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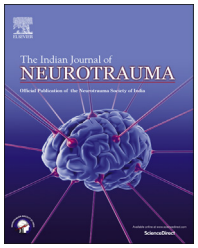

Original Article

Theco-peritoneal shunt for post-traumatic hydrocephalus – A valuable adjunct?

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ABSTRACT

Introduction: Majority of patients undergoing decompressive craniectomy (DC) for traumatic brain injury may develop post-traumatic hydrocephalus (PTH). This remains a challenge to manage as most patients are tracheotomised and run high risk of shunt infection and malfunction following VP shunt. Theco-peritoneal shunt (TP shunt) may be an attractive alternative in this scenario.

Aims & objectives: To assess the role of TP shunt in PTH with respect to the patient population, complications & clinical outcome in TBI.

Materials & methods: In this retrospective study over 32 months (September 2009–April 2012) records of all patients of TBI who underwent TP shunt at a level 1 trauma centre were analysed. Clinical, radiological and follow up data were reviewed in all cases.

Observations & results: A total of 22 TP shunts were done in the study period. Of these 77.2% patients had severe head injury, 18.2% had moderate head injury and 4.5% had mild head injury. The most common initial CT findings were acute SDH in 86.4% and contusions in 81.8% of the patients. 95.5% patients underwent DC at initial admission. Most common presentations of PTH were bulging flap (95.5%), vomiting (72.7%) and headache (63.3%).

91.9% of the patient had tracheostomy in situ. Of these, 5 patients (25%) had positive tracheal cultures and six (27.3%) patients had scalp bedsore/flap infection prior to TP shunt. Following TP shunt, 77.3% patients showed clinical &/or radiological improvement. Shunt infection and revision rates were 0% and 22.7% respectively.

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Conclusions: PTH in patients with TBI needs a holistic approach in view of scalp bed sores, tracheostomy and altered ventricular anatomy. TP shunt is a safe, minimally morbid procedure which should be considered as an attractive alternative to VP shunt in patients with PTH.

1. Introduction

Patients with of traumatic brain injury (TBI) are at increased risk of developing Post-traumatic hydrocephalus (PTH) following decompressive craniectomy.[1,2] PTH is an active and progressive process of excessive cerebrospinal fluid (CSF) accumulation due to liquorodynamic disturbances following cranio-cerebral injury.[3] Communicating hydrocephalus is more common in this setting and its management remains challenging as most patients are tracheostomised and are at high risk of shunt infection and/or malfunction following ventriculo-peritoneal shunt. Concomitant factors such as decubitus ulcers on scalp, skin abrasions and lacerations over chest and neck, altered ventricular anatomy in these patients further increases the risk of placing ventriculo-peritoneal shunt. Theco-peritoneal shunt (TP shunt) is a relatively simple extra cerebral procedure for communicating hydrocephalus especially in tracheostomised patients. The aim of this study was to evaluate TP shunt as an alternative to ventriculo-peritoneal shunts (VP Shunt) in PTH.

2. Material and methods

In this retrospective study over 32 months (September 2009–April 2012) at JPNA Trauma Centre, All India Institute of Medical Sciences, New Delhi, all patients who underwent TP shunt for communicating PTH with/without previous VP shunt malfunction were included in the study. The demographic profile, mode of injury, initial GCS and CT finding (Table 1) at admission were reviewed for all the patients. Factors predisposing to shunt malfunction such as tracheostomy, scalp bedsore/surgical site infection, associated thoracoabdominal injuries, with lacerations over chest, back and neck reviewed. Ventriculomegaly with periventricular lucencies on CT along with bulging flap of decompressive craniectomy and/or neurological deterioration/non-improvement were taken as criteria for PTH. Chhabra® lumbar-peritoneal hydrocephalus shunt system was used in all patients. Neurological outcome and complications were analysed for all patients. TP shunt malfunction was diagnosed on basis of neurological deterioration/non-improvement in the presence of re-bulging

Table 1 – Demographic profile, mode of injury, initial features with associated injuries.

