Turk Neurosurg 33(1):10-17, 2023

Original Investigation

DOI: 10.5137/1019-5149.JTN.30032-20.2

Received: 01.04.2020
Accepted: 23.05.2020

Published Online: 04.03.2021

Post Traumatic Hydrocephalus in Indian Subpopulation: An Institutional Experience

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ABSTRACT

AIM: To identify subgroups of cases with ventriculomegaly who will benefit from the cerebrospinal fluid (CSF) diversion procedures, n patients with post-traumatic hydrocephalus (PTH), and to identify the risk factors for its development to minimize its occurrence.

MATERIAL and METHODS: We analyzed 500 head injury cases who were admitted over a one and a half year period in our institute with PTH, to assess them for treatment benefit by CSF diversion procedures and associated risk factors for its development. The patients were assigned to one of three groups: Group 1 had ventriculomegaly with periventricular lucency (PVL), and raised intracranial pressure (ICP) 2.1% (10/500). Group 2 had ventriculomegaly with PVL, and normal opening pressure 1.75% (7/500) and Group three had no ventriculomegaly 95.75% (483/500).

RESULTS: The incidence of radiological PTH in this study was 3.4% (17/500), and it developed after an average interval of 16.43 ± 23.7 (SD) in Group 1 and 19.76 ± 8.9 (SD) weeks in Group 2. Lower Glasgow Coma Score (GCS) ($p<0.001$), decompressive craniotomy ($p<0.001$) and requirement for prolonged ventilatory support ($p<0.001$) were significantly associated with the development of PTH. Significantly better results were found in cases with PTH and high opening CSF pressure (≥ 15 mmHg) on Lumbar puncture ($p<0.001$). Decompressive craniotomy cases required significantly more shunt revisions compared to conservatively managed cases ($p<0.05$).

CONCLUSION: CSF diversion procedures help to improve ventriculomegaly cases with documented evidence of raised ICP but not in cases without raised ICP. The subgroup of PTH, which cannot be treated by CSF diversion procedures, can only be managed by minimizing many of the risk factors for its development. In cases with severe head injury, a low GCS, and prolonged ICU stay, decompressive craniotomy should be used judiciously. The duration of mechanical ventilation should be minimized and combined with necessary measures to improve GCS.

KEYWORDS: Hydrocephalus, CSF dynamics, CSF diversion, Post-traumatic, Risk factors

ABBREVIATIONS: AIS: Abbreviated injury score, **CSF:** Cerebrospinal fluid, **EDH:** Extradural hematoma, **GCS:** Glasgow Coma Score, **ICP:** Intracranial pressure, **IVH:** Intraventricular hemorrhage, **LOS:** Length of stay, **MV:** Mechanical ventilator, **NPH:** Normal pressure hydrocephalus, **SAH:** Subarachnoid hemorrhage, **SDH:** Subdural hematoma, **PTH:** Post-traumatic hydrocephalus

INTRODUCTION

Post-traumatic hydrocephalus (PTH) is one of the uncommon complications of brain injury responsible for a failure to improve, or neurological deterioration. It has been reported to occur in 2–30% of cases. Cerebrospinal fluid (CSF) diversion procedures have mixed results in such cases. A few studies suggest that cases with a high opening CSF pressure tend to have good results. Studies have suggested measuring parameters such as resistance to CSF flow, compliance of the CSF fluid space, ventricular and lumbar opening pressure, and tracing of B waves (12-14,18). Measuring opening pressure and mean CSF pressure in such cases is easy to carry out and helps in decision making. The identification of risk factors

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that may help to minimize the severity of PTH is not easy as such factors are not directly responsible for its development. Poor GCS, prolonged mechanical ventilator (MV) support, and decompressive craniotomy can activate a pathological pathway leading to hypoxia and fluctuating CSF pressure. This causes periventricular atrophic changes and oozing of CSF through the ependymal lining from the ongoing injury. The culmination of these events is increased resistance in the CSF drainage pathway leading to raised intracranial pressure (ICP) and worsening neurological condition. Comparing the results of the CSF diversion procedure in the subgroups with and without a raised ICP helps to understand where CSF diversion is more effective and minimize the use of this invasive procedure and, therefore, its complications.

