Post-traumatic Hydrocephalus : Experience in 17 Consecutive Cases

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Abstract

Background: Ventriculomegaly after head injury is one of controversial debate. Currently there is no definite way to distinguish post-traumatic hydrocephalus (PTH) from cerebral atrophy. The favourable outcome is only from CSF shunting in patients with true post-traumatic hydrocephalus, not hydrocephalus exvacuo.

Method: 17 patients with post-traumatic hydrocephalus were retrospectively reviewed from January 1993 to February 1996 to determine risk factors and guidelines for the management of this problem.

Results: These 17 patients represented 1.6 per cent of the 1080 head-injured patients seen at Songklanagarind Hospital during that period. 385 patients were classified as severe head injury in whom 7 were complicated with post-traumatic hydrocephalus. Our study found a high incidence of correlation between PTH and decompressive craniectomy. The late effect of decompressive craniectomy may cause CSF blockage around the convexities and hydrocephalus. The diagnoses were based on clinical manifestations and CT scan appearances. The outcome was related closely to the initial GCS score and the method used for diagnosis.

Conclusion: Post-traumatic hydrocephalus was 1.8 per cent in patients with severe head injury. Late neurological deterioration confirmed by CT scan findings was more useful than CT scan findings alone. CSF shunting was effective in patients with ventriculomegaly who had clinical signs and symptoms of increased intracranial pressure from post-traumatic hydrocephalus.

Key word : Hydrocephalus - Post-trauma - Seventeen Cases - Experience

Post-traumatic hydrocephalus (PTH) is one of the sequels of head injury. The controversial problem is distinguishing true post-traumatic hydrocephalus from hydrocephalus exvacuo which is secondary to severe brain damage, as both conditions present with ventriculomegaly. It is always

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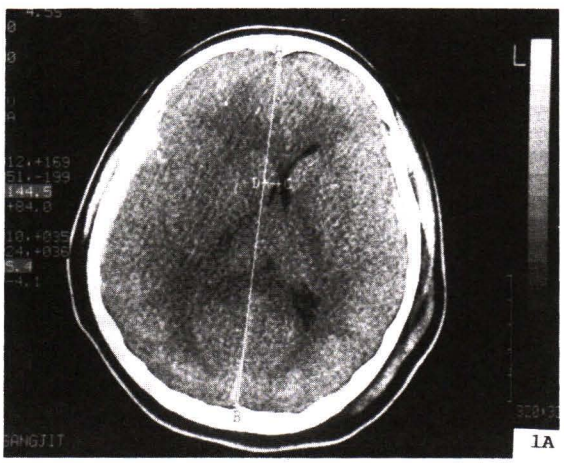
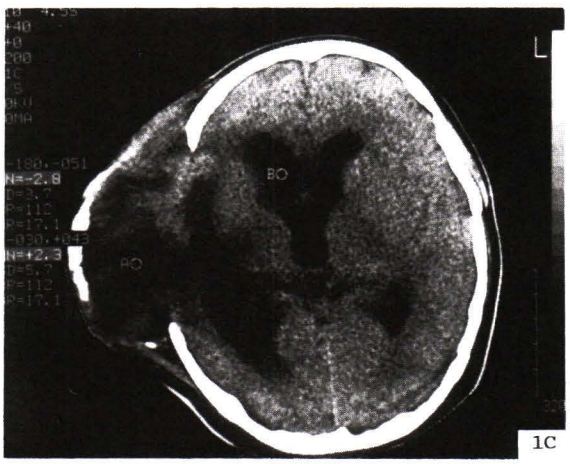
in doubt whether patients with ventriculomegaly are suffering from hydrocephalus or traumatic cerebral atrophy, and if they will benefit from the surgical placement of a ventricular shunt. Some patients do not improve after a CSF shunting operation. What is the suitable criteria for patient selection for CSF shunting? Our objectives were to describe the possible risk factors of PTH and to present the criteria for selection, based on our experience at Songklanagarind Hospital, for CSF shunting in patients with post-traumatic ventriculomegaly. Our experience may give an alternative management of post-traumatic ventriculomegaly and hydrocephalus.