Case no.	Age (year)/sex	Mode of injury[a]	Initial GCS	Initial CT features[b]	Initial manage-ment	Menin-gitis	Associated injuries[c]	Extensive skin abrasions
1	31/M	RTA	E1VetM2	SDH, Cont. SAH, Oedema	DC	+	–	–
2	42/M	RTA	E4V5M6	SDH, Oedema	DC	–	–	–
3	35/M	RTA	E1VetM2	SDH, Cont.	DC	–	BTA, BTC	+
4	23/M	RTA	E1VetM4	SDH, Cont	DC	–	–	–
5	3/M	FFH	E3VetM4	SDH, Cont	DC	–	–	–
6	22/M	RTA	E1VetM4	Cont, Oedema	DC	–	BTA, BTC	–
7	7/F	RTA	E2V4M5	SDH, Cont	DC	–	BTA, BTC	–
8	12/M	FFH	E3VetM5	SDH, Cont,	DC	–	–	–
9	56/M	RTA	E3VetM4	SDH, Cont.	DC	–	–	–
10	3/M	RTA	E3VetM4	SDH	DC	–	–	–
11	42/M	RTA	E1VetM5	SDH, Cont, SAH	DC	–	BTA, BTC	+
12	50/M	RTA	E1VetM4	SDH, Cont	Conser-vative	–	–	–
13	35/M	RTA	E2VetM3	SDH, Cont	DC	–	–	–
14	4/F	RTA	E1VetM4	SDH, Cont	DC	+	–	–
15	20/M	RTA	E3VetM3	SDH, Cont	DC	–	BTC	–
16	63/M	RTA	E3VetM4	SDH	DC	–	–	–
17	38/M	RTA	E1VetM4	SDH, Cont	DC	–	BTA, BTC	–
18	20/M	FFH	E1VetM2	Cont, Oedema	DC	–	BTA, BTC	+
19	45/M	RTA	E1V2M5	SDH, cont. SAH, Oedema	DC	–	–	–
20	40/M	RTA	E1VetM4	Cont, Oedema	DC	–	BTA	–
21	10/M	RTA	E1VetM2	SDH	DC	–	–	–
22	8/M/	FFH	E3VetM5	SDH, Cont.	DC	–	–	–

a Road traffic injuries (RTA), Fall from height (FFH).
b Acute subural haematoma (SDH), Contusion – (Cont), Subarachnoid haemorrhage (SAH), Diffuse oedema (Oedema).
c Blunt trauma abdomen (BTA), Blunt/penetrating trauma chest (BTC).

of DC flaps and persistent ventriculomegaly with periventricular lucencies.

3. Results

3.1. *Demographic and clinical features at initial trauma (Table 1)*

A total 22 patients underwent TP shunt during the study period. Of these 20 were male and 2 were female. The mean age was 27.68 ± 18.2 years (mean ± S.D.), range 3–63 years. Most common mode of injury was road traffic injuries (81.8%), followed by fall from height in 18.2%. 17 (77.2%) patients had severe head injury, 4 (18.2%) had moderate head injury and 1 (4.5%) had mild head injury.

3.2. *Initial management of patients (Tables 1–3)*

The Initial CT findings and clinical features are given in Tables 2 and 3 respectively. 21 patients (95.4%) underwent decompressive craniectomy; while 1 patient was managed conservatively. 2 patients had postoperative meningitis which was treated with intravenous antibiotics in anti-meningitic doses. No hydrocephalus was seen at time of discharge in these two patients.

Mean duration of presentation of PTH after DC was 64.2 ± 94.5 days with a range of 15–421 days.

3.2.1. *Factors predisposing to VP shunt infections/ malfunction (Table 4)*

Five (25%) of the 20 patients with tracheostomy in-situ had positive BAL microbial cultures, with redness around tracheostomy site. The common isolate was *Acinetobacter* sp. in all these cases. All patients of scalp bedsore/flap infection had at least one culture report positive over period of hospital stay and most common isolate was mixed skin flora. VP shunt was exteriorised in all 7 patients with suspected shunt malfunction, however no CSF culture was positive. CSF biochemical analysis showed evidence of meningitis in 1 patient. TP shunt was done when 3 consecutive CSF cultures were sterile.

3.2.2. *TP shunt Indication(s)*

Amongst the twenty-two patients who underwent TP shunt, twenty patients (91.9%) had tracheostomy in situ and twenty-one (95.4%) patients had ventilatory support at time of consideration for TP shunt. Of these, seven patients also had exteriorised VP shunts. One patient (case 12) presented with GCS of 13 and had to be intubated after he developed aspiration pneumonitis.

Table 2 – CT features at initial trauma.