In this study, we aimed to find out the comparative results of CSF diversion in head injury cases diagnosed with PTH with or without raised ICP. We also assessed the association of risk factors to identify cases that would not have benefited from CSF diversion procedures.

Improving our understanding will help reduce the likelihood of developing PTH and helping to improve the management of these patients.

MATERIAL and METHODS

This study was carried out in the Department of Neurosurgery at our institute on over 500 head injury cases admitted between July 2018 and December 2019. The records of brain injury cases who had ventriculomegaly with periventricular lucency (PVL) were traced. Institutional ethical clearance with detailed patient informed consent to participate in the study was taken, with institute ethical committee permission no.IEC/2020/205.

Inclusion & Exclusion Criteria

Ventriculomegaly and PVL developing after an initial normal ventricular appearance were included. We excluded cases that had ventricular dilatation before sustaining the head injury as history suggestive of infective intracranial pathology. Ventricular dilatation due to obstructive pathology such as a congenital anomaly, brain tumors, and normal pressure hydrocephalus were excluded as were those with a history of spontaneous intraparenchymal, intraventricular bleed, and subarachnoid hemorrhage due to nontraumatic causes.

The enrolled patients were divided into three groups. Group 1 had ventriculomegaly and PVL with raised ICP as measured on lumbar CSF tapping to be greater than 15 mmHg; Group 2 included cases with ventriculomegaly and PVL and with a CSF pressure between 10–15 mmHg and Group 3 included cases with no ventriculomegaly and PVL. These cases were managed conservatively. Mannitol injection and diuretics (furosemide) were used to reduce cerebral edema, with other supportive treatment. Details of the clinical features at the time of admission were noted, including age, sex, Glasgow Coma scale, neurological deficits, seizures, loss of consciousness, headache, vomiting, etc. We also recorded the noncontrast computed tomography (NCCT) findings immediately following head injury. These included the presence of intradural and extradural bleeds, cerebral contusions, mass effect indicated by midline shift, the Evans index, periventricular lucency, and ventriculomegaly. The Evans index (ratio of maximum width at the level of the frontal horn to the maximum internal diameter at the same level) was calculated, and a ratio greater than 0.3 was classed as ventriculomegaly. Hypodensity around the ventricles was labeled as PVL.

The decision to measure the CSF opening pressure was made mainly due to deteriorating GCS level or a lack of improvement in the clinical features together with the presence of ventriculomegaly and PVL on a screening head NCCT. Lumbar CSF opening pressure with mean pressure was measured over 30 minutes, and it was confirmed at the time of introducing the cannula into the ventricle by connection to the pressure transducer system.

Physician observations and blood pressure evaluation were continuously measured to analyze CSF pressure only during artifact-free periods and to exclude arterial hypertension. Rapid changes in pressure synchronous with respiration and with systole, ranging between 10–35 mmHg, were considered normal CSF pulsations. Abnormal pressure pulsations in the form of B waves were identified as repetitive pulsations in the CSF occurring one to six times per minute for periods of more than 10 minutes. An opening pressure and an average pressure above 15 mmHg were classed as high, 10–15 as intermediate, and below 10 as normal.

We also considered other factors which may be relevant to the development of PTH such as duration of hospital stay, the number of days in ICU, days on MV support, together with other systemic injuries as measured with the abbreviated injury score (AIS) ≥3. The AIS score helped with the evaluation of systemic injury, for example to limb, abdomen, chest, pelvis, or spine. Such cases generally required surgical intervention, which could have secondarily aggravated the brain injury. Coagulopathy and the presence of shock at initial presentation were also analyzed for an association with PTH. Although these factors are not primarily related to the development of PTH, they may contribute to its development. Hypoxia and fluctuating ICP while on MV support may aggravate an already evolving pathological cascade with impending periventricular atrophy and periependymal translocation of intraventricular fluid leading to secondary hypoxic brain injury and prolonging the length of stay in hospital.

Spss.20 version was used for data entry and analysis. Since the samples selected for the study were nongaussian and slightly skewed to the right, the significance level was tested using nonparametric formulas; the Chi-square test, Fischer's exact test, and the Mann Whitney test. For analysis of the independent contribution of predictive factors, a p-value of <0.05 was considered statistically significant.

