CSF Lumbar Drainage: A Safe Surgical Option in Refractory Intracranial Hypertension Associated with Acute Posttraumatic External Hydrocephalus

R. Manet, E. A. Schmidt, F. Vassal, D. Charier, and L. Gergelé

Abstract *Introduction*: External lumbar drainage (ELD) of cerebrospinal fluid (CSF) in posttraumatic refractory intracranial hypertension (ICHT) is controversial. We report our experience of ELD in ICHT associated with acute disturbance of CSF flow within subarachnoid spaces (SASs). *Materials and Methods*: Four adult patients admitted to the neurointensive care unit for severe TBI who presented with secondary ICHT are retrospectively reported. When refractory to second-tier therapy, if external ventricular drainage were not possible or failed, and in the absence of an indication for craniotomy to treat a mass lesion or decompressive craniectomy, we assessed the evolution of CSF volume within cranial SAS and checked the presence of basal cisterns and the absence of tonsillar herniation to evaluate interest in and the safety of ELD. *Results*: As second-tier therapy failed to lower intracranial pressure (ICP; mean ICP 37 ± 5 mmHg), and computed tomography (CT) showed abnormally enlarged cranial SAS following traumatic subarachnoid hemorrhage, patients received ELD. ICP decreased, with immediate and long-term effect (mean ICP 5 mmHg ± 2 mmHg). There were no complications to report. *Discussion*: Acute traumatic external hydrocephalus may explain some of the specific situations of secondary increased ICP, with a "normal" CT scan, that is refractory to medical treatment. In these situations, lumbar drainage should be considered to be a safe, minimally invasive, and effective surgical option.

Keywords Traumatic brain injury • Head trauma • Traumatic subarachnoid hemorrhage • Intracranial pressure • Intracranial hypertension • Second-tier therapy • External lumbar drainage • External hydrocephalus • Posttraumatic hydrocephalus

Introduction

Management of traumatic brain injuries (TBIs) remains a major neurocritical care (NCC) issue. In particular, maintaining appropriate cerebral perfusion pressure (CPP) to guarantee a steady cerebral blood flow (CBF) can be challenging when intracranial pressure (ICP) rises.

Our attention has recently been focused on a few cases of a secondary increase in ICP that becomes refractory to standard NCC, several days after the initial TBI and contrasting with a paradoxical "normal" cranial computed tomography (CT) scan (with no brain edema, no mass lesion, and visible sulci and basal cisterns). These observations are in accordance with other studies describing the limits of cranial CT to evaluate ICP level [1–3]. Thus, in the four reported patients, "normal" head CT scan was associated with severe refractory intracranial hypertension (ICHT).

For decades, lumbar puncture has been strictly contraindicated in situations of increased ICP. However, the data supporting this concept are quite old [4, 5] (even if a few other old data report safe indications [6]). The first use of lumbar drainage to treat ICHT was published in the 1990s [7]. During the past decade, large series were published, confirming the safety of this option for treating different situations involving ICHT [8–15].

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We report our experience of the use of external lumbar drainage (ELD) of cerebrospinal fluid (CSF) to treat some of the precisely selected patients in whom we suspected acute disturbance of CSF flow within the subarachnoid spaces (SASs) as a cause of secondary refractory ICHT.

Materials and Methods

We retrospectively analyzed four adult cases (two women, two men, mean age 53.5 ± 7 years) admitted to our NNC unit for TBI between November 2010 and September 2013, with a mean initial Glasgow Coma Scale (GCS) score of 10 (±1). All patients presented delayed (mean delay 8 ± 3 days) ICHT, which worsened their level of consciousness and required them to be sedated. They received continuous ICP monitoring by means of an intraparenchymal probe (Codman; Johnson & Johnson, MA, USA) and benefited from first-tier therapy when ICP increased above 20 mmHg and/or to maintain adequate CPP; above 60 mmHg, according to BTF/AANS 2007 guidelines [16], and adjusted by regular dynamic autoregulation assessments). If this first line of treatment failed, patients were scanned to rule out any indication for craniotomy to enable mass lesion evacuation, for decompressive craniectomy (consensual discussion between the neurosurgical and NCC teams), and to reevaluate the possibility of external ventricular drainage (EVD). Then, they received a burst suppression barbiturate coma and mild hypothermia (33–35 °C) as second-tier measures. When this maximal conservative treatment failed, patients were scanned again to reevaluate standard surgical options. In the absence (or failure) of these indications, we paid special attention to the presence of SAH on initial cranial CT and to the evolution of SAS volume between the admission and the last cranial CT. We also checked radiological conditions previously described by Münch et al. [11]: the presence of basal cisterns and the absence of tonsillar herniation. Afterward, if these opportune conditions were met, and if CSF volume within the SAS had paradoxically increased in that context of ICHT, a tunneled lumbar drain (Codman) was introduced by a neurosurgeon through a Tuohy needle into the SAS at the L4–L5 or L5–S1 level, at the bedside in the NCC unit, after acute osmotherapy, in the lateral/supine position (to limit pressure gradients between the cranial and spinal SAS to avoid the risk of downward herniation). A careful initial CSF withdrawal was achieved at a slow rate (mL by mL), in the presence of the attending neurosurgeon and intensivist, with a continuous papillary examination. When ICP and/or CPP reached an adequate level, the patient was repositioned with head elevation and the sterile collecting system of CSF drainage was fixed 20 cm above the tragus to maintain safe, continuous CSF drainage. Lumbar CSF output and pressure were monitored every hour to avoid the risk of overdrainage and pressure gradients.

