ventricles, and transependymal edema [3]. Sunken flap syndrome (SFS) is a rare entity after DCs or cerebrospinal fluid (CSF) diversion procedures for PTH after DCs that can lead to neurological deterioration in patients [4]. The incidence of SFS is still not precisely known. SFS, also known as 'the sinking skin syndrome' or 'syndrome of the trephined', was first described by Grant and Norcross in 1939 [5]. The symptoms of SFS range from neurological to psychological features, which includes postural headaches, motor and cognitive impairment, slurred speech, and possibly coma or death [6]. The pathophysiology of SFS is still being researched. After DC, atmospheric pressure is transmitted directly to the intracranial compartment, pushing scalp inwards which can be further exacerbated by CSF diversion procedures, such as ventriculoperitoneal (VP) shunt surgery [7, 8]. The identified risk factors for predicting SFS in the patients undergoing DC are a large sized DC, a distance of the medial craniectomy border from the midline (MMD) of less than 2.5 cm and multiple surgeries [9]. There is little literature on risk factors associated with SFS in patients undergoing VP shunt procedures for hydrocephalus after DCs.

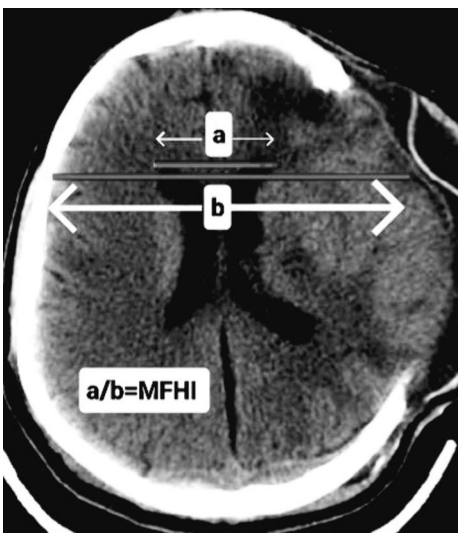

Fig. 1 Schematic diagram showing method of calculation of modified frontal horn index (MFHI); a = largest width of the frontal horns, b = bicortical distance in the same plane, MFHI = a/b

The aim of this study is to determine the incidence of SFS in patients undergoing shunt procedures for PTH. The another main objective was to establish a relationship between several clinical and radiological features with SFS in the patients undergoing shunt procedures for PTH and to ascertain risk factors that may predict development of SFS with special attention towards MFHI.

Methods

This retrospective study was conducted in a tertiary care trauma centre in the Department of Neurosurgery over period of 18 months from June 2021 to November 2022. A total of 564 DCs were performed during this period. Indications for surgery were diffuse cerebral edema with or without midline shift together with subdural or parenchymal lesions. The surgical procedures involved unilateral or bilateral frontotemporoparietal DC. Unfortunately 16 patients, who underwent bilateral DCs, died within 15 days, and were, therefore, excluded. In addition, a total of 106 patients who died within 15 days of surgery, a period that we believe is not long for PTH to develop, were excluded from the study. Ultimately, a total of 422 patients were included in this study, of which 60 patients developed PTH. The criteria for PTH were: MFHI > 33%, enlargement of the temporal horns, lateral ventricles and third ventricle along with transependymal edema. All patients were treated with VP shunt procedures after diagnosis of PTH. Chhabra's medium pressure VP shunt was used as it was easily available and inexpensive. During surgery, the VP shunt was inserted via Keen's point, Frazier's point, or Kocher's point, depending on the surgeon's preference. Initial demographic variables such as age, gender, mode of injury, and level of consciousness (GCS) at the time of admission were recorded.

Initial radiological parameters were used as intraventricular haemorrhage (IVH) and subarachnoid haemorrhage (SAH). Postoperative radiological parameters were used: MMD (patients were categorised into two groups: MMD less than 2.5 cm and more than 2.5 cm), subdural hygroma (SDHy), and interhemispheric hygroma (IHH). PTH was categorised into two groups: communicating type and obstructive type.

