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Research Article

Post Traumatic Hydrocephalus in Severe Head Injury - Risk Factors

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Abstract

Posttraumatic hydrocephalus (PTH) is a frequent and serious complication that follows a traumatic brain injury (TBI). Its incidence varies greatly from study to study, largely based on different criteria for its diagnosis. The purpose of this study is to identify the risk factors like age, admission Glasgow Coma Scale (GCS), decompressive craniectomy (DC) and findings in initial Computed tomography (CT) scan like Subarachnoid hemorrhage (SAH), Intraventricular hemorrhage (IVH) and skull base fracture which may predispose for the development of hydrocephalus in traumatic severe head injury patients.

This is a single center prospective observational study in which patients with age > = 14 and GCS < = 8 are followed with regular CT scan for a period of 4 months during January 2013 to January 2016 admitted in our hospital. A total of 32 post traumatic hydrocephalus cases have been identified among 489 cases included in the study resulting in the incidence of 6.54%. Mean duration of presentation was 48.76 +/-33.26 days. 82% of patients in hydrocephalus group had SAH while 52% in non-hydrocephalus group had SAH in initial scan (P = 0.001). Decompressive craniectomy was done in 69% patients with hydrocephalus while only 32% of non-hydrocephalus group underwent DC (P = 0.00001). Other parameters namely age, sex, GCS, IVH and skull base fractures were not significantly associated with development of hydrocephalus.

To conclude decompressive craniectomy and SAH significantly increases the probability of development of post traumatic hydrocephalus while other factors like IVH, base of skull fracture, age and admission GCS do not increase the development of hydrocephalus.

Keywords: Hydrocephalus; Glasgow Coma Scale; Decompressive Craniectomy; Computed Tomography; Subarachnoid Hemorrhage; Intraventricular Hemorrhage; Skull Base Fractures.

Introduction

Posttraumatic hydrocephalus (PTH) is a frequent and serious complication that follows a traumatic brain injury (TBI). Its incidence varies greatly from study to study, largely based on different criteria for its diagnosis. It could greatly impact morbidity following a TBI and could result in increased mortality if it is not recognized and treated.

The pathogenesis of hydrocephalus after traumatic brain injury is either due to obstruction to CSF flow in the ventricle leading to obstructive hydrocephalus or due to blockage of arachnoid granulation resulting in communicating hydrocephalus by the blood. Risk factors for PTH are yet to be fully identified, as early diagnosis and treatment will reduce the morbidity and mortality.

Decompressive craniectomy done to treat raised intracranial pressure after head injury has been associated with a significant incidence of hydrocephalus. As there was conflicting evidence about the role of subarachnoid hemorrhage, intraventricular hemorrhage, skull base fracture, severity of injury, age and duration of coma in the development of PTH, we performed prospective study to identify whether these factors will predispose for the development of hydrocephalus.

We analyzed all severe head injury patients with regular CT scans to identify the incidence and risk factors for post traumatic hydrocephalus.

Aims and Objectives

- To assess the incidence of post traumatic hydrocephalus in severe head injury.
- Role of age, admission GCS, craniotomy and decompressive craniectomy in the development of hydrocephalus.
- Role of subarachnoid hemorrhage, intra ventricular hemorrhage and skull base fracture in the development of hydrocephalus.

Materials and Methods

We did a prospective observational study of 709 severe head injury patients who presented to our hospital during January 2012 to July 2015.

Four hundred eighty-nine patients were included in the study. The severity of injury was defined as duration of coma of at least 6 hours and a Glasgow Coma Scale (GCS) total score of 8 or less in the acute phase. Post Traumatic Hydrocephalus was defined as Evans ratio > 0.3, accompanying transependymal edema, the presence of either clinical worsening or failure to make neurological improvement over time.

Post Traumatic Hydrocephalus was defined as the presence on any of the control CT scans of both of the following criteria: 1) Evans ratio > 0.3 (the greatest width of the frontal horns divided by the largest biparietal distance between the inner tables of the skull) and 2) Gudeman CT criteriaas. Gudeman CT criteria include enlarged anterior horns of lateral ventricles and enlarged temporal horns and third ventricle in the presence of normal or absent sulci with periventricular translucency.

All severe head injury patients were examined by neurosurgeon in emergency department. Patients with age > = 14, GCS < = 8 are included in the study. Patients were excluded from the analysis if they were known to have had neurologic deficits before the trauma; those who were expired, lost follow up and CSF analysis suggestive of infection.