MATERIAL AND METHOD

1080 head-injured patients were studied retrospectively between January 1993 and February 1996. Of these 1080 patients, 17 were diagnosed with PTH. Medical records and CT scan film were reviewed. General and specific data were collected. Special attention was given to the initial GCS score at ER, how PTH was diagnosed, time between injury and shunting, and the results of treatment. The patients were classified into 2 groups based on method to diagnosis:

Group A: PTH was defined on both clinical and radiographic grounds. The clinical signs and symptoms of PTH included late neurological deterioration and increased bulging at the cranial defect. A CT scan was used to confirm the diagnosis. The details of the CT findings in PTH are described below. The CT scan of a patient in group A is shown in Fig. 1.





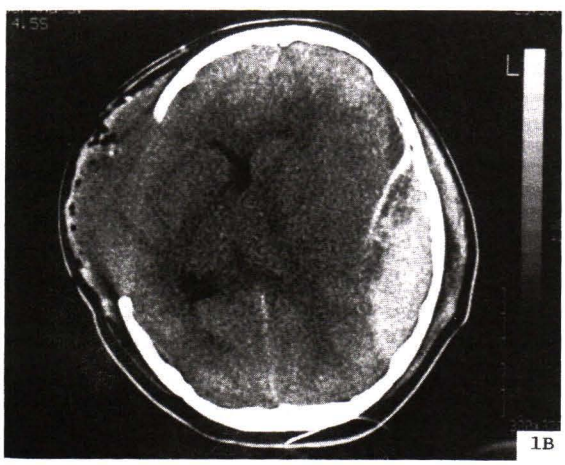


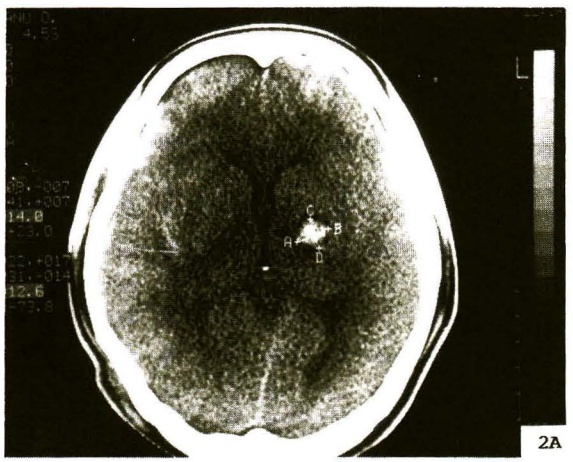
Fig. 1. Fig. 1A was the first CT scan done on December 8, 1993 showing thin acute subdural hematoma at the right parietal area and right hemispheric swelling. The midline was shifted to the left 5.4 mm. Due to the patient's clinical signs and symptoms of uncal herniation, an emergency craniotomy was performed to removed the hematoma. During the clot removal, the brain was bulging severely from the craniotomy site. Decompressed craniectomy was done for closure enhancement. Fig. 1B was the immediate postoperative CT scan showing contralateral epidural hematoma. Fig. 1C was the CT scan done 8 months after the time of head injury showing severe hydrocephalus. Third ventricle and temporal horns were enlarged. Sulci were not seen.

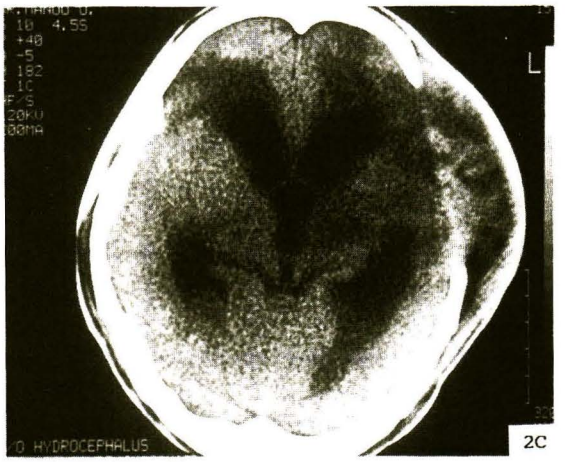
Group B: All of the patients in this group were not recovered from the initial severe brain injury. Late neurological deterioration from PTH can't be detected because of masking by the initial insults. So the diagnosis of PTH was based on CT scan finding only. In our hospital, the patients were routinely sent for a repeat CT scan because of follow-up observations or no neurological improvement within a period of 1 or 2 months. The CT scan of a patient in group B is shown in Fig. 2.