Other additional variables recorded were GCS score at the time of detection of PTH, side of DC (right or left sided), hospital stay, and ventilator stay.

Patients were divided into two groups on the basis of development of SFS following VP shunt surgeries: the patients with SFS (group A) and the patients without SFS (group B), and comparative analysis was done.

Outcome analysis was done between both groups on Glasgow outcome scale (GOS) at 6 months following trauma.

The presentation of the categorical variables was done in the form of number and percentage (%). The quantitative data with normal distribution were presented as the means ± SD and the data with non-normal distribution as median with 25th and 75th percentiles (interquartile range). The data normality was checked using Kolmogorov–Smirnov test. Comparison of the variables which were quantitative and not normally distributed in nature was analysed using Mann–Whitney test (for two groups) and Kruskal–Wallis test (for more than two groups) and variables which were quantitative and normally distributed in nature were analysed using independent t test (for two groups) and ANOVA (for more than two groups). If a cell had an expected value of less than 5, Fisher's exact test was used. Univariate analysis and multivariate logistic regression were performed to identify the most significant risk factors for SFS. The receiver operating characteristic (ROC) curve was used to determine cut-off value, sensitivity, specificity, positive predictive value, and negative predictive value of MFHI for the prediction of SFS.

Data entry was done in Microsoft excel spreadsheet and final analysis was done using of Statistical Package for Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, ver 25.0.

For statistical significance, a *P* value of less than 0.05 was considered statistically significant.

Results

Of the total of 422 patients enrolled in the study, 14.21% ($n=60$) patients developed PTH. In a total of 60 patients who developed PTH after DCs in TBIs, 25% ($n=15$) of patients developed SFS after insertion of VP shunt (group A) and rest of the patients were categorised into group B. The mean age of patients who developed PTH was 34.2 ± 11.1 years with a male predominance (66.7%, $n=40$). Road traffic accidents were the most common mode of injury (66.7%). 88.53% ($n=53$) of patients had communicating-type hydrocephalus (Table 1).

There were no significant differences in terms of age [32.87 ± 14.47 (group A) and 34.64 ± 9.88 (group B), $P=0.595$], gender ($P=0.067$), and mode of injury ($P=0.148$). The mean GCS score on admission was [8.33 ± 1.35 (group A), 7.91 ± 1.95 (group B), $P=0.441$] and at the time of detection of PTH was [8.93 ± 0.7 (group A), 9.73 ± 2.57 (group B), $P=0.064$] (Table 2). Among the preoperative radiological findings, IVH was significantly associated with the development of SFS

Table 1 Descriptive analysis of patients who developed post-traumatic hydrocephalus following decompressive craniectomy for traumatic brain injuries

Age (years)	
Mean ± SD	34.2 ± 11.1
Median (25th–75th percentile)	34.5 (25–39.5)
Range	16–60
Gender	
Female	20/60 (33.33%)
Male	40/60 (66.67%)
Mode of injury	
Fall from height	10/60 (16.67%)
Fire	1/60 (1.67%)
Physical assault	9/60 (15.00%)
Road-traffic accidents	40 (66.67%)
Intraventricular haemorrhage	10/60 (16.67%)
Subarachnoid haemorrhage	23/60 (38.33%)
Side of decompressive craniectomy	
Right	37/60 (61.67%)
Left	23/60 (38.33%)
Distance from midline (cm)	
> 2.5	28/60 (46.67%)
< 2.5	32/60 (53.33%)
Subdural hygroma	28/60 (46.67%)
Interhemispheric hygroma	33/60 (55.00%)
GCS at admission	
Mean ± SD	8.02 ± 1.82
Median (25th–75th percentile)	8 (7–9)
Range	4–12
GCS at time of development of hydrocephalus	
Mean ± SD	9.53 ± 2.27
Median (25th–75th percentile)	9 (8–10)
Range	6–15
Modified frontal horn index	
Mean ± SD	40.53 ± 5.1
Median (25th–75th percentile)	39 (36.75–43.5)
Range	34–55
Type of hydrocephalus	
Communicating	53/60 (88.33%)
Obstructive	7/60 (11.67%)
Sunken flap	15/60 (25.00%)
Duration of hospital stay (days)	
Mean ± SD	28.02 ± 13.89
Median (25th–75th percentile)	23 (17.75–39)
Range	10–72
Duration of ventilator stay (days)	
Mean ± SD	13.72 ± 10.12
Median (25th–75th percentile)	9 (6–22)
Range	1–45