Results

In all cases, the maximal medical intensive therapy failed to lower ICP (mean ICP 37 ± 5 mmHg), initial cranial CT showed traumatic SAH and the last CT scan showed no mass lesion, small ventricles, abnormally enlarged cranial SAS, visible basal cisterns, and no tonsillar herniation (Fig. 1). In 1 patient EVD insertion failed; in the 3 others, no EVD placement was achieved because of the small ventricle size.

The 4 patients received ELD. This procedure resulted in the immediate and long-lasting control of ICP (mean ICP 5 ± 2 mmHg; Fig. 2). None of the patients presented any other episodes of uncontrolled ICHT during their stay in the NCC unit. The need for sedation and other medical measures to lower ICP dropped dramatically, immediately after the drainage. After a short period of steady low ICP/adequate CPP, a weaning trial was achieved and the lumbar drain was removed. None of the 4 patients received a permanent CSF shunt. We had no complications to report; in particular, no pupillary changes, no subdural bleeding, no infection, and no occlusion of the catheter. Early outcome at ICU discharge was favorable in the 4 cases (mean modified Rankin Scale [mRS] = 2 ± 1).

Discussion

Therapeutic Strategies in Posttraumatic Refractory Raised ICP

First-tier treatment of traumatic raised ICP can be considered to be consensual [16]. However, the management of ICHTs that are refractory to these initial measures remains controversial. Thus, physicians no longer have to choose between uncertain solutions.

The efficiency and safety of medical solutions (in particular the use of barbiturate coma and mild therapeutic hypothermia) are regularly discussed. Similarly, except for the EVD and the evacuation of mass lesions, the surgical options also remain uncertain. The place of decompressive craniectomy in the management of traumatic ICHT is highly controversial, since the conclusions of the only two published randomized prospective trials ([17, 18] are still extensively discussed [19–21]. In any case, it is important to highlight the very high morbidity/mortality rate in these situations of uncontrolled ICP, despite second-tier measures.

