Post Traumatic Hydrocephalus: A Review of 68 Cases

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ABSTRACT

Objective: To evaluate different factors affecting post traumatic hydrocephalus.

Material and Methods: This retrospective study was conducted in the department of Neurosurgery, Postgraduate Medical Institute, Lady Reading Hospital Peshawar from July 2009 to June 2011. Record of all head injury patients was reviewed. Out of 5438 admitted patients with head injury, those who developed post traumatic hydrocephalus were included in the study.

Results: A total of 68 patients with post traumatic hydrocephalus were diagnosed over the last two years. Out of these 47 (69%) were male and 21 (31%) female. Mostly they were of young age group between 08 years to 41 years. Mean age of presentation being 20 years. A large group 56 (82%) out of 68 patients were in state of moderate to severe head injury, diagnosed within matter of hours or days, as acute post traumatic hydrocephalus. Those who developed after two weeks, fall in group chronic post traumatic hydrocephalus. Out of these, 55 (81%) patients underwent some sort of CSF diversion procedure like external ventricular drain (EVD) or Ventriculo Peritoneal (VP) Shunt. Subarachnoid hemorrhage, intraventricular hemorrhage, intracranial hematoma, posterior fossa bleed, meningitis and craniotomy were recognized associative factors for development of PTH. There was improvement in about 59% after surgery. And the long term prognosis, expressed in GOS, was good in 44% of the cases.

Conclusion: Post traumatic hydrocephalus is a dangerous complication and needs critical consideration and early diagnosis in severe head injury cases.

Key Words: Head injury, Post traumatic hydrocephalus, External ventricular drain, VP shunt.

INTRODUCTION

Post traumatic hydrocephalus is a well known manageable complication after head injury. Its incidence varies greatly in different studies. It is more common in cases with severe head injury with GCS 3 to 8 on presentation, as compared to mild and moderate head injury. The incidence of PTH is significantly lower in cases of mild head injuries and ranges from 0.75% to 10.7%.^{1,23} This larger variation in incidence may be due to associated risk factors like subarachnoid hemorrhage, intraventricular hemorrhage and craniotomy procedure for such cases. However the incidence in patients with hydrocephalus, a previous head injury has been implicated as a cause in 2 to 50%.^{4,6,10} On the other hand head injury patients have the incidence of hydrocephalus from 1% to 90%.^{5,7,8,9, 12,13,15,16}

The dilatation of ventricles after head injury is common due to post traumatic cerebral atrophy (hydrocephalus en vacuo) which is well differentiated from PTH.²⁷ Post traumatic hydrocephalus is an active and progressive process of CSF accumulation following cranial injury.¹⁴ Ventricular enlargement is a consequence of space left by degeneration of white matter, which was clear around lateral ventricles in most of chronic hydrocephalus with more than two weeks after head injury. However it is usually associated with other abnormalities in acute cases diagnosed on CT scan findings such as acute subdural hematoma, intraventricular hemorrhage, subarachnoid hemorrhage, supratentorial clot with unilateral ventricular dilatation, posterior fossa space occupying lesion, intracerebral contusion and pneumocephalus. Delayed intraventricular hematoma and hydrocephalus have known association following evacuation of post traumatic acute subdural hematoma.^{2,17,18,19} Hypoxic – ischemic insult is main contributory factor in chronic cases with post traumatic hydrocephalus,^{11,26} these may have minimal or no changes in earlier post traumatic CT scans. They may have few or no symptoms or rarely present with symptoms that necessitate urgent intervention. On the other hand acute cases are observed in shorter post traumatic periods by the help of serial CT scans on clinical grounds.

MATERIAL AND METHODS

Patients

All admitted 5438 patients with head injury at Neurosurgical Unit, Neurosurgical ICU, or Neurotrauma Unit PGMI/Govt. of Lady Reading Hospital Peshawar from July 2009 to June 2011 were retrospectively studied. Data analyzed regarding the incidence, age, sex, mode of injury, clinical findings, CT Scan finding, onset of Post traumatic hydrocephalus, neurosurgical intervention and outcome. Records, OT registers, previous admission charts and available CT scans were scrutinized to take help for relevant information. We encountered 68 patients of PTH, representing 1.25% of the total 5438 head injury cases.