RESULTS

There were 17 (3.4%) cases out of 500 that met the inclusion criteria for radiological PTH. The patient population was further subcategorized into the three described groups. Of the 500

cases, there were 10 (2%) in Group 1, 7 (1.4%) cases in Group 2 and 484 (96.8%) in Group 3 (Table I). Male and female were present in the ratio of 4.6:1 in Groups 1 and 2 (with PTH) and 1.09 in Group 3 (no PTH). The mean age in Group 1 was 38.24 ± 18.9 (SD), Group 2 was 42 ± 22.83 (SD), and Group 3 had a mean age of 45 ± 29.25 (SD) years (Table I). The difference in mean age at presentation was not statistically significant. In both groups, the majority of cases were the victims of a road traffic accident, and the next most frequent cause was falling from a height. The severity of the head injuries was quantified and rated on the GCS (Table I), and the findings were 64.70% (11/17) cases of PTH (7/10 in Group 1& 4/7 in Group 2) and 14.46% in Group 3 (70/484 cases), and it was significantly associated with the development of PTH ($p<0.001$). In all, 35.29 % of cases (3/10 in Group 1, and 3/7 in Group 2, of a total 17 cases with PTH) had moderate grade head injury (GCS 9–12) as compared to 34.50% (167/484) in the non-PTH category. Moderate head injury was also responsible for the development of PTH, but it was not significantly associated with it ($p<0.218$) (Table I). Radiological features were noticed mostly on head NCCT and ventriculomegaly together with periventricular lucency were assessed on subsequent follow up with NCCT of the head (Figure 1, 2). All the cases in the PTH group had an Evans ratio greater than 0.3 and were significantly associated with the development of PTH. Cerebral contusion was the commonest initial finding on NCCT of the head in cases that developed PTH and was found in 64.70% (11/17: 7/10 in Group 1, 4/7 cases in Group 2) and in 21.90% cases in Group 3 (106/484). The presence of cerebral contusion (Figure 1) was significantly associated with the development of PTH. It was followed in descending order by acute subdural hematoma (SDH), subarachnoid hemorrhage (SAH), and intraventricular hemorrhage (IVH) in both groups (Table I). The average period before the detection of ventriculomegaly with PVL on head NCCT (Figure 2) was 16.43 ± 23.7 weeks in Group 1 and 19.76 ± 8.9 weeks in Group 2 (Table I). The mean CSF opening pressure in Group 1 was 22.24 ± 8.92 (SD) mm Hg and 10.21 ± 8.71 (SD) mmHg in Group 2, while the mean pressure measured over a 30 minute period was 22.83 ± 18.21 in Group 1 and 12.34 ± 7.43 in Group 2 but it was not significantly associated with the development of PTH. An improvement in the GCS was noticed in 9 out of 10 cases (90%) in Group 1, but only 1 case of 7 (14.28%) with GCS < 12 improved. An improvement in the GCS was noticed in cases with a low GCS (<8) of around 85.7% (6/7) with raised opening pressure. One case each in Group 1 and Group 2 developed septicemia, ventriculitis, and multiorgan dysfunction syndrome and finally succumbed to his illness and died (Table I).

The duration of ICU stay and length of stay (LOS) in days on ventilator support was found to be significantly associated with the development of PTH. The hospital LOS in cases with ventriculomegaly was 36 ± 18.94(SD) days and without ventriculomegaly was 12.43 ± 26.6 (SD) days. The LOS in ICU in cases with ventriculomegaly was 18.32 ± 14.65 (SD) days and 9.83 ± 14.1 (SD) days in cases without ventriculomegaly. The requirement for MV support was 18 ± 13.43 (SD) days in cases with ventriculomegaly and 9.83 ±14.1 (SD) days in cases without ventriculomegaly. Other systemic injury, although associated with brain injury, was not significantly contributory to the development of PTH (Table II).

