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Post-traumatic hydrocephalus : Presentation, management and outcome — An apex trauma centre experience

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Abstract: The reported incidence of post-traumatic hydrocephalus (PTH) in literature varies from 0.7-29%. It is a treatable complication of head injury and presents with different clinical syndromes. We manage 38 cases of post-traumatic hydrocephalus in our hospital between January 2009 and April 2010. Clinicoradiology was studied at initial trauma, and at presentation with symptoms suggestive of increased ICP. Post ventriculo-peritoneal shunt outcome was analyzed. Sixty-eight percent cases were of severe head injury. Contusions (73.6%) and SAH (50%) were the most common CT findings. Sixty-five percent cases had been managed by decompressive craniectomy at the time of initial injury. Deterioration in consciousness (57.8%) and hemiparesis (26.3%) were most common presenting features of PTH, usually after 65±38 days (mean±SD) of initial injury. Patients were managed by antibiotic impregnated ventriculo-peritoneal shunt. Shunt revision and infection rates were 18.4% and 12.8% respectively. Thirty cases (78.9%) improved, 2 (5.2%) showed no improvement and 6 (15.7%) died. PTH is a treatable complication of head injury with a favorable outcome and therefore should be aggressively managed by CSF shunting.

Keywords: head injury, hydrocephalus, ventriculo-peritoneal shunting

INTRODUCTION

Post-traumatic hydrocephalus (PTH) as a clinico-pathologic entity has been recognized since Dandy's report in 1914[1]. It is relatively rare and only a few series in the world literature describe it with variable rates of incidence[2,3]. Recognition of PTH is often confounded by attributing the unresolved or added symptom to the primary or secondary injury inflicted upon brain by the trauma. Established causes of secondary brain injury like cerebral edema, hypoxia, ischaemia and infection running in the neurosurgeon's mind surrendering to an unfortunate fatalistic outcome often precludes early identification of this treatable cause of morbidity and mortality. PTH is an active and progressive process of excessive cerebrospinal fluid (CSF) accumulation due to liquorodynamic disturbances following cranio-cerebral injury[4].

MATERIAL AND METHOD

This retrospective study was carried out at Jai Prakash Narayan Apex Trauma Centre, All India Institute of Medical Sciences, New Delhi. Case records of all 38 patients of PTH managed in our centre between January 2009 and April 2010 were reviewed. The aim of the study was to evaluate the clinical and radiological features of each case, both at the time of initial injury and at the time of presentation with features of hydrocephalus. Cases were reviewed with demographic profile, mode of injury, GCS and CT findings at time of initial presentation. Line of management at time of initial injury – surgical or conservative – was noted. All clinical features at the time of presentation of PTH were included to complete the spectrum. Non-contrast CT scan brain showing ventriculomegaly with periventricular lucencies was taken as radiological criteria of hydrocephalus. This was to exclude ventriculomegaly due to loss of brain volume (hydrocephalus ex-vacuo).

All patients were managed with medium pressure antibiotic impregnated ventriculoperitoneal shunts. Post-operative results and complications were analyzed.

RESULTS

Clinical features at the time of presentation of head injury: Out of 38 patients of PTH, 31 were male and 7 female. The mean age was 33.7 +/- 16 (mean +/- SD) with the range from 2 to 65 years. Road traffic accident was the most common mode of injury with 26 cases

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(68%); 12 (32%) were due to fall from height. Severe head injury (GCS d"8) was present in 26 (68%) cases at initial trauma.

CT scan findings at initial trauma :

Features	No. of Cases	%
Cerebral contusions	28	76
Subarachnoid Haemorrhage	19	50
Subdural haematoma	18	47
Intraventricular haemorrhage	3	8
Edema	3	8

Management of cases at initial trauma : Decompressive craniectomy (DC) was done in 25 (65%) patients, and 13 (35%) cases were managed conservatively.

Clinical features at the time of presentation of PTH: In our study, patients presented with various combinations of clinical features after 65 +/- 38 days (mean +/- SD) of initial injury. Decrease in GCS, found in 22 (58%) cases, was the most common presentation.