All the patients were regularly examined from the time of admission for added neurological deficits and CT brain was done whenever required. Also CT brain was taken for all the patients at the time of admission, 14, 30 and 60 days irrespective of clinical condition even when patient shows good clinical improvement.

The factors like age, sex, admission GCS, initial CT findings like SAH, IVH, Skull base fracture and whether craniotomy or decompressive craniectomy are compared between hydrocephalus and non-hydrocephalus groups.

An informed consent is obtained from all the patient attendees. The information collected regarding all the cases were recorded in a master chart. The Statistical analysis was performed on a computer by STATA 11.1 (College station, TX USA). The continuous variables were expressed as Mean and Standard deviation. Categorical variables were expressed as frequency and percentage. Independent t-test was used to find the significance between different groups. Chi- square test and fisher's exact test was used to find out association between the categorical variables. A probability value of less than 0.05 was considered statistically significant ($P < 0.05$).

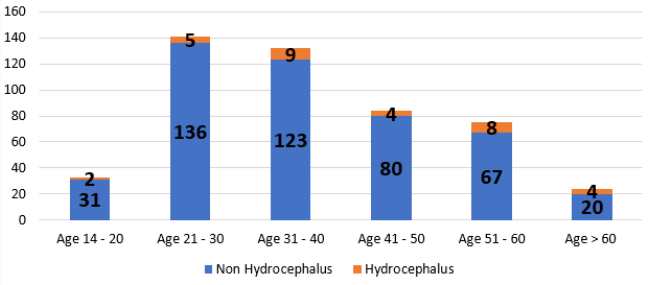
Results

Out of 709 patients who were admitted and managed for severe head injury in our institute, 489 patients met the inclusion criteria and were included for the analysis. Parameters included in the analysis were age, sex, Glasgow coma scale at admission, subarachnoid hemorrhage, intraventricular hemorrhage, skull base fractures and Decompressive craniectomy. The results were as follows.

Age

The age group ranges from 20 years to 65 years in hydrocephalus group and 15 years to 66 years in non-hydrocephalus group. The mean age and standard deviation in hydrocephalus group is 41.21 ± 13.37 and in non-hydrocephalus group is 36.74 ± 12.51. The unpaired t- test did not differ significantly ($P = 0.27$).

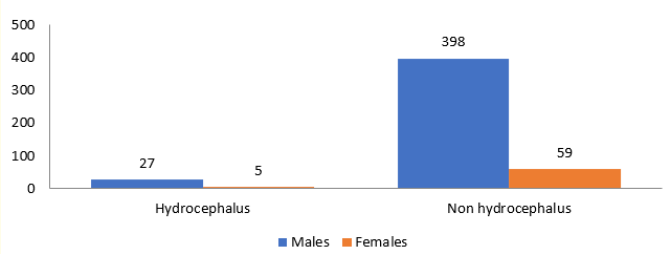
Out of 32 patients who developed hydrocephalus, 17 patients are from younger age, 4 from middle aged and 10 from older age group. There is no influence of age on development of hydrocephalus as p-value is found to be 0.26 which is not statistically significant (Graph 1).



Graph 1: Age distribution in Hydrocephalus and Non-Hydrocephalus groups (Age on X axis, n value on Y axis).

Sex

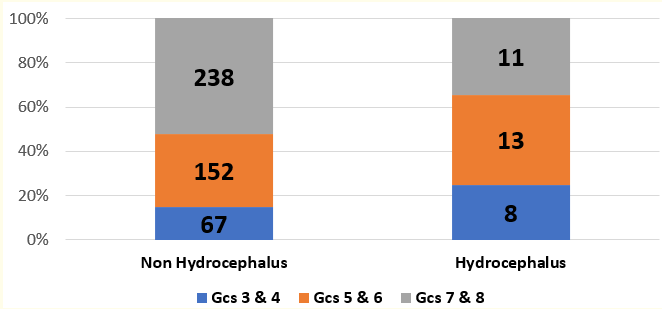
Out of 489 patients, 425 (86.9%) were males and 64 (13.1%) were females. Out of 32 patients who developed post traumatic hydrocephalus, 27 were males and 5 were females with P- value 0.65 which is not statistically significant. Hence there is no influence of Sex on development of post traumatic hydrocephalus (Graph 2).



Graph 2: Sex distribution in hydrocephalus and Non hydrocephalus groups.