A baseline CT scan was obtained for all patients, with the exception of those transferred from a surrounding provincial hospital, to compare ventricular size during treatment. Radiological diagnosis of PTH was based on the presence of a distended appearance of the anterior horns of the lateral ventricles with enlargement of the temporal horns and third ventricle and normal or absent sulci[1-4]. Periventricular interstitial brain edema also aided in the diagnosis[5,6].

The possible risk factors of developing PTH are decompressive craniectomy, type of traumatic lesions, evidence of CNS infection, traumatic subarachnoid hemorrhage (SAH), and brainstem injury[7,8].

The Glasgow outcome scale was used to assess the outcome; GR as good recovery, MD as moderate disability, SD as severe disability, VS as vegetative state, and D as death[9].





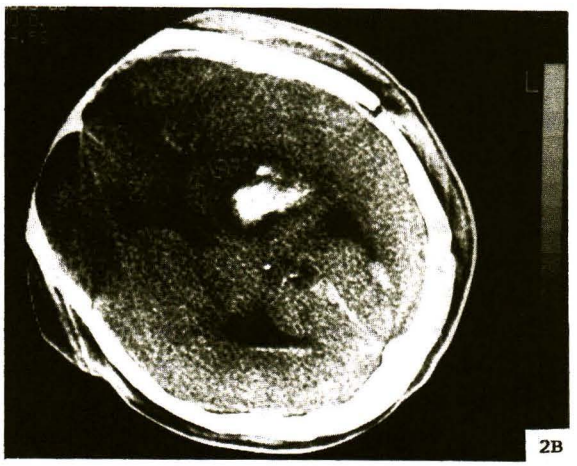


Fig. 2. Fig. 2A was the first CT scan done in the ER on August 11, 1993 showing small intracerebral hematoma of the size 14x12.6x1 slice at left basal ganglia. Thin acute subdural hematoma was noted at left fronto-parietal area. The midline was not shifted. Neurological deterioration led to performing of craniotomy for subdural clot removal. Four days after admission, the second CT scan, (Fig. 2B) was performed after the craniotomy due to neurological deterioration, which showed basal ganglia hematoma enlargement. Finally, a craniectomy was done. Fig. 2C was the CT scan done 2 months after head injury showing severe hydrocephalus. All ventricles were enlarged including third ventricle and both temporal horns. There was low density lucency at both frontal horns. Sulci were absent. (Note the similarity of the ventricular enlargement in Fig. 1 and 2.)

RESULTS

General Clinical Findings:

Between January 1993 and February 1996, 1080 head injury patients with varying severity (classified by Glasgow Coma Scale score) were admitted to Songklanagarind Hospital, 17 (1.6 per cent) of these were diagnosed with PTH. 915 medical records of these patients were found. There were 385 patients with severe head injury, comprising the prevalence of 42.1 per cent. There were 17 cases with the diagnosis of PTH the general data of which is summarized in Table 1. Mean age of the patients was 30 years with a range from 11 to 80 years. Our data indicate that there were 4 times as many males as females (4.6:1). The most common cause of injury (12 cases) was motorcycle accidents. We obtained the initial GCS at ER in 12 cases as these patients were transferred directly from the scene of the accident to our hospital. Of these cases, 7 patients were considered as comatose (2T in 1 case, 3T in 2 cases, 5T in 2 cases, and 6T in 2 cases) comprising of 1.8 per cent in patients with severe head injury (7/385). The 5 remaining cases had GCS scores of 9T, 11, 11,13, and 14. The 5 cases whose initial GCS scores were not known, because of incomplete referral notes, had been treated at provincial general hospitals and referred for further management. In this group of patients, the initial GCS scores at our ER were 6T, 8T, 9T, 11, and 14.