Radiology

Initial CT scan of all those suspected PTH patients admitted to neurosurgical unit in emergency was compared to the serial CT scans taken on clinical suspicion. Those with positive clinical suspicion and increasingly dilating ventricular system on CT scan were included. And an Evans' index ≥ 0.30 was taken into consideration if previous scan was comparatively in normal limits. Those with dilated temporal horns (\geq 2 mm) of lateral ventricles, dilated 3^{rd} ventricle (≥ 5 mm), periventricular hypodensity (translucency) and diminution of fissures, sulci and cisterns all favored points in PTH were also taken into consideration for diagnosis. All cases with dilated ventricular system with 60 years age or more, severe comorbidities or previous VP shunted patients were excluded. Other associated CT findings were also noted. And unilateral ventriculomegally noted in some of large hematomas.

The patients with low GCS, external ventricular drains were put in, and those patients with GCS 10 and

above, ventriculoperitoneal shunt were inserted.

Data analysis was performed through SPSS version 10.0. Frequencies and percentages were computed to present categorical variables like age, sex, CT findings, per op findings, and improvement.

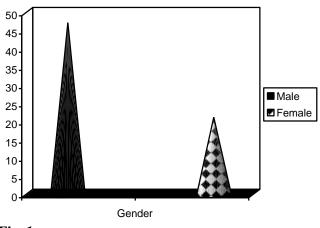
RESULTS

A total of 68 patients were diagnosed as PTH of total 5438 admission head injury cases. Out of these 47 (69%) were male and 21 (31%) female. Mostly they were of young age group between 08 years to 41 years. Mean age of presentation was 20 years. A large group of (82%) patients presented in state of severe head injury, and were diagnosed within matter of days, as acute post traumatic hydrocephalus. Remaining (18%) patients, who developed PTH after two weeks, fall in group chronic post traumatic hydrocephalus. Acute PTH cases are associated with of intracranial pathologies like intraventricular, intracerebral hematoma or subarachnoid hemorrhage.

Out of these, 55 (81%) patients underwent some sort of CSF diversion procedure like external ventricular drain (EVD) or Ventriculo Peritoneal (VP) Shunt. Subarachnoid hemorrhage, intraventricular hemorrhage, intracranial hematoma, posterior fossa lesion, meningitis and craniotomy were recognized associative factors for development of PTH. There was improvement in about 59% after surgery. And the long term prognosis, expressed in GOS, was good in 44% of the cases. There were 4 mortalities recorded in first group.

DISCUSSION

Ventricular enlargement is a common sequel of severe





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Total Admissions (GCS)	No. of Cases	Incidence of PTH
Mild head injury (13 – 15)	2565	02 (3% of all PTH)
Moderate head injury (9 – 12)	1636	10 (15%)
Severe head injury (3 – 8)	1237	56 (82%)

Table 1: Total head injury admissions between July2009 and June 2011.

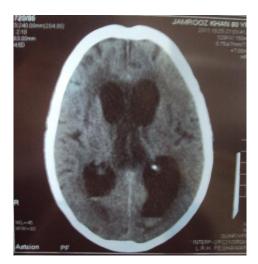
Table 2: CT scan findings.

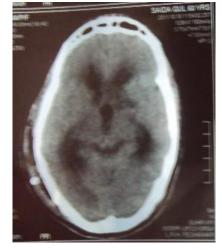
CT scan findings	Incidence
Intraventricular hemorrhage	33 (48.5%)
Subarachnoid hemorrhage	12 (17.6%)
Cerebral contusions	06 (8.8%)
Intracranial hematomas (EDH, SDH)	12 (17.6%)
Depressed skull fracture	02 (02.9%)
Firearm Injury (Metallic Foreign Bodies)	03 (04.4%)

closed head injury.^{21,22,24} The accurate incidence of post traumatic hydrocephalus is not known. In some large head injury series, the incidence of post traumatic hydrocephalus was reported to range from 1.5% to 4%.^{9,24} Other series shows the incidence being from 0.7 - 86%.¹⁷⁻¹⁹ But the incidence of symptomatic hydrocephalus has been reported to be from 0.7% to 29%.^{17,18} Hence, it is important to distinguish between PTH and cerebral atrophy as the latter usually do not respond to neurosurgical procedures.^{28,30} Dandy reported the PTH for the first time in 1914.²

PTH may present with various clinical syndromes including obtundation, failure to improve and a tetrad of symptoms including psychomotor retardation, memory loss, gait ataxia and incontinence.^{29,31} Sometimes, the patient may be too injured to demonstrate clinical signs and symptoms of PTH, or may present with atypical symptoms ²⁹. According to Groswasser, et al when a patient is in a state of prolon**Table 3:** Outcome following neurosurgical intervention.