Clinical features at time of PTH	Cases	%
Decrease in level of consciousness (GCS)	22	58
Hemiparesis	10	26
Persistent/Worsening headache	9	23
Vomiting	7	18
Arrest of neurological improvement	4	11
Speech problems/Dysphasia	4	11
Seizures	3	8
Incidentally on follow-up scans prior to cranioplasty	2	5
Incontinence of urine	1	3

Results of ventriculoperitoneal shunting: Thirty patients (78%) showed improvement in clinical features after ventriculo-peritoneal shunting. Two cases (5%) had no improvement and six (15%) died. The cause of death in 3 patients was uncontrolled septicemia and ventilator associated pneumonia. In other 3 cases, the cause of death was ventriculitis and uncontrolled infection which could not be resolved despite attempts to either revise or exteriorize the shunt or Ommaya reservoir placement. Overall, shunt infection occurred in 5 cases (12.8%) cases, and blockade in 2 (5%) cases. In these 7 cases (18.4%), shunt revision was done.

DISCUSSION

Incidence of PTH in world literature is quite variable, ranging from 0.7 to 29 %[2,3,5,6,7]. In many cases, initial brain damage leading to cerebral atrophy with secondary ventriculomegaly (hydrocephalus ex-vacuo) can give a false impression of PTH and if only CT finding of ventriculomegaly is taken into account, then the incidence rises to 30-88 % [8,9]. Kishore et al found that only 13.7 % of patients with ventriculomegaly had PTH[7]. In the present study 26 cases out of 768 cases of severe head injury admitted at our center developed PTH. Thus the incidence was 3.4 % in severe head injuries.

PTH may present with various clinical syndromes including obtundation, failure to improve, psychomotor retardation, memory loss, gait ataxia and incontinence[2,10]. Prolonged coma or arrest in clinical progress in conscious patients should arouse suspicion of hydrocephalus. In such patients funduscopy may reveal papilledema[2,20]. PTH commonly occurs in first year post-trauma and has been described as early as within 7 hours of injury [2,11, 13]. In our series 1 patient was diagnosed on 13th post-trauma day and 1 as late as 182nd day.

CT scan findings at time of initial trauma have been described with variable rates of incidence of findings[2,8,12,13,14,15,16]. Subarachnoid hemorrhage (SAH) has been cited as the most important pathology leading to development of PTH[2,10,15,16,18]. Increase in outflow resistance and CSF pressure in experimental rats following subarachnoid infusion of plasma or whole blood has been found[18]. 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[19]. Sub-dural hematoma[17], intra-ventricular hemorrhage[13], diffuse edema and cerebral contusion[14] have been found as the most common CT findings in other studies. Contusions, SAH and subdural hematoma were common findings in our study,.

Decompressive craniectomy (DC) has been found to be associated with development of PTH, [2,12,15,20,21] by altering CSF pressure dynamics, mechanical blockage around convexities [19] or inflammation of arachnoid granulations by post-surgical debris[21]. It leads to flattening of normally dicrotic ICP waveforms due to transmission of pressure pulse through the open cranium[21]. Since arachnoid granulations function as pressure-dependent one-way valves from subarachnoid space to draining venous sinuses, the disruption of pulsatile ICP dynamics

results in decreased CSF outflow. Hence, early cranioplasty should lead to restoration of normal intracranial pressure dynamics and spontaneous resolution of hydrocephalus. Higher incidence of PTH has been found with extended DC and re-operation [17]. In our study 25 out of 38 cases of PTH had undergone DC.

Significant ventricular dilatation may require radionuclide cisternography[8,10]. Overnight ICP recording, lumbar or ventricular infusion tests or even diagnostic lumbar puncture drainage are of importance in diagnosis of PTH especially if CT Scan is inconclusive[2,8,10,20]. The present study forms the basis on which future studies can be planned in our high input trauma centre where in only 16 months we have treated 38 cases of so called 'a rare clinical entity'- post-traumatic hydrocephalus.

CONCLUSIONS

In our study we have found that post-traumatic hydrocephalus can occur even in milder head injuries. It has a variable period of presentation after initial injury. Clinical features vary widely in individuals. Its resemblance with hydrocephalus ex-vacuo can be confounding on CT scans. Needless to emphasize that it is usually lost in oblivion due to other causes of neurological deterioration or lack of progress keeping the neurosurgeon obsessed. It also calls for use of other techniques to diagnose PTH rather than only relying on CT scan findings.

PTH is a treatable complication of head injury with a favorable outcome. It should thus be aggressively sought for and managed.

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