CT features	% (Cases)
Acute subdural haematoma	86.4% (19)
Contusion	81.8% (18)
Diffuse oedema	27.3% (6)
Subarachnoid haemorrhage	13.6% (3)
Intraventricular haemorrhage	0%

Table 3 – Clinical features at presentation with PTH.

Clinical features at time of presentation with PTH	% (Cases)
Bulging DC flap	95.4% (21)
Vomiting	72.7% (16)
Headache	63.6% (14)
Seizure	31.8% (7)
Deterioration in GCS	18.2% (4)
Arrest of neurological development	18.2% (4)
Incidentally diagnosed on follow-up scans	13.6% (3)
Urinary incontinence	9.2% (2)
Hemi paresis	9.2% (2)

3.2.3. *Cranioplasty*

In our centre cranioplasty is offered to patients with GOS of at least 4 or when patient's relatives specifically request for the procedure. Of the 22 patients in this study, 3 presented with PTH at variable time period following cranioplasty and the remaining 19 developed PTH before cranioplasty could be considered.

3.3. *Results following TP shunting (Table 5)*

Mean follow-up duration was 18.4 ± 8.8 months (range 3–32 months). Seventeen patients showed improvement (77.3%) in clinical and radiological features after TP shunting.

Two patients showed no clinico-radiological improvement (9.2%) and 3 died (13.6%). The cause of death in all 3 patients was ventilator associated pneumonitis and sepsis. TP shunt malfunction was seen in 5 cases (22.7%). All patients with suspected shunt blocks underwent revision surgery. During surgery however, free flow was seen on removal of distal (abdominal) end, and it is hypothesized that the act of pulling out the distal end released the block. The distal end was reinserted through a separate abdominal incision in all cases. The CSF analysis and culture were sterile in all these patients.

Amongst the three patients who were asymptomatic at routine follow up, progressively increasing DC flap bulge was seen in two cases, along with persistent seizures which were poorly controlled on medications. The third case (case 12) was managed conservatively on acetazolamide. The patient subsequently developed progressively increasing headache and vomiting, which prompted the TP shunt.

4. Discussion

PTH as an entity is recognised since Dandy's report in 1914.[4] Incidence of PTH varies from 0.7 to 29% in different case

Table 4 – Associated factors in patients with PTH.

Factors	%(Cases)
Tracheostomy	91.9% (20)
Extensive chest/neck injuries involving skin	13.6% (3)
Scalp bed sores/flap infection	27.3% (6)
Blunt/penetrating trauma chest	27.3% (6)
Blunt trauma abdomen (conservatively managed)	31.8% (7)
Previous VP shunt malfunction/infection	31.8% (7)

Table 5 – Results and complications following TP shunt.

Case no	GCS at PTH presentation	PTH presentation after initial injury (days)	Tracheostomy ⫫	Scalp sore/flap infection	Previous VP shunt	TP shunt complication	GCS at discharge	GOS at 3 months
1	E1VtM2	40	+, BAL	–	+	–	E1VtM4	2
2	E4VtM5	80	+	–	–	–	E4V1M6	4
3	E1VtM2	15	+	+	+	–	E2VtM5	3
4	E1VtM3	30	+	–	+	–	E4VtM6	4
5	E4VtM5	70	+	+	–	Block	E4VtM6	4
6	E1VtM4	40	+	–	–	Block	E4VtM4	3
7	E4VtM6	421	+, BAL	–	–	–	E4V5M6	5
8	E4VtM6	285	+	–	–	–	E4V5M6	5
9	E2VtM5	34	+	–	–	–	E4VtM5	3
10	E4V5M6	20	–	–	+	–	E4V5M6	5
11	E2VtM2	30	+	+	+	–	E4VtM2	2
12	E4V3M6	35	–	–	–	Block	DIED	1
13	E1VtM2	35	+, BAL	–	–	–	DIED	1
14	E4VtM4	30	+, BAL	–	–	–	E4VtM4	3
15	E2VtM3	30	+	+	–	–	E4VtM3	2
16	E4VtM5	25	+	–	+	–	E4VtM5	4
17	E1VtM4	32	+	+	–	Block	E4VtM4	3
18	E1VtM2	36	+, BAL	+	–	Block	DIED	1
19	E1VtM4	45	+	–	–	–	E4VtM4	3
20	E1VtM4	25	+	–	–	–	E4VtM4	2
21	E1VtM2	40	+	–	–	–	E4VtM4	3
22	E1VtM5	35	+	–	+	–	E4VtM6	4