Results of CSF Diversion Procedure (Table III)

The mean time interval following head injury to the diagnosis of PTH in Group 1 was 16.43 ± 23.7 (SD) weeks and 19.76 ± 8.9 (SD) weeks in Group 2. Of these 8 cases (4 in Group 1 and 4 in Group 2) who had earlier undergone craniotomy, 4 cases required a revision of shunt surgery on 7 occasions (4 revisions due to blockage at the distal end and 3 due to shunt infection). The average interval between shunt malfunction and subsequent revision was 12 ± 9.8 (SD) weeks, in Group 1 and 15.24 ± 9.64 (SD) weeks in Group 2. EVD was done three times in patients who had developed shunt infection. It was replaced with an Omaya reservoir later on with intraventricular instillation of antibiotics at regular intervals. It was finally replaced with a VPS after the ventriculitis had subsided. Nine patients (6 in Group 1 and 3 in Group 2) who were managed conservatively without craniotomy had undergone VPS after being diagnosed with PTH. Of these, 2 had shunt malfunctions (due to blockage at the abdominal end) leading to neurological deterioration. The remaining 7 cases did not require revision. The 9 shunt revisions were due to blockage of the shunt at the distal end in 6 (67%) and shunt infection 3 (33%) (Table III).

Follow up (Table III)

Following VPS in cases with ventriculomegaly (17/500), neurological improvement was seen in (9 out of 10 cases in Group 1 and 1 case out of 7 in Group 2, as measured on the Glasgow outcome score with a mean follow up of 16 ± 14.92 (SD) months in Group 1 and 12.34 ± 11.45 (SD) months in Group 2. A total of 11.76% (2/17) cases succumbed to septicemia and died, and 58.82% cases (9/10 in Group 1 & 1/7 in Group 2) ultimately improved radiologically with a decrease in the size of the ventricles and with reduced periventricular lucency on NCCT (Figure 3A, B).

DISCUSSION

The recognition of PTH as a complication of head injury occurred after it was first reported by Dandy and Blackfan in 1914 (5). It is an important secondary brain injury that needs early attention. It is responsible for the neurological deterioration or a failure to improve in critically ill patients with a poor GCS (GCS ≤ 8), and sometimes in cases with moderate head injury (GCS 9-12). Ventriculomegaly, and periventricular lucency on NCCT, reflects the development of hydrocephalus, but it may be either due to atrophic changes or defects in the absorption of CSF. It is difficult to establish the etiology on radiological findings alone. The incidence of PTH reported in the literature varies between 0.7–29 %. A few studies had reported its incidence at around 30%–86% when only radiological criteria of ventriculomegaly were used. In the present study, 3.4% of cases (17/500) had ventriculomegaly with PVL, but only 2% (10/500) cases were found to have true hydrocephalus (ventriculomegaly with PVL and raised ICP) as established after lumbar puncture CSF pressure monitoring and measuring opening pressure during the introduction of the VPS.

Table I: Demography and Clinical Profile of Brain Injury Cases

Variables	Ventriculomegaly with mean pressure >15 mmHg n=10 (Group 1)	Ventriculomegaly with CSF pressure <15 mmHg n=7 (Group 2)	Brain injury cases without ventriculomegaly n=483 (Group 3)	p (significant when <0.05)
Sex				
Male	8 (80%)	6 (85.71%)	245 (50.82%)	
Female	2 (20%)	1 (14.28%)	238 (28.51%)	
Age	38.24 ± 18.9	42 ± 22.83	45 ± 29.25	0.078
Mechanism				
Traffic accidents	9 (90%)	6 (60%)	410 (84.71%	0.660
Fall	1 (10%)	1 (14.28%)	74 (15.28%)	0.650
Brain lesions				
Cerebral contusion	7 (70%)	4 (57.14%)	106 (21.90%)	**0.001**
Acute EDH	3 (30%)	-	33 (6.81%)	0.139
Acute SDH	3 (30%)	2 (28.57%)	38 (7.85%)	0.234
Traumatic SAH	2 (20%)	1 (14.28%)	22 (4.54%)	0.168
Traumatic Intraventricular haemorrhage	2	-	20 (4.13%)	-
Mideline shift in mm	12 ± 8.43	11.21 ± 7.48	45 (9.29%)	0.814
Evaans ratio				
<0.03	-	-	55 (11.36%)	
>0.03	10	7	-	**0.039**
Duration between accident and detection of ventriculomegaly with PVL (weeks)	16.43 ± 23.7	19.76 ± 8.9	-	0.221
Lumbar CSF opening pressure	22.24 ± 8.92	10.21 ± 6.72	-	**0.001**
Mean pressure	22.83 ± 18.21	12.34 ± 7.43	-	**0.018**
Highest peak pressure	24.29 ± 14.82	12.69 ± 9.87	-	**0.001**
Initial GCS				
<8	7 (70%)	4 (57.14)	70 (14.46%)	**0.014**
8-12	3 (30%)	3 (42.85)	167 (34.50%)	0.218
12-15	-	-	247 (51.03%)	-
Clinical outcome				
GCS<8				
Gcs>8	6	1	118 (24.38%)	**0.001**
GCS 12-15	3	-	310 (64.09%)	0.223
Mortality	1	1	22 (4.54%)	0.128

