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External hydrocephalus in adult neurocritical patients

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Abstract

Background: External hydrocephalus (EH) in adults is an