BAL – Bronchoalveolar lavage (BAL) culture positive.

series.[5–7] If only CT findings are considered the incidence for PTH approaches 30–88%,[8,9] due to post-traumatic cerebral atrophy and secondary ventriculomegaly, which may be seen in larger subset of patients. CT criteria (Gudeman[8]) and clinical evaluation is cornerstone of PTH management, radionuclide cisternography, overnight ICP recording, lumbar/ventricular infusion tests and even diagnostic lumbar drainage help in decision making in ambiguous cases with inconclusive CT scans.[5,8,10,11] Other imaging modalities MRI, single positron emission tomography may serve as ancillary tests.[11]

Clinical presentation may vary from seizures, memory loss, gait ataxia, urinary incontinence to obtundation, arrest in neurological improvement and prolonged coma.[12] On fundus examination papilloedema may be revealed in such patients. PTH may occur as early as within 7 h of injury,[13] however in our series 1 patient was diagnosed on day 15th post trauma and as late as 421 days post trauma.

Subarachnoid haemorrhage (SAH) has been cited as most important pathology in many studies,[5,14] CT findings of subdural haematoma, intraventricular haemorrhage, cerebral contusion and diffuse oedema at the time of initial trauma have been cited as the most common pathology in other studies.[5,12–14] In our study TBI with subdural haematoma and/or contusions were the most common findings. Alteration of CSF pressure dynamics, mechanical blockage around convexities, fibrous thickening of leptomeninges and inflammation of arachnoid granulations by mechanical debris have been implicated in the development of PTH.[1,2,5,15,16] DC patients have flattening of normal dicrotic ICP waveforms due to pulse pressure transmission through open cranium. The disruption of pulsatile ICP dynamics result in decreased CSF outflow, since arachnoid granulations function as one way pressure dependents valves. Hence early cranioplasty should lead to restoration of normal ICP dynamics and spontaneous resolution of PTH. In our study 21 (95.4%) patients of PTH underwent DC. Higher incidence of PTH has been reported after extended DC and reoperations.[1] Role of meningitis in postoperative period may be an underestimated risk factor in these studies in patients with communicating hydrocephalus.

CSF diversion via shunting is an accepted treatment for PTH. TP shunt for communicating PTH is extra cerebral procedure, relatively easier to perform and can be done under general anaesthesia or even there are reports of local anaesthesia use. It can be especially useful in patients with tracheostomy and with scalp bed sores as VP shunt is contraindicated in these patients due to extremely high risk of shunt infection/malfunction. TP shunt obviates the need for ventricular puncture, which may be difficult in patient with DC, in part due to altered ventricular anatomy and surface landmarks alteration. Technically although TP shunt appears easier for CSF diversion, intraoperative patient positioning may be difficult, and use of transportation boards, laparoscopy assisted abdominal end insertion in lateral position have been described in literature.[17] We position the patient lateral, and tilt the table by 30° for the insertion of the peritoneal end of the catheter.

Functioning of TP shunt is difficult to assess. CT scan evidence of reduction in ventricular size is usually delayed up to few weeks (Fig. 1). Clinical evaluation of status and preoperative and postoperative imaging comparison can be used to assess functional status of TP shunt. In addition to Serial CT scans and clinical evaluation, various techniques - laparoscopic assisted patency evaluation, intrathecal DTPA