GCS Glasgow coma scale, *GOS* Glasgow outcome scale

Table 2 Comparative analysis of patients developing sunken flap syndrome (SFS) with those who did not develop SFS following shunt procedures for post-traumatic hydrocephalus after decompressive craniectomy

Parameters	Present ($n=15$)	Absent ($n=45$)	Total ($n=60$)	*P* value
Age (years)	32.87 ± 14.47	34.64 ± 9.88	34.2 ± 11.1	0.595‡
Gender				
Female	2 (13.33%)	18 (40%)	20 (33.33%)	0.067*
Male	13 (86.67%)	27 (60%)	40 (66.67%)	
Mode of injury				
Fall from height	2 (13.33%)	8 (17.78%)	10 (16.67%)	0.148*
Firearm injuries	0 (0%)	1 (2.22%)	1 (1.67%)	
Physical assault	5 (33.33%)	4 (8.89%)	9 (15%)	
Road-traffic accidents	8 (53.33%)	32 (71.11%)	40 (66.67%)	
Intraventricular haemorrhage	8 (53.33%)	2 (4.44%)	10 (16.67%)	< 0.0001*
Subarachnoid haemorrhage	6 (40%)	17 (37.78%)	23 (38.33%)	0.878†
Side of decompressive craniectomy				
Right	7 (46.67%)	30 (66.67%)	37 (61.67%)	0.168†
Left	8 (53.33%)	15 (33.33%)	23 (38.33%)	
Distance from midline (cm)				
> 2.5	8 (53.33%)	20 (44.44%)	28 (46.67%)	0.55†
< 2.5	7 (46.67%)	25 (55.56%)	32 (53.33%)	
Subdural hygroma	8 (53.33%)	20 (44.44%)	28 (46.67%)	0.55†
Interhemispheric hygroma	7 (46.67%)	26 (57.78%)	33 (55%)	0.454†
GCS at admission	8.33 ± 1.35	7.91 ± 1.95	8.02 ± 1.82	0.441‡
GCS at time of development of hydrocephalus	8.93 ± 0.7	9.73 ± 2.57	9.53 ± 2.27	0.064‡
Type of hydrocephalus				
Communicating	9 (60%)	44 (97.78%)	53 (88.33%)	0.0006*
Obstructive	6 (40%)	1 (2.22%)	7 (11.67%)	
Duration of hospital stay(days)	26.93 ± 12.2	28.38 ± 14.52	28.02 ± 13.89	0.731‡
Duration of ventilator stay(days)	11.73 ± 7.67	14.38 ± 10.81	13.72 ± 10.12	0.308‡
GOS at 6 months	2.67 ± 0.82	2.71 ± 1.25	2.7 ± 1.15	0.899‡
Modified frontal horn index	47.53 ± 4.29	38.2 ± 2.61	40.53 ± 5.1	< 0.0001‡

‡ Independent t test, * Fisher's exact test, † Chi square test

GCS Glasgow coma scale, *GOS* Glasgow outcome scale

in the univariate analysis ($P<0.0001$) (Table 2). Association of SAH was nonsignificant ($P=0.878$). MMD, ($P=0.55$), side of DC ($P=0.168$), SDHy ($P=0.55$), and IHH ($P=0.454$) were not associated with development of SFS. Communicating-type hydrocephalus was associated with development of SFS ($P=0.0006$). MFHI [47.53 ± 4.29 (group A), 38.2 ± 2.61 (group B), $P<0.0001$] was a significant risk factor in development of SFS (Table 2).