***- :** Means no data available, **CSF:** Cerebrospinal fluid, **EDH:** Extradural hematoma, **SDH:** Subdural hematoma, **SAH:** Subarachnoid hemorrhage, **PVL:** Periventricular lucency, **GCS:** Glasgow coma scale.*

Table II: Associated Risk Factors for PTH

Sl.no	Risk factors	Cases without ventriculomegaly (n=483)	Cases with ventriculomegaly & CSF pressure >10 mmHg (n=17)	p (significant when <0.05)
1	Hospital LOS in days	32.43 ± 26.6 (p≤0.001)	36 ± 18.94	**0.001**
2	ICU LOS in days	30.23 ± 17.81 (p≤0.001)	38.32 ± 14.65	**0.001**
3	Mechanical ventilation LOS in days	19.83.0 ± 14.1 (p≤0.001)	17.14 ± 13.43	**0.001**
4	MOF	17 (3.51%)	1 (6.25%)	0.246
5	Sepsis	8 (1.65%)	1 (6.25%)	0.341
6	Coagulopathy	6 (1.23%)	2 (12.50%)	0.276
	Other systemic injury			
7	AIS score abdomen ≥3	9 (1.85%)	2 (12.50)	0.451
8	AIS score chest ≥3	12 (2.46%)	2 (12.50)	0.243
9	AIS score pelvis ≥3	5 (1.03%)	2 (12.50)	0.378
10	AIS score limbs ≥3	15 (3.09%)	4 (25%)	0.215
11	Operations for other injuries	17 (3.51%)	2 (12.50%)	0.413
12	Shock at admission	5 (1.03%)	1 (6.25%)	0.212
13	Blood transfusion	22 (4.54%)	8 (50%)	0.213
14	Mortality	22 (4.54%)	2 (12.50%)	0.434

PTH: *Post-traumatic hydrocephalus,* ***ICU:*** *Intensive care unit,* ***LOS:*** *Length of stay,* ***AIS:*** *Abbreviated injury score.*

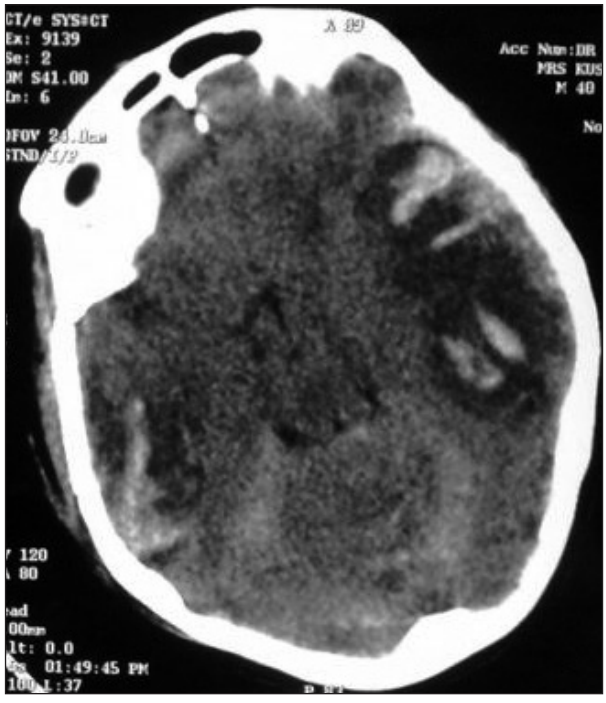

Figure 1: Head non-contrast computed tomography (NCCT) of a 60-year old female immediately after head injury showing multiple contusions in bilateral frontal and temporal lobes with surrounding edema.