Table 1. Summary of about the 17 patients with post-traumatic hydrocephalus.

study period (months)	38
no. of total head-injured patients	1,080
No. of PTH (%)	17 (1.6)
Male:Female ratio	4.6:1
mean age (years)	30
common cause of injury	Motorcycle (12/17)
known initial GCS score at ER (cases)	12
total craniectomy cases	69/1080
PTH with craniectomy (cases)	7
Length of time from injury to diagnosis -within 2 months - > 2 months	 12 3
time between onset of injury and CSF shunting -within 90 days ->90 days	 8 7
mean follow-up period (months)	12
improvement after shunting (cases)	8/16
mortality	1/17

Specific Clinical Findings:

Of the 17 cases with PTH, 7 cases had a large craniectomy for closure enhancement after a clot or contusional brain tissue was removed. The other case having a craniectomy had a depressed compound fracture skull. During the study period, 69 patients had a decompressive craniectomy due to malignant brain swelling after hematoma removal. Most of these cases involved an acute subdural hematoma. In this group of patients, 10 per cent (7/69) developed PTH later.

In twelve patients, the diagnosis of PTH was reached from 4 days to 2 months after the initial head injury. The other three patients were diagnosed at 4, 6, and 10 months post injury. One patient, with a brainstem injury, had the most rapid onset of PTH from obstructive hydrocephalus. This patient died from severe injury to the vital structure. The exact time of injury in two cases could not be identified. One patient presented with a chronic subdural hematoma. The other patient had an incomplete medical record. The most common symptom was deterioration of consciousness. We found a bulging craniectomy site in all 7 patients who had received a decompressive craniectomy. The other clinical signs and symptoms were progressive headache (2 patients), papilledema (1 patient), severe dizziness (1 patient), dementia + gait apraxia (1 patient), and cheyne-stokes respiration (1 patient). Acute subdural hematoma was the most common pathology in patients from both groups.

7 of the 9 patients in group A, who had clinical signs and symtoms of PTH, showed improvement after treatment. There were 8 patients in group B who were diagnosed by follow-up CT scan. There was only one case in this group who improved with CSF shunting. In total, 8 cases of the 17 cases with PTH showed improvement after shunting. The outcomes of group A and B are shown in Table 2. The timing between the onset of injury and the shunting operation was within 90 days in 8 cases, and more than 90 days in 7 cases. Two cases improved with conservative treatment consisting of acetazolamide and close observation.

CT findings:

CT scans of 12 patients were reviewed. The 2 patients transferred from other facilities did not have early CT scan films. Follow-up CT scan films of the remaining 10 patients showed a definite increase in the size of the anterior and temporal

Table 2. Comparison of the initial GCS scores and outcome of the patients in group A and B (see text).

	Patient Group A	Patient Group B
no.of patients	9	8
Initial GCS score		
-2T-6T	1	6
-8T-15	5	0
-unknown	3	2
outcome		
-GR	1	0
-MD	3	1
-SD	4	1
-VS	0	6
-D	1	0

GR=good recovery, MD=moderate disability, SD=severe disablity, VS=vegetative state, D=death, 'unknown' means the transferred patients with no record of an initial GCS score.

horns, and the third ventricle. Absence of sulci and periventricular lucency were found in 3 and 4 cases respectively. Subsequent scans to diagnose hydrocephalus were done 4 to 33 weeks after the injury with the average time being 14 weeks. Only 2 cases had evidence of subarachnoid hemorrhage on CT scans done immediately after arrival at emergency room. One showed a blood clot in sulci and the other in the sylvian fissure. The CT scans of two patients are illustrated in Fig. 1 and 2. CT findings of patients in group A and B were indistinct.

Possible risk factors

16 of 17 cases with PTH can determine possible risk factors as shown in Table 3. Two cases with poor outcome had more than one factor. A case whose possible risk factor could not be determined was diagnosed as diffuse axonal injury.

Outcome

The overall results are shown in Table 4. The follow-up period ranged from 3 months to 40 months. The mean follow-up period was 12 months. The patients with poor outcome were in group B and comatose on arrival. Five of the seven patients who had an initial GCS score of 2T-6T, were still in VS as shown in Table 4. Six patients in group B were also in VS as shown in Table 2.