Table 3 Multivariate logistic regression to find out significant risk factors of sunken flap syndrome

Variables	Beta coefficient	Standard error	*P* value	Odds ratio	Odds ratio lower bound (95%)	Odds ratio upper bound (95%)
Modified frontal horn index	0.459	0.128	0.000	1.582	1.231	2.034
Intraventricular haemorrhage	2.246	2.511	0.371	9.450	0.069	1296.027
Type of hydrocephalus						
Communicating				1.000		
Obstructive	− 0.189	2.834	0.947	0.828	0.003	213.757

Table 4 Receiver operating characteristic curve of modified frontal horn index for predicting sunken flap syndrome

Variables	Values
Area under the ROC curve (AUC)	0.944
Standard error	0.0505
95% confidence interval	0.853–0.987
P value	< 0.0001
Cut off	> 43
Sensitivity (95% CI)	93.33% (68.1–99.8%)
Specificity (95% CI)	97.78% (88.2–99.9%)
PPV (95% CI)	93.3% (68.1–99.8%)
NPV (95% CI)	97.8% (88.2–99.9%)
Diagnostic accuracy	96.67%

ROC receiver operating characteristic curves, *AUC* area under curve, *CI* confidence interval

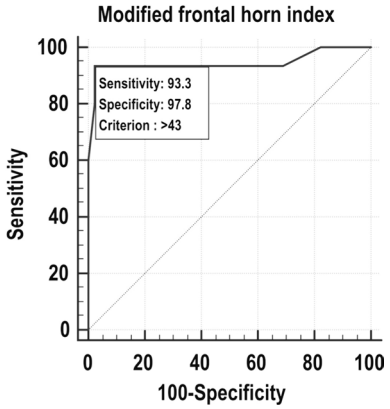

Fig. 2 Receiver operating characteristic curve of modified frontal horn index for predicting the sunken flap syndrome

Hospital stay [26.93 ± 12.2 (group A) and 28.38 ± 14.52 (group B), $P = 0.731$] (in days) and ventilator stay [11.73 ± 7.67 (group A) and 14.38 ± 10.81 (group B), $P = 0.308$] (in days) were not associated with development of SFS. Outcome (GOS) was independent of presence of SFS [2.67 ± 0.82 (group A) and 2.71 ± 1.25 (group B), $P = 0.899$] (Table 2).

Multivariate logistic regression was applied to the above-mentioned significant variables of univariate analysis. We found only independent predictor of development of SFS: MFHI ($P = 0.000$, odds ratio: 1.582, confidence interval: 1.231–2.034 (Table 3).

Receiver operating curves (ROC) were plotted and analysed by calculating area under curve (AUC) to establish how MFHI predicted the development of MFHI. We found that sensitivity was 93.33% and specificity was 97.78% with 96.67% diagnostic accuracy. An AUC of 0.944 was identified with confidence interval 85.3–98.7%, $P < 0.0001$. Cutoff value of MFHI for predicting SFS was 43 (Table 4, Fig. 2).