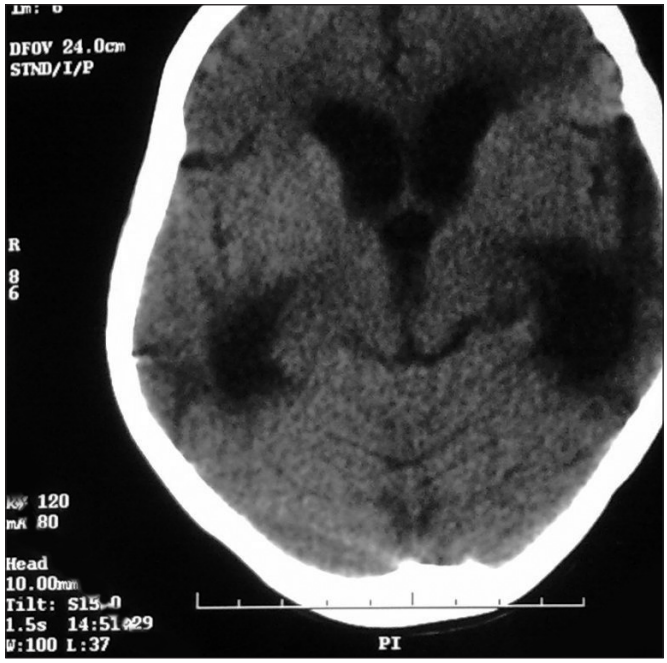

Figure 2: NCCT head done in the same case 3 months later following neurological deterioration showing dilatation of the third and lateral ventricles with periventricular lucencies.

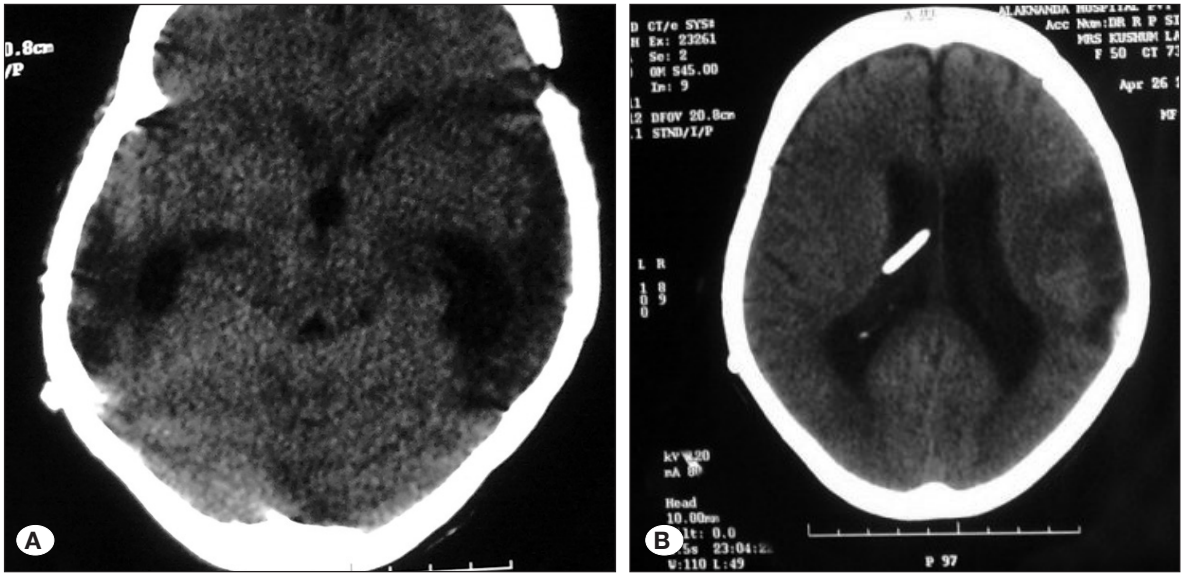

Figure 3: A) NCCT head 2 months after ventriculoperitoneal shunt for PTH in the same case revealed a decrease in the size of the frontal and temporal horns with reduced periventricular lucency. **B)** NCCT head in the same case at a higher level shows the proximal shunt tip in the atrium and the ventricle size was decreased compared to the previous scan.