DISCUSSION

In our study, PTH was about 1.6 per cent of the total cases with head injury (17 cases of 1080 cases of head injury) and 1.8 per cent of patients with severe head injury (7 cases in 385 cases with severe head injury) which correlates to the current literature. It has been reported that 0.7 - 8 per cent of head injury patients experience ventricular enlargement[8,10-12]. Three reports showed the criteria for diagnosing PTH or ventricular enlargement. The first article reported the incidence of ventricular enlargement to be 72 per cent in patients with severe head injuries. Enlargement was defined by the ventricle-brain per cent ratio (VBR)[13]. The second article used CT scan appearance and found that the incidence of ventricular enlargement was about 30.5 per cent (61/200) in patients with severe head injuries[3]. Only 11 patients of the total 61 patients with ventricular enlargement met the criteria for hydrocephalus. The study in the third article showed that the incidence of normal and high pressure hydrocephalus was 20 per cent in patients with severe head injuries[5]. They used profiles from the CSF dynamic study to define which patients suffered from true hydrocephalus. In our series, the criteria for defining ventricular enlargement from true hydrocephalus was clinical manifestation and CT scan appearances[3,8]. Our study included head-injured cases at all levels of severity. Nearly half of the patients with PTH in our series should not be comatose.

Possible risk factors (as shown in Table 3)

1. In our study, PTH was associated with decompressive craniectomy. Further control study needs to support this concept. It has been known a long time that this operative procedure does not improve the outcome, but causes infarction to the protruding brain tissue[12,14,15]. Late effects may cause a blockage of CSF flow around the convexities and may finally produce hydrocephalus. Although a number of potential causes of PTH are readily identifiable, late effects of decompressive craniectomy have not been reported[7]. But recently, Polin et al[16] studied 35 patients who underwent bifrontal decompressive craniectomies for refractory cerebral edema and later found shunt-dependent hydrocephalus during the postoperative course in 10 patients (28.6 per cent). The high incidence of the correlation between PTH and decompressive craniectomy in our series (7 cases) may

Table 3. Comparison of possible risk factors for PTH development and outcome of 17 patients with PTH.

Possible risk factors	GR	MD	SD	VS	D
Decompressive craniectomy		3		4	
ASDH and/or contusion without decompressive craniectomy	2		2	1	
Brainstem injury					1
CSDH			1		
SAH+contusion+meningitis			1		
SAH+decompressive craniectomy				1	
Unknown			1		

GR = good recovery, MD = moderate disability, SD = severe disability, VS = vegetative state, D = death,
ASDH = acute subdural hematoma, CSDH = chronic subdural hematoma, SAH = traumatic subarachnoid hemorrhage.

Table 4. The overall results of the cases with post-traumatic hydrocephalus classified into 2 groups by initial GCS scores. An initial GCS score of 2T-6T were patients who were transferred directly from the scene of the accident.

Initial GCS score	GR	MD	SD	VS	D	Total
2T-6T	0	1	1	5	0	7
8T-14	1	1	2	0	1	4
unknown	0	2	2	1	0	6
Total	1	4	5	6	1	17

GR = good recovery, MD = moderate disability, SD = severe disability, VS = vegetative state, D = death.

support the concept. The outcomes in this group were disappointing because 4 cases had VS, and 3 cases had MD. CSF shunting reduced the bulging of the cranial defect in all cases, but the outcomes were still very poor. The patients who had a decompressive craniectomy for closure enhancement often suffered from severe head injuries.

2. Acute subdural hematoma and cerebral contusion were most commonly found in our study. These two types of lesions may result in CSF flow obstruction at the convexities or basal cistern by associated intracranial hypertension or later with adhesive arachnoiditis(7).

3. We found evidence of traumatic SAH in only 2 of 10 CT scans. Because of the size of the study, we cannot conclude that traumatic SAH is not a risk factor. Many reports showed PTH can occur secondary to traumatic SAH by CSF absorption blockage or CSF flow impediment from adhesive arachnoiditis(7).

4. There was one case with prior meningitis from CSF rhinorrhea and one case with brainstem injury producing acute hydrocephalus.