Glasgow coma scale at admission

Among the severe head injury patients, GCS score of 7, 8 constitutes 55%, 5, 6 constitutes 32% and 3, 4 constitutes 14%. Most of the patients with GCS 3, 4 were excluded from the study due to death and lost follow-up. Out of 32 patients who developed hydrocephalus, GCS 7,8 constitutes 44% and 5,6 constitutes 38%. GCS does not seem to influence the development of post traumatic hydrocephalus as p value is 0.44 (Graph 3).



Graph 3: Relation between admission GCS and Post traumatic hydrocephalus.

Subarachnoid hemorrhage

261 patients out of 489 had subarachnoid hemorrhage in our study with hemispheric region is the most common location. 26 patients out of 32 had subarachnoid hemorrhage in various locations like cerebral hemispheres, sylvian fissure, and basal cisterns. Subarachnoid hemorrhage greatly influences the development of hydrocephalus in head injury patients as Fisher exact test showed significant difference (P = 0.0008). (Graph 4).

Graph 4: Relation between Subarachnoid hemorrhage and Post traumatic hydrocephalus.

Intraventricular hemorrhage

Only 152 patients had intraventricular hemorrhage in our study constituting less than 1/3 of total severe head injury. Only 14 patients in hydrocephalus and 138 patients in non-hydrocephalus group had intra ventricular hemorrhage in our study. As P- value was 0.116 in our study, intra ventricular hemorrhage does not seem increase the risk of hydrocephalus in head injury (Graph 5).

Graph 5: Relation between Intraventricular hemorrhage and Post traumatic hydrocephalus.

Skull base fractures

194 patients had base of skull fracture in our study in various locations extending from anterior to posterior cranial fossa.50% of patients in hydrocephalus group had skull base fracture while it constitutes only 39% in non-hydrocephalus group. Most common location was middle cranial fossa in our study. Fisher exact test showed no significant difference between two groups (p = 0.262) (Graph 6).

Graph 6: Graph depicting relation between skull base fractures and post traumatic hydrocephalus.
Skull base fractures. No skull base fractures.

Decompressive craniectomy

Total of 229 surgeries were performed which include both craniotomy and decompressive craniectomy out of 489 severe head injury patients constituting 47%. The most common indication was unilateral hemispheric subdural hemorrhage. 23 patients in hydrocephalus group and 206 patients in non-hydrocephalus group underwent surgery which includes both craniotomy and decompressive craniectomy. The difference was significant by Fisher exact test (p = 0.005) which indicates that surgery itself increases the risk of post traumatic hydrocephalus (Graph 7).

Graph 7: Graph depicting Relation between surgery and development of hydrocephalus.

Out of 489 patients, 61 patients underwent decompressive craniectomy. Unilateral frontotemperoparietal decompression was done for all the patients. 22 patients in hydrocephalus group and 39 patients in non-hydrocephalus group underwent decompression in our study. The p-value was 0.0001 as calculated by Fisher exact test which is highly significant. Decompressive craniectomy definitely increases the risk of developing hydrocephalus (Graph 8).

Graph 8: Relation between decompressive craniectomy and hydrocephalus.
No decompressive craniectomy. Decompressive craniectomy

Discussion

In this prospective study which occurred over the course of 4 years in our hospital, we observed 489 cases of severe head injury. Daily neurological examination was performed on all the patients and CT brain was done whenever there were new neurological deficits or patient fails to improve over time. On following for 4 months, 32 patients developed hydrocephalus. The initial CT findings and other factors like age, admission GCS, surgery and decompression are compared between hydrocephalus and non-hydrocephalus groups.

Incidence of PTH in world literature is quite variable, ranging from 0.7 to 29% [1-4]. Kishore., *et al.* found that only 13.7% of patients with ventriculomegaly had PTH [3]. In the present study, 32 cases out of 489 cases of severe head injury admitted at our center developed PTH. Thus, the incidence was 6.54% in severe head injuries.

PTH commonly occurs in first year post trauma and has been described as early as within 7 hours of injury [1,5-7]. In our series 1 patient was diagnosed on 14th post-trauma day and 1 as late as 190th day. The mean duration of presentation was 56.46 days with standard deviation of 41.59 (mean ± SD) (56.46 ± 41.59).

The age in hydrocephalus group ranged from 20-65 years with mean age and standard deviation of 41.21 ± 13.37. The maximum number of patients belongs to younger age group (20-40 years) constituting 53% of patients with hydrocephalus. The age does not seem to increase the risk of PTH in our study (p = 0.27). The sex distribution was not uniform with maximum number of patients belongs to males constituting 87% of cases in hydrocephalus group.