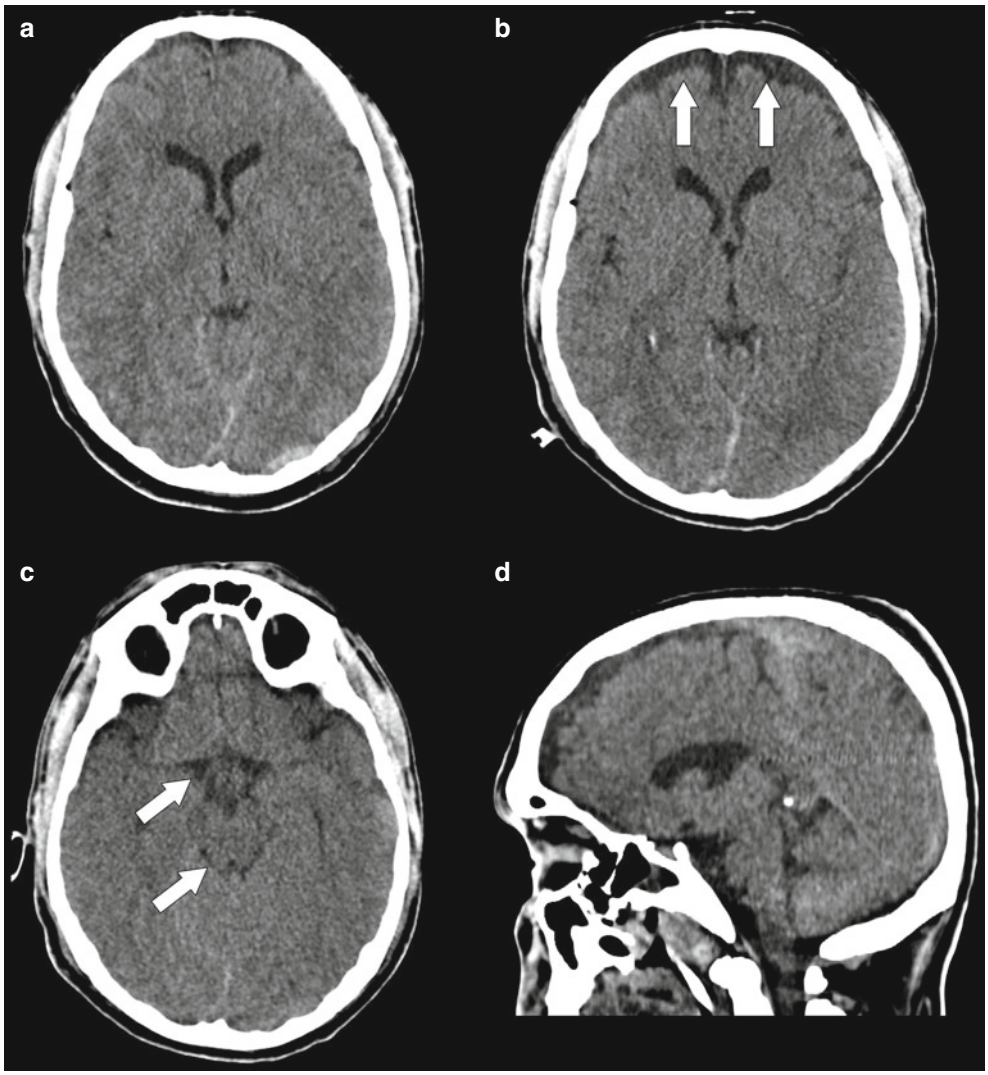

Fig. 1 Example of a cranial CT scan showing an abnormal accumulation of cerebrospinal fluid (CSF) within the subarachnoid spaces (SAS; presumed acute posttraumatic external hydrocephalus) simultaneous with a secondary rise in intracranial pressure (ICP). (**a**) At the time of the admission cranial CT scan, ICP was presumed to be relatively low according to clinical (awake patients) and transcranial Doppler findings. (**b**) The last CT scan before lumbar drainage, performed when a rise in ICP (mean ICP 37 ± 5 mmHg) became refractory to first- and second-tier therapies shows an abnormal accumulation of CSF within the SAS (*white arrows*). The examination also shows small ventricles, predicting difficulties and limited efficiency of extraventricular drainage (EVD) insertion. (**c**) Presence of basal cisterns (*white arrows*) and (**d**) the absence of tonsillar herniation (*white arrows*)

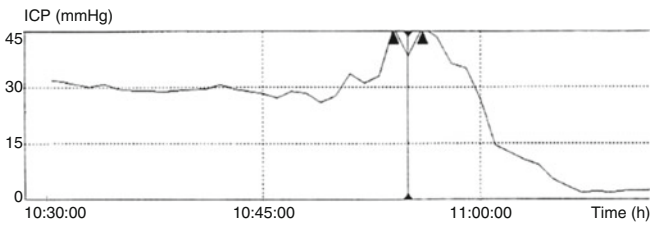

Fig. 2 Example of ICP monitoring during placement of an external lumbar drain. Careful initial CSF withdrawal was achieved at a slow rate (mL by mL), in the presence of the attending neurosurgeon and intensivist, with a continuous papillary examination. When ICP and/or cerebral perfusion pressure (CPP) reached an adequate level, the patient was repositioned with head elevation and the sterile collecting system of CSF drainage was fixed 20 cm above the tragus to maintain safe continuous drainage. Lumbar CSF output and pressure were monitored every hour to avoid the risk of overdrainage and pressure gradients

Lumbar drainage in raised ICP is also highly controversial and has previously received strong criticism [22]. Most authors recommend the placement of an EVD before ELD. We strongly agree on this concept and, as far as possible, we use EVD first. But in the cases reported here, EVD insertion failed in 1 case and was not achieved in the 3 others because of the small ventricle size.

A few publications described CSF lumbar drainage without prior EVD, with encouraging results [23, 24]. Our report tends to confirm a possible safe use of lumbar drainage to treat ICHT without prior EVD. But, contrary to these authors, our decision to treat patients with ELD without prior EVD was first based on a presumed pathological mechanism of ICHT (described afterward) and not on every situation of raised ICP with "safe criteria" as described by Münch et al. [11] (the presence of basal cisterns and the absence of tonsillar herniation).