5. Results of treatment related closely to the initial GCS score and the method of diagnosis.

Initial GCS Scores

The 7 patients who were comatose on arrival (GCS score = 2T-6T) had disappointing results; 5 with VS, 1 with SD, and 1 with MD. In contrast, the 10 patients with initial GCS scores of 8T-14 and unknown initial GCS scores had more favourable outcomes; 1 with GR, 3 with MD, 4 with SD, 1 with VS, and 1 D. The disabled patients showed some improvement after CSF shunting but clinical improvement did not come up to a favoura-

ble outcome. There were 2 cases who showed improvement with conservative treatment.

Method of Diagnosis

The comparison of the patients in groups A and B will help guide the diagnosis of PTH in the future. Clinical signs and symptoms are necessary for diagnosis of PTH. CT scan appearances should be used only to confirm the diagnosis. CT scan should not be the sole factor in diagnosis. Seven of the nine patients in group A showed improvement after treatment. The outcome of patients in group A was more favourable than those in group B. Only one patient in group B, an 11 year-old boy, improved to MD after 16 months of follow-up.

The reasons for the poor outcome of patients in group B and patients who arrived comatose might be directly related to the severity of their injuries as the combination of hydrocephalus and brain atrophy might be involved. CSF shunting corrected one part of the patients' suffering but structural damage from the previous diffused axonal injury could not be corrected.

CSF dynamic study might be useful to identify which patients have true hydrocephalus, but can be performed only in large, well equiped medical centers[5]. Investing in equipment to diagnose the small group of patients with PTH might be inappropriate in a developing country. 18 - 20 per cent of patients with ventriculomegaly met the criteria for hydrocephalus[3,5]. The diagnosis should be based strictly on the late neurologic deterioration and CT scan findings as described in the above section.

In conclusion, there was a wide variation of incidence of PTH in the literature. They ranged from 0.7 - 72 per cent occurrence rate in patients with severe head injuries[3,5,8,10,11,13,17]. In our study, PTH was 1.6 per cent of total head-injured patients and 1.8 per cent of patients with severe head injury. The decompressive craniectomy for closure enhancement in the situation of malignant brain swelling after acute subdural hematoma removal should be done with caution. Because the late effect of this procedure may be one of the causes of PTH. Patients with severe head injuries who had an initial GCS score between 3 and 8 and patients who were mainly diagnosed by CT showed little benefit from CSF shunting. The patients with ventriculomegaly who had clinical signs and symptoms of PTH were good candidates for CSF shunting.

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ฮัยโดรเซฟาลัสหลังบาดเจ็บที่ศีรษะ: ประสบการณ์จากผู้ป่วย 17 ราย

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ศักดิ์ชัย แซ่เฮ้ง, พ.บ., บุญเลิศ ศรีไพโรจน์กุล, พ.บ.*,*

คณะผู้วิจัยได้ศึกษาผู้ป่วยฮัยโดรเซฟาลัสหลังบาดเจ็บที่ศีรษะ 17 รายในโรงพยาบาลสงขลานครินทร์ ระหว่างเดือนมกราคม พ.ศ.2539 ถึงเดือนกุมภาพันธ์ พ.ศ.2539 พบมีจำนวนร้อยละ 1.6 ของผู้ป่วยบาดเจ็บที่ศีรษะ (17/1080) และร้อยละ 1.8 ของผู้ป่วยบาดเจ็บที่ศีรษะรุนแรง (7/385) มีความสัมพันธ์กับการผ่าตัดเอากะโหลกออกเพื่อลดความดัน การวินิจฉัยแยกโรคระหว่างฮัยโดรเซฟาลัสจริง ๆ กับสมองเหี่ยวซึ่งทำให้โพรงสมองโตขึ้นได้เหมือนกันควรใช้ลักษณะอาการทางคลินิกทางระบบประสาทที่เลวลงภายหลังร่วมกับภาพรังสีซีทีสแกน ซึ่งจะทำให้การรักษาได้ผลด้วยการใส่เครื่องระบาย

คำสำคัญ : ฮัยโดรเซฟาลัส – หลังบาดเจ็บที่ศีรษะ – ผู้ป่วย 17 ราย

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