Table III: Result of CSF Diversion Procedure in PTH Cases

Initial procedure	CSF pressure 10-15 mmHg (Group 1)	CSF pressure>15 mmHg (Group 2)	p (significant when <0.05)
Decompressive Craniotomy prior to developing PTH	4	4	
VP shunt in cases of craniotomy	4	4	**0.001**
VP shunt in cases with initial conservative treatment	6	3	0.238
EVD	3	5	0.347
Omaya reservoir placement	3	5	0.542
Revision (no of shunt surgery)	7	6	**0.001**
Mean Duration required for revision. After primary VP shunt in weeks	12 ± 9.8 (SD)	15.24 ± 9.64 (SD)	0.238
Follow up duration in months	16 ± 14.92 (SD)	12.34 ± 11.45 (SD)	0.342

CSF: *Cerebrospinal fluid,* ***VP Shunt:*** *Ventriculo-peritoneal shunt,* ***EVD:*** *External ventricular drainage.*

In this study and others, male cases have been observed to be more predisposed to PTH than females (4.6:1), which is even higher with the gender predominance seen in head injuries in normal and in present study 1.09:1 (245:238). This suggests that men are more vulnerable to developing this complication (3,10,15-18). A patient population between 30-40 years is more prone to develop PTH as reflected in the average age at presentation and being detected following head injury. In this study, the specific age and sex were not significantly associated, although a predominance of higher age and male sex is seen.

PTH commonly occurs in the first year following head injury and has been noted to occur as early as within 7 hours of the injury (6). In our series, 1 patient was diagnosed 21 days after the trauma and 1 case as late as 12 months. The average duration was 16.43 ± 23.7 (SD) weeks. In this study, we noticed that cases in Group 1 who had overt features of PTH presented earlier compared to other groups, and this association was significant. Post-traumatic ventriculomegaly is a slow passive process due to cerebral tissue necrosis and commonly requires more than 3–6 months to develop. The detection of such cases can be clinically challenging. A predisposing etiology in cases with no improvement, or neurological deterioration combined with CT findings and CSF dynamic studies may help to detect it. In contrast to ventriculomegaly due to brain atrophy, PTH is an active process and usually presents within 3 months of head injury with a progressive worsening of neurological status (1,9,10,15).

In the present study, 2.1% of cases had established PTH with raised ICP. Most of the time, PTH is associated with a severe head injury, but in a few cases, though uncommon, it may also occur following moderate head injury (9). In our study, a GCS below 8 was significantly associated with the development of PTH, but there were cases with moderate head injury also although they were not significantly associated with its development. PTH may present with various clinical features, including obtundation, a failure to improve, psychomotor retardation, memory loss, ataxia, and incontinence. Prolonged coma or arrest in clinical progress in conscious patients should raise the suspicion of hydrocephalus (9,10,18,19). Sometimes, the patient may be too injured to demonstrate clinical signs and symptoms of PTH or may present with atypical symptoms (7,19). According to a few studies (7,9,10), when a patient is in a state of prolonged coma or when there is an arrest in the clinical progress of conscious cranio-cerebrally injured patients, communicating hydrocephalus should be suspected (Figure 2). In such patients, funduscopy may reveal papilledema, which was detected in all 10 Group 1 cases.

SAH has been cited as the most important pathology leading to the development of PTH in few studies (2,7,8,20,21). Obliteration of the subarachnoid spaces with fibrous thickening of the lepto-meninges, particularly in the sulci of the convexity and base of the brain, has been suggested as the main etiological factor. In the present study and others, cerebral contusion has been found to be the most common CT finding, followed by SAH, SDH, and IVH.