CSF Outflow Disturbance as a Cause of Secondary Raised ICP After TBI

Secondary ICHT may affect a significant proportion of patients with TBI. Bruce and colleagues [25] reported 28 % secondary ICHT among 49 head-injured patients. In an analysis of 201 TBI, Stocchetti et al. [26] described a highest mean ICP in 85 patients between days 3 and 4 and in 41 patients after day 4. These specific patterns of rising ICP were associated with worse outcomes.

Various pathophysiological elements have been previously described in these specific patterns of secondary raised ICP [25, 27]: severe cerebrovascular congestion (described as "hyperemic syndrome"), edema in relation to different mechanisms (delayed ischemic insult, hyponatremia, traumatic vasospasm), delayed traumatic hemorrhage, and hyperleukocytosis.

However, we did not found any data concerning acute or subacute CSF flow impairment following head trauma. Literature concerning posttraumatic hydrocephalus [28–34] is heterogeneous (in particular incidence of 0.7 to 45 % has been reported) and concern mostly patients with a late diagnosis (several weeks after the head trauma). Traumatic SAH is reported to be a major risk factor.

In our observation, a mild traumatic SAH was present on every initial CT scan. The paradoxical accumulation of CSF around the brain at the same time as a rise in ICP leads us to suspect acute impairment of CSF flow within the SAS. The immediate and long-lasting efficacy of ELD in controlling ICP tends to reinforce our hypothesis.

Conclusion

Overall, even if further data are needed to confirm the pathophysiological hypothesis, we assume that certain situations of secondary ICHT after TBI with traumatic SAH could be understood to be acute external hydrocephalus. In these very specific situations, lumbar drainage of CSF should be considered a safe and effective treatment for ICHT that is refractory to first- and second-tier medical measures, and is less invasive than other surgical options (in particular, decompressive craniectomy).

Conflict of Interest None.

References

1. Hirsch W, Beck R, Behrmann C, Schobess A, Spielmann RP (2000) Reliability of cranial CT versus intracerebral pressure measurement for the evaluation of generalised cerebral oedema in children. Pediatr Radiol 30(7):439–443
2. Eide PK (2003) The relationship between intracranial pressure and size of cerebral ventricles assessed by computed tomography. Acta Neurochir 145(3):171–179; discussion 179
3. Kouvarellis AJ, Rohlwink UK, Sood V, Van Breda D, Gowen MJ, Figaji AA (2011) The relationship between basal cisterns on CT and time-linked intracranial pressure in pediatric head injury. Childs Nerv Syst 27(7):1139–1144
4. Cushing H (1909) Some aspects of the pathological physiology of intracranial tumors. Boston Med Surg J 141:71–80
5. Hepburn H (1938) The risk of spinal puncture. Can Med Assoc J 39:449–450
6. Levinson A, Greengard J, Lifvendahl R (1926) Cerebrospinal fluid in the new-born. Am J Dis Child 32(2):208–218
7. Baldwin HZ, Rekate HL (1991–1992). Preliminary experience with controlled external lumbar drainage in diffuse pediatric head injury. Pediatr Neurosurg 17(3):115–120
8. Abulhasan YB, Al-Jehani H, Valiquette M-A, McManus A, Dolan-Cake M, Ayoub O et al (2013) Lumbar drainage for the treatment of severe bacterial meningitis. Neurocrit Care 19(2):199–205
9. Javouhey E, Richard N, Stamm D, Floret D (2008) Lumbar drainage as treatment of refractory intracranial hypertension in bacterial meningitis. Intensive Care Med 34(6):1166–1167
10. Manosuthi W, Sungkanuparph S, Chottanapund S, Tansuphaswadikul S, Chimsuntorn S, Limpanadusadee P (2008) Temporary external lumbar drainage for reducing elevated intracranial pressure in HIV-infected patients with cryptococcal meningitis. Int J STD AIDS 19(4):268–271
11. Münch EC, Bauhuf C, Horn P, Roth HR, Schmiedek P, Vajkoczy P (2001) Therapy of malignant intracranial hypertension by controlled lumbar cerebrospinal fluid drainage. Crit Care Med 29(5):976–981
12. Murad A, Ghostine S, Colohan ART (2008) Controlled lumbar drainage in medically refractory increased intracranial pressure. A safe and effective treatment. Acta Neurochir Suppl 102:89–91
13. Murad A, Ghostine S, Colohan ART (2011) Role of controlled lumbar CSF drainage for ICP control in aneurysmal SAH. Acta Neurochir Suppl 110(Pt 2):183–187
14. Murad A, Ghostine S, Colohan ART (2012) A case for further investigating the use of controlled lumbar cerebrospinal fluid drainage for the control of intracranial pressure. World Neurosurg 77(1):160–165
15. Tuettenberg J, Czabanka M, Horn P, Woitzik J, Barth M, Thomé C et al (2009) Clinical evaluation of the safety and efficacy of lumbar cerebrospinal fluid drainage for the treatment of refractory increased intracranial pressure. J Neurosurg 110(6):1200–1208
16. Brain Trauma Foundation and American Association of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury, 3rd edn. [BTF Web site]. Available at: https://www.braintrauma.org/pdf/protected/Guidelines_Management_2007w_bookmarks.pdf. Accessed 6 February 2014
17. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, Klug G, Wallace D, Henning R, Tibballs J (2001) A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Childs Nerv Syst 17(3):154–162
18. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, Kossmann T, Ponsford J, Seppelt I, Reilly P, Wolfe R, DECRA Trial Investigators, Australian and New Zealand Intensive Care Society Clinical Trials Group (2011) Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 364(16):1493–1502
19. Sahuquillo J, Martínez-Ricarte F, Poca MA (2013) Decompressive craniectomy in traumatic brain injury after the DECRA trial. Where do we stand? Curr Opin Crit Care 19(2):101–106
20. Walcott BP, Kahle KT, Simard JM (2013) The DECRA trial and decompressive craniectomy in diffuse traumatic brain injury: is decompression really ineffective? World Neurosurg 79(1):80–81