Neurosurgical intervention	GCS Level	No. of cases	Outcome
Ventriculo- peritoneal shunt	13 - 15 = 00 09 - 12 = 06 03 - 08 = 09	15	08 – good improvement 05 – moderate improvement 02 – no improvement
External ventricular drain	13 - 15 = 00 09 - 12 = 06 03 - 08 = 09	34	18 – good improvement 06 – no improvement 10 – died / No improvement
External ventricular drain followed by VP shunt	13 - 15 = 00 09 - 12 = 06 03 - 08 = 09	06	04- good improvement 01 – no improvement 01 – died / No improvement





PTH with remnant of Intraventricular bleed

A Symptomatic case of PTH.

ged coma or when there is an arrest in the clinical progress in conscious cranio-cerebral injured patients, communicating hydrocephalus should be suspected.

In the severe head injury, PTH takes only few

weeks or less to develop, whereas ventriculomegally related to cortical atrophy evolves slowly over a period of 6 months or more post trauma. However, cortical atrophy did not strictly correlate with PTH, particularly in the most severe cases, suggesting a different mechanism of development. Cortical atrophy seemed to be strongly related to documented anoxia and Diffuse Axonal Injury, whereas PTH develops probably due to CSF blockage around the convexities such as suggested by the high correlation with decompressive craniectomy.^{3,19,20} Forglou described obliteration of subarachnoid spaces with fibrous thickening of the leptomeninges particularly in sulci and base of the brain.^{3,30} This finding is in agreement with Marmarou et al. Moreover Wood et al. found that patients who have had clinical symptoms of hydrocephalus for less than 6 months have a better prognosis.

Our study indicates that younger age, shorter duration of coma, severe head injury (GCS 3 - 8), and decompressive craniectomy in acute phase could be the risk factors for the development of PTH. Moreover intraventricular hematoma and subarachnoid hemorrhage highly favours the development of PTH in acute phase just in a matter of hours or days. A correlation between PTH and Duration of coma was also reported by Meyers et al.²⁶

Patients who benefited from surgery were those, who manifested a deterioration of clinical status. This data seems to suggest that selection of the patients for surgery can be defined principally on clinical basis. Deterioration involved many functions, but consciousness and behavior were the most indicative. Lewin has reported on of the largest series of patients with ventricular dilation following severe head injury. In his series 59 patients with generalized ventricular dilation had an evidence of obstruction in the basal cistern.³³

SPECT may be helpful in differentiating ventricular enlargement due to cortical atrophy and hydrocephalus. PTH influences the functional and behavioral outcome of severe TBI and represents an important prognostic factor for posttraumatic epilepsy.²³

CONCLUSION

According to our study results Posttraumatic hydrocephalus is particularly rare in mild and moderate head injury cases as compared to the higher incidence in cases with severe head injury. Traumatic intraventricular hemorrhage and / or SAH were the commonest risk factors as seen on initial CT scans.

PTH commonly developed in the first two to three weeks when the patents were convalescing on the wards. Clinical and radiological monitoring is of great concern especially in patients with a high risk factor for earlier diagnosis and according management plan. Surgical outcome is significant once diagnosis is established in time, 44% cases in our study with good outcome.

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REFERENCES

- 1. Marmarou A, Foda MA, Bandoh K, et al. Posttraumatic ventriculomegally: Hydrocephalus or atrophy? A new approach for diagnosis using CSF dynamics. J Neurosurg 1996; 85: 1026-35.
- Dandy W, Blackfan KD: Iternal hydrocephalus. An experimental, clinical and pathological study. Am J Dis Child 8, 1914: 405-462.
- II-Choi, Hyung-Ki Park, Jae-Chil Chang, Sung-Jin Cho, Soon-Kwan Choi, Bark-Jang Byun. Clinical factors for development of posttraumatic hydrocephalus after decompressive craniectomy. J Korean Neurosurg Soc 2008; 43: 227-231.
- Laws ER, Mokri B. Occult hydrocephalus: results of shunting correlated with diagnostin tests. Clin Neurosurg 1977; 24: 316-33.
- Zander E. Die posttraumatic hydrozephalie and IHRE. Behandlungsmoglicheiten. Schweiz Med Wochenschr 1969; 99: 1624-9.
- 6. Salmon JH. Adult hydrocephalus. Evaluation of shunt therapy in 80 patients. J neurosurg 1972; 37: 423-8.
- Pedersen KK, Haase J. Isotope liquography in demonstration of communicating obstructive hydrocephalus after severe cranial trauma. Acra Neurol Scand 1973; 49: 10-30.
- Lewin W. Preliminary observations on external hydrocephalus after severe head injury. Br J Surg 1968; 55: 747-51.
- 9. Koo AH, LaRoque RL. Evaluation of head trauma by computed tomography. Radiology 1977; 123: 345-50.
- Symon L, Hinzpeter T. The enigma of normal pressure hydrocephalus: tests to select patients for surgery and predict shunt function. Clin Neurosurg 1977; 24: 285-315.
- 11. Adams JH, Mitchell DE, Grahm DI, Doyle D, Diffuse brain damage of immediate impact type. Brain 1977; 100: 489-502.
- 12. Foroglou G. L'Hydrocephalie posttraumatique. Neurochirurgie 1976; 22: 108-11.
- 13. French BN, Dublin AB. The value of computer tomography in the management of 1000 consecutive head