Discussion

DC has been proposed as a life-saving procedure in patients with refractory high ICP after TBIs [10]. This procedure is associated with a significant reduction in mortality but unfortunately also with numerous complications that increases patient morbidity [11]. PTH after DC is a debilitating complication that significantly affects patient's quality of life [2, 7, 10, 11]. Although the incidence of PTH as reported in the literature, ranges from 0.7% to 86%, according to recent studies 11.9–36% of all patients undergoing DC may develop PTH, with the reduction in numbers likely due to limitation of diagnostic criteria [2, 10–12]. Several risk factors for predicting PTH after DCs have been identified, including age, IVH, craniectomy margins < 2.5 cm from the midline, SDHy, and IHH [2, 3, 11–14]. The pathophysiology of PTH after DCs remains poorly understood in the literature. Disturbance of CSF flow dynamics, malabsorption of CSF due to mechanical blockage of the arachnoid granulations by blood or blood products, and obstruction of CSF flow in the ventricles due to IVH are the main pathophysiological mechanisms responsible for the development of PTH [15]. DCs can disrupt normal dicrotic ICP pulsatile waveforms that drive CSF from the subarachnoid space into the venous sinuses. Impairment of proper drainage of CSF following loss of pulsatile ICP dynamics after DCs can lead to decrease in CSF flow which may result in PTH [16, 17]. The treatment of PTH after DC is challenging. CSF diversion procedures such as VP shunt and external ventricular drainage are routinely used to treat PTH after DCs. The decision of simultaneous cranioplasty and CSF diversion procedures largely depends on the patient's general condition and their efficacy is still under research [18, 19].

SFS is one of the main complications after VP shunt insertion in patients who have developed PTH [20] (Figs. 3, 4). The incidence of SFS in patients of PTH undergoing shunt procedures is not defined; however, in our study, 25% patients developed SFS after shunt insertion for PTH. Our finding shows that SFS following shunt insertion in patients of PTH following DCs is not uncommon. SFS is characterized by the sinking of the scalp on the side of DC, deep beyond the edges of the bone defect along with neurological deterioration. Sinking of parenchyma of side of DC towards contralateral side can lead to paradoxical brain herniation, which in turn causes debilitating neurological deficits [21]. Negative pressure gradient between atmospheric and intracranial pressure across skin flap over DC site leads to inwards shifting of