A comparative model of the CSF system is difficult to define for the measurement of raised ICP derived from manometric data using low volume, short duration infusions into the subarachnoid space to describe the mechanisms governing intracranial pressure dynamics. The mathematical model from a different study indicates that steady-state elevations of ICP are regulated primarily by a nonlinear, pressure-dependent outflow resistance to CSF reabsorption. Model computer simulation experimental studies suggest that parameter changes within the resistance relationship can have a profound effect on ICP. In contrast, intracranial compliance acts only to buffer transient changes in volume prior to the establishment of steady-state CSF pressure. Raised opening pressure and mean pressures reflect the measurement of the high-pressure compartmental system and may help in deciding on the implications of a CSF diversion procedure as in this study. We divided cases with ventriculomegaly and periventricular lucency into two groups, one with normal and the other with slightly raised ICP. We found significantly improved results in cases with raised ICP as measured by the opening pressure on lumbar tapping of the spinal CSF, which has also been suggested in a few other studies (6,12-15). Decompressive craniotomy (DC) is associated with the development of PTH by altering CSF pressure dynamics, mechanical blockage around convexities, or inflammation of arachnoid granulations by post-surgical debris (4,22). It leads to flattening of the normal dicrotic ICP waveforms due to the transmission of the pressure pulse through the open cranium. Since arachnoid granulations function as pressure-dependent one-way valves from the subarachnoid space to the venous sinuses, disruption of the pulsatile ICP flow dynamics occurs. Hence, early cranioplasty should lead to the restoration of normal ICP dynamics and the spontaneous resolution of hydrocephalus. A higher incidence of PTH has been found with extended decompressive craniotomy and re-operations because of the increased chances of blockage of the CSF pathway through basal cisterns and subarachnoid granulation tissue. In our cases who underwent craniotomy as primary treatment and subsequently developed PTH, shunt revision was more frequent, but it was due to either shunt malfunction or shunt infection.

Various studies (1,3,9-11,18) have been conducted to determine the association of the admission GCS score, age, sex, decompressive craniotomy, IVH, and features of traumatic SAH (on CT scan) with the development of hydrocephalus. However, other factors such as duration of MV support, ICU stay in the hospital, total LOS in the hospital, presence of other systemic injury, coagulopathy, and presence of septicemia may be related. Although these factors are not primarily responsible for its development, they may cause a secondary insult to the brain. In this study, a significant relation between prolonged ventilator support, and prolonged stay in the ICU and hospital were significantly associated with the development of PTH. Hypoxic episodes combined with compartmental pressure changes during mechanical ventilation may trigger a cascade of pathological changes in the compliance of the airway and the intracranial space leading to decreased compliance and contributing to the development of PTH. Prolonged hospital stay is related to prolonged MV requirement, frequent compartmental changes, and decreased compliance of the intracranial space, which may contribute to the development of PTH as observed in this study and others (6,8). This is one of the few studies that have considered individual systemic injury (with AIS score ≥3). The definitive surgical treatment in these patients may have affected the duration of mechanical ventilation, the LOS in hospital, and may have aggravated factors that could have adversely affected the treatment of the brain injury. We included the rating of systemic injuries as they were often severe enough to adversely affect brain injury management and to reduce the chances of inter-observer variation. As these individual injuries were fewer in number in this study, their statistical association as risk factors could not be identified, but these factors are observed to adversely affect head injury management in such cases as there are more chances of associated shock, septicemia, chest infections, hypoxic phenomena, and prolonged ICU and hospital stay (11).

CONCLUSION

CSF diversion procedures help to improve ventriculomegaly in cases with documented evidence of raised ICP but not in cases without. ICP should be monitored before planning for CSF diversion procedures to avoid an unnecessary invasive procedure and its possible complications. A subgroup of PTH which cannot be treated by CSF diversion procedures can be managed only by minimizing risk factors responsible for

its development. Attention should be paid to factors such as decompressive craniotomy, severe head injury with a low GCS and prolonged ICU stay. The judicious use of decompressive craniotomy and minimizing the duration of mechanical ventilation together with necessary measures to improve GCS should be a priority.

AUTHORSHIP CONTRIBUTION

Study conception and design: VCJHA

Data collection: VCJHA, NJHA

Analysis and interpretation of results: VCJHA, NJHA

Draft manuscript preparation: VCJHA, NJHA

All authors (VCJHA, NJHA) reviewed the results and approved the final version of the manuscript.

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