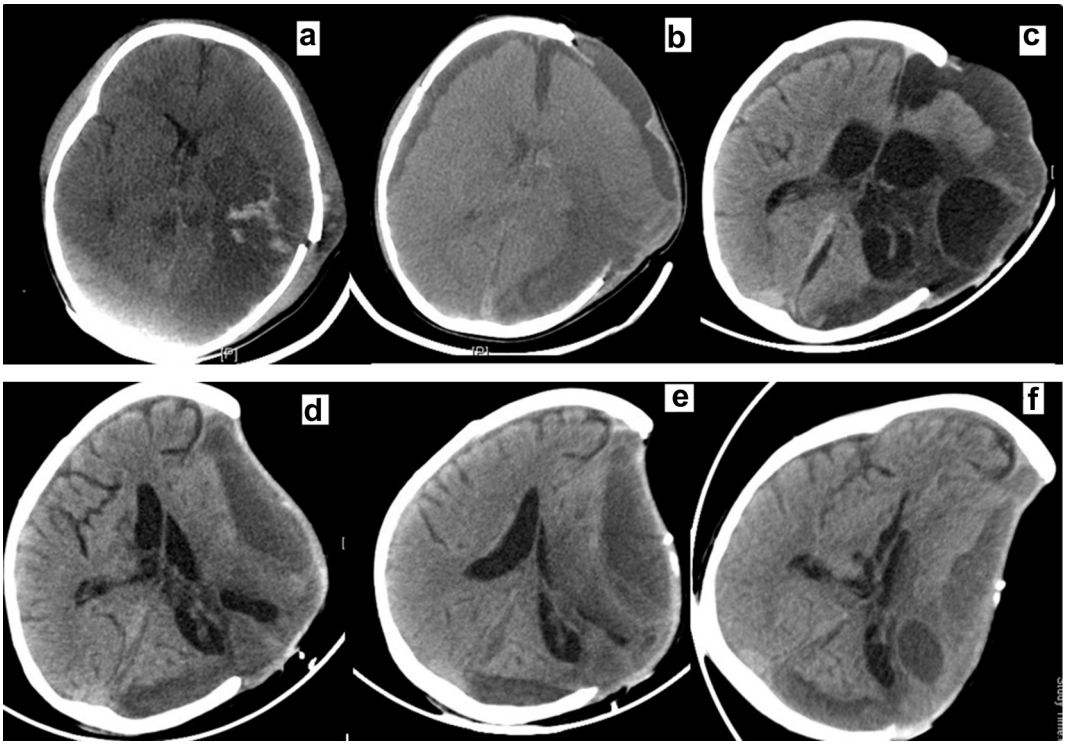

Fig. 1 – (a–c) Plain CT head of a 4-year-old male child who sustained Road traffic injuries, showing evidence of contusion with overlying parietal bone fracture (a), and was subjected to left fronto-temporo-parietal DC (b), developed flap bulge and PTH with Subdural hygroma on POD 35 (c). Plain CT head of the same patient following TP shunt at day 3 post shunt (d), 10 days post shunt (e) and 32 days post shunt (f) showing progressive decrease in ventricular size and flap bulge with resolution of subdural hygroma.

(diethylene triamine penta acetic acid), LP shuntography and thermosensitive determination of patency have used,[17] although we do have experience with any of these methods.

Patient improvement has been reported after VP shunt for PTH in 78.9% of patients in a previous study done at our centre.[12] In the current study, 77.3% patients showed improvement. In a study done by Aoki,[18] comparing TP shunt and VP shunts, incidence of shunt infection and malfunction was lower in TP shunt. Shunt revision rates of 11%–50% have been reported in various case series.[17] Sarkari et al[12] reported VP shunt infection and revision rates of 12.8% and 18.4%, and respectively in PTH. In our study, rates of malfunction and revision were slightly higher (22.7%, 5 cases). Blunt trauma to the abdomen and peritoneal adhesion blocking the distal end may be the cause in 4 patients. TP Shunt infection rates are described from 1% to 9% in different studies.[17] However most of these studies have only few patients with PTH.[1,2,19–21] Mortality rate (13.6%) in our studied compared favourably with Sarkari et al (15.7%).[12]

Presence of tracheostomy may increase shunt infection rates. Scalp bed sores/flap infections are common issues in obtunded polytrauma patients and combined with altered ventricular anatomy, risk of malposition of ventricular catheter and proximal end infections are increased. In our study 91.9% patients were tracheostomised and 25% had a positive microbial culture report. 6 (27.3%) patients had scalp sore/flap infection and 3 (13.6%) had extensive skin injuries involving neck/chest. Despite all these injuries no shunt infection was observed in patients who underwent TP shunt in our study.

Over drainage of TP shunt manifesting as sinking bone flap/syndrome of trephine, acquired ACM and rarely SDH has been previously reported. However, the incidence of over drainage is markedly less as compared to VP shunt and is rates vary from 1 to 15%.[17] In our study no over drainage complication was noted.

PTH is a challenging entity to treat in TBI patients. Although the sample size is small, this study shows that TP shunt is an attractive alternative for managing patients of communicating PTH, especially in presence of multiple risk factors such as scalp bed sores and/or tracheostomy.

5. Conclusions

In our study we have found that PTH in tracheostomised, polytrauma patients can be effectively managed with TP shunt. Our study shows that TP shunt is a safe, minimally morbid procedure which should be considered as an attractive alternative to VP shunt in patients with PTH.

Conflicts of interest

All authors have none to declare.

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