The retrospective study conducted by Byung-Rae Cho, MD also showed similar results that age and sex had no relation with post traumatic hydrocephalus [8].

The maximum number of patients falls into GCS score of 7, 8 constituting 55% of all cases. Out of 32 patients who developed hydrocephalus, 44% were from GCS scores of 7, 8, 38% were from 5, 6 and 3, 4 constitutes only 18%. The GCS among severe head injury patients was not considered as a risk factor for post traumatic hydrocephalus in our study as Fisher exact test does not show any significant difference between two groups. The same result was seen in retrospective study done by Byung-Rae Cho, MD [8].

Subarachnoid haemorrhage (SAH) has been cited as the most important pathology leading to development of PTH [1,9-12]. Obliteration of subarachnoid spaces with fibrous thickening of lepto-meninges particularly in sulci of the convexity and base of brain as a result of SAH has been suggested [13]. 54% of patients had subarachnoid haemorrhage in our study on admission CT brain. In hydrocephalus group, 82% of patients had subarachnoid haemorrhage and in non-hydrocephalus group, only 52% had subarachnoid haemorrhage. SAH definitely increases the risk of developing hydrocephalus in our study (p = 0.001). The study done by Anthony Marmarou showed SAH influences the post traumatic hydrocephalus [14].

Intra ventricular haemorrhage usually results in acute obstructive hydrocephalus, but some studies showed that it increases the risk of hydrocephalus after acute phase causing communicating hydrocephalus. Retrospective cohort study done by Mohd Aidil Mohd Nor showed intraventricular hemorrhage is associated with increased risk of hydrocephalus [15].

31% of patients with severe head injury had intraventricular haemorrhage in our study. IVH constitutes 44% in hydrocephalus group and 31% in non-hydrocephalus group. When fisher exact test was applied, there was no statistical significance between IVH and post traumatic hydrocephalus (0.116). The same result was seen in retrospective study done by Byung-Rae Cho, MD. Study performed by Areal Kaen., *et al*. also showed no association between IVH and hydrocephalus [8,16].

Retrospective cohort study done by Mohd Aidil Mohd Nor showed base of skull fracture increases the risk of post traumatic hydrocephalus probably by altering CSF circulation at the base of brain [15]. In our study skull base fracture was seen in 50% of patients in hydrocephalus group and 39% of patients in non-hydrocephalus group. There was no association between skull base fracture and hydrocephalus in our study (p = 0.262). Multivariate analysis done by Areal Kaen., *et al.* showed no association between skull base fracture and hydrocephalus [16].

Surgery was performed in 47% of severe head injury patients in our study. 72% of patients in hydrocephalus group and 45% in non-hydrocephalus group underwent surgery. We found that surgery increases the chances of developing hydrocephalus (P =

0.005) in our study. Though there were many studies stating that decompressive craniectomy increases the risk of hydrocephalus but no studies are there to show any surgery will increase the risk of hydrocephalus.

Decompressive craniectomy (DC) has been found to be associated with development of PTH by altering CSF pressure dynamics, mechanical blockage around convexities or inflammation of arachnoid granulations by post-surgical debris. 9% of patients with severe head injury underwent decompressive craniectomy in our study. In hydrocephalus group decompression was done in 69% and in non-hydrocephalus group 32% underwent decompression. Our study also showed similar results that DC definitely rises the risk of hydrocephalus in severe head injury (P = 00001). Retrospective study done by Phuenpathom N also showed similar result [16]. Study done by Ariel Kaen and his colleagues found that the decompressive craniectomy significantly increases the risk of PTH and IHH predicts the development of hydrocephalus [16]. Study done by Czosnyka., *et al.* and Choi., *et al.* on the influence of craniectomy on the CSF circulation identified large craniectomy with < 25mm width at the sagittal sinus increases the risk of PTH [17,18].

Conclusion

In our study, we found that the incidence of post traumatic hydrocephalus was 6.54% in severe head injury. Risk factors like age, sex and admission GCS does not seems to increase the risk of post traumatic hydrocephalus. Subarachnoid hemorrhage is the only initial CT finding which increases the risk of post traumatic hydrocephalus whereas other findings like intra ventricular hemorrhage and skull base fracture does not seem to be the significant risk factors for post traumatic hydrocephalus. Sub arachnoid haemorrhage and decompressive craniectomy are two main factors which significantly increases the risk of post traumatic hydrocephalus.

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