21. Honeybul S, Ho KM, Lind CR (2013) What can be learned from the DECRA study. World Neurosurg 79(1):159–161
22. Grady MS (2009) Lumbar drainage for increased intracranial pressure. J Neurosurg 110:1198–1199
23. Abadal-Centellas JM, Llompart-Pou JA, Homar-Ramírez J, Pérez-Bárcena J, Rosselló-Ferrer A, Ibáñez-Juvé J (2007) Neurologic outcome of posttraumatic refractory intracranial hypertension treated with external lumbar drainage. J Trauma 62(2):282–286; discussion 286
24. Llompart-Pou JA, Abadal JM, Pérez-Bárcena J, Molina M, Brell M, Ibáñez J (2011) Long-term follow-up of patients with posttraumatic refractory high intracranial pressure treated with lumbar drainage. Anaesth Intensive Care 39(1):79–83
25. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzel B (1981) Diffuse cerebral swelling following head injuries in children: the syndrome of malignant brain edema. J Neurosurg 54:170–178
26. Stocchetti N, Colombo A, Ortolano F, Videtta W, Marchesi R, Longhi L (2007) Time course of intracranial hypertension after traumatic brain injury. J Neurotrauma 24(8):1339–1346
27. Unterberg A, Kiening K, Schmiedek P, Lanksch W (1993) Long-term observations of intracranial pressure after severe head injury. The phenomenon of secondary rise of intracranial pressure. Neurosurgery 32(1):17–23; discussion 23–24
28. Dandy WE, Blackfan KD (1914) Internal hydrocephalus: an experimental, clinical, and pathological study. Am J Dis Child 8:406
29. Cardoso ER, Galbraith S (1985) Posttraumatic hydrocephalus – a retrospective review. Surg Neurol 23:261–264
30. Groswasser Z, Cohen M, Reider-Groswasser I, Stern MJ (1988) Incidence, CT findings and rehabilitation outcome of patients with communicative hydrocephalus following severe head injury. Brain Inj 2(4):267–272
31. Kammersgaard LP, Linnemann M, Tibæk M (2013) Hydrocephalus following severe traumatic brain injury in adults. Incidence, timing, and clinical predictors during rehabilitation. Neurorehabilitation 33(3):473–480
32. Katz RT, Brander V, Sahgal V (1989) Updates on the diagnosis and management of posttraumatic hydrocephalus. Am J Phys Med Rehabil 68(2):91–96
33. Tian H-L, Xu T, Hu J, Cui Y, Chen H, Zhou L-F (2008) Risk factors related to hydrocephalus after traumatic subarachnoid hemorrhage. Surg Neurol 69(3):241–246
34. Mazzini L, Campini R, Angelino E, Rognone F, Pastore I, Oliveri G (2003) Posttraumatic hydrocephalus: a clinical, neuroradiologic, and neuropsychologic assessment of long-term outcome. Arch Phys Med Rehabil 84:1637–1641