injuries. Surg Neurol 1977; 7: 171-83.

- Erico R. Cardoso, Sam Galbraith. Posttraumatic hydrocephalus – A retrospective review. Surg Neurol 1985; 23: 261-4.
- Granholm L, Svengaard N. Hydrocephalus following traumatic head injuries. Scand J Rehab Med 1972; 4: 31-4.
- Gudman SK, Kishore PRS, Becker DP, Lipper MH, Girevendulis AK, et al. Computer tomography in the evaluation of incidence and significance of post-traumatic hydrocephalus. Radiology 1981; 141: 397-402.
- Cardoso ER, Galbraith S: Posttraumatic hydrocephalus — a retrospective review. Surg Neurol 1985; 23: 261-264.
- 18. Hawkins TD, Lioyd AD, Fletcher GI, Hanka R: Ventricular size following head injury : a clinicoradiological study. Clin Radiol 1981; 27: 279-289.
- Erban P, Woertgen C, Luerding R, Bogdahn U, Schlachetzki F, Horn M. Long – term outcome after hemicraniectomy for space occupying right hemispheric MCA infarction. Clin Neurol Neurosurg 2006; 108: 384-387.
- 20. Woertgen C, Rothoerl RD, Schebesch KM, Albert R. Comparison of craniotomy and craniectomy in patients with acute subdural hematoma. J Clin Neurosci 2006; 13: 718-21.
- Strich SJ. The pathology of brain damage due to blunt head injuries, In: Walker AE, Caveness WF, Critchley M. eds. The late effects of Head injury, Springfield: Charles C. Thomas 1969: 501-26.
- 22. Levin HS, Meyers CA, Grossman RG, Sarwar M. Ventricular enlargement following closed head injury. Arch Neurol 1981; 38: 623-9.
- 23. Letizia M, Riccardo C, Elisabetta A, Felice R, Llaria P, Giuseppe O. Posttraumatic hydrocephalus: A clinical,

neuroradiologic, and neuropsychologic assessment of long – term outcome. Arch Phys Med Rehabil 2003; 84: 1637-41.

- 24. Kishore PR, Lipper MH, Miller JD, et al. Posttraumatic hydrocephalus in patients with severe head injury. Neuroradiology 1978; 16: 261-5.
- 25. Foroglou G, Zander E: (Post-traumatic hydrocephalus and measurement of cerebrospinal fluid pressure). Acta Radiol Diagn (Stockh) 1972; 13: 524-530.
- 26. Meyers CA, Levin HS, Eisenberg HM, Guinto FC, Eary versus late ventricular enlargement following closed head injury, J Neurol Neurosurg Psychiatry 1983; 46: 1092-7.
- 27. Desiderio R, Rewati R, Jesus S, Sanjay J, Ashok K, Santosh D. Post-traumatic hydrocephalus in severe head injury series of 22 cases.
- Cardoso ER, Galbraith S: Post traumatic hydrocephalus, a retrospective review. Surg Neurol 1985; 23: 261-264.
- 29. Beyerl B, Black PM: Post traumatic hydrocephalus. Neurosurg 1984; 15: 257-26.
- 30. Guyot LL, Micheal DD:Post traumatic hydrocephalus, Neurol Res 2000; 22: 25-28.
- Spanu G, Knerich R, Messina AL, Karussos G: Post traumatic hydrocephalus. Riv Neurol 1985; 55: 185-196.
- 32. Groswasser Z, Cohen M, Reider-Groswasser I, Stern MJ: Incidence, CT findings and rehabilitation outcome of patient with communicative hydrocephalus following severe head injury. Brain Injury 1988; 2: 267-272.
- Levin HS, Meyers CA, Grossmann RG, Sarwa M: Ventricular enlargement after closed head injury. Arch Neurol 1981; 38: 623-629.