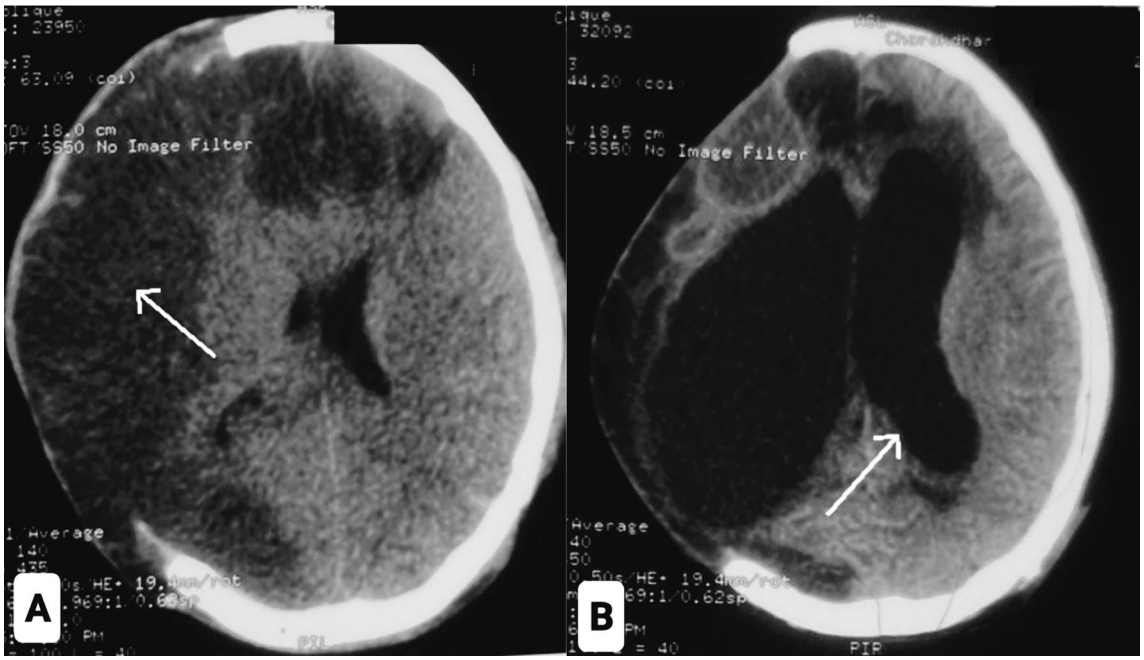

Fig. 3 **A** Postoperative axial computed tomography (CT) scan of the head of a patient showing massive external brain herniation (thin arrow) with diffuse cerebral edema after decompressive craniectomy for traumatic brain injury **B** Axial CT scan head of same patient at 39th day of surgery showing hydrocephalus (thin arrow) with massive transependymal edema

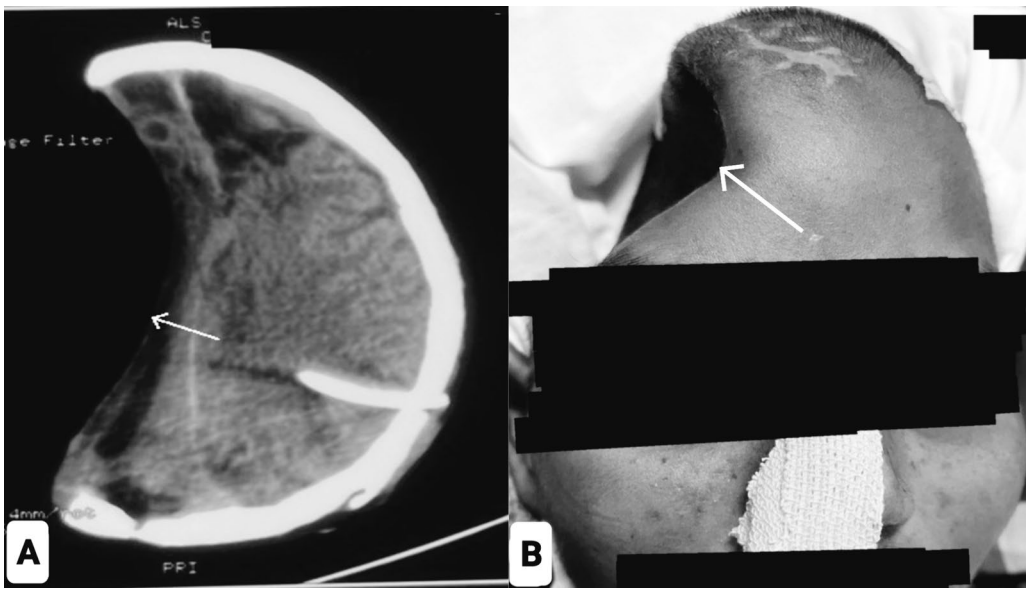

Fig. 4 **A** Axial Computed Tomography (CT) head of same patient mentioned in this figure showing sunken flap (thin arrow) after ventriculoperitoneal shunt insertion, **B** Clinical image of same patient showing inwardly sunken skin flap (thin arrow)

the flap, which is further aggravated by CSF diversion procedures [20, 22].

The development of PTH after DC is a complex process. After DC, the brain parenchyma shifts towards the operative site and mechanical or inflammatory blockage of subarachnoid spaces occurs (rebound phase). Disruption of pulsatile ICP dynamics leads to obstruction of CSF outflow (hydrodynamic phase) [3]. Both rebound and hydrodynamic phases are required for the development of PTH. PTH can lead to raised

ICP which leads to decreased compliance in the brain [23]. After VP shunt insertion, a significant pressure gradient is created across the skin flap on the DC site. This positive atmospheric pressure causes inwards sinking of the brain parenchyma and the skin flap. Hypothetically, we propose a nomenclature for this phase as 'reversal phase' and this phase is necessary to cause SFS after VP shunt insertion in the patients developing PTH following DCs. Decreased brain compliance has a greater propensity to shrink following shunt insertion [24, 25].

Risk factors predicting the development of SFS after VP shunt procedures for PTH after DCs are poorly understood and not well-described.

In this study, risk factors predicting SFS in the patients with PTH following DCs were IVH, communicating-type hydrocephalus and MFHI. MFHI was the only significant factor found in multivariate analysis.

Although earlier studies showed that the size of the cerebral ventricles did not predict ICP [26], increased MFHI along with other features like transependymal edema might reflect a raised ICP. Therefore, higher MFHI is probably an indicator of raised ICP, which further signifies reduced compliance of the brain. Reduced compliance of brain is the best predictor of rapid and marked reduction of the ventricular size [24–26]. Therefore, authors conclude that MFHI might be an indirect causative element for development of SFS after VP shunt insertion in the patients of PTH.

In this study, MFHI values have high sensitivity and specificity to predict the development of SFS in the patients of PTH following DCs. MFHI > 43 was highly associated with the development of SFS.

Triple F therapy, (1) flat head end, (2) fluid administration, and (3) flap replacement (autologous bone or mesh), is the cornerstone of the management of SFS [27]. The role of early cranioplasty in SFS is not well-elaborated, but some studies have shown the advantageous effect of early cranioplasty in SFS [28, 29]. Since SFS is a sequelae of PTH following shunt procedures, we propose that early cranioplasty should be considered in patients of PTH following DCs with MFHI > 43, although to verify this statement, we need more large scale prospective cohorts.

Previous literature suggest that early cranioplasty (within 3 months of DCs) are associated with lower incidence of PTH [30, 31] due to restoration of normal CSF circulation dynamics. So indirectly, early cranioplasty can be preventive measure for development of SFS.

This study is not without limitations. The retrospective nature of this study is prone to bias. A large-scale prospective study could have produced more authentic and significant results. The discussion of treatment strategies in patients of this study could have been more informative regarding validation of conclusion, such as benefits of early cranioplasty.

Conclusions

PTH is a debilitating complication following DCs for TBIs. SFS is one of the common complications following shunt procedures for PTH after DCs. MFHI is significant risk predictor for SFS. MFHI > 43 is associated with higher chances of developing SFS following shunt insertion in PTH. Early cranioplasty following DCs might prevent the development of SFS. We hope that this study adds valuable information regarding our knowledge of SFS in patients of PTH after DCs.

Abbreviations

DC	Decompressive craniectomy
ICP	Intracranial pressure
TBI	Traumatic brain injury
PTH	Post-traumatic hydrocephalus
SFS	Sunken flap syndrome
MFHI	Modified frontal horn index
VP Shunt	Ventriculoperitoneal shunt
SAH	Subarachnoid haemorrhage
SDHy	Subdural hygroma
IHH	Interhemispheric hygroma
IVH	Intraventricular haemorrhage

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Author contributions

Data acquisition was performed by VY; analysis of data was conducted by AS and RS; drafting of the manuscript was written by MM and RP; conception and design was provided by NP; critical revision was done by AS and NP. All authors read and approved the final manuscript.

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All patients evaluated in this study were operated and managed in the hospital for free.

Availability of data and materials

All data that support the findings of this study are available from the neurosurgery department of Institute of Medical Sciences, Banaras Hindu University. Data are, however, available from the author when requested with permission.

Declarations

Ethics approval and consent to participate

This study was conducted after getting ethical clearance from institutional review board (IRB), Banaras Hindu University on 24 May 2021. Written informed consent was waived by committee because of observational nature of the study.

Informed consent

We confirm that all data incorporated into this study are anonymized. Consent for participation was obtained from each patient prior to the study. Clinical image of the patient was taken with proper consent of relatives of the patient. All the figures used in manuscript is original and not used elsewhere.

Consent for publication

Not applicable.

Competing interests
The authors declare that they have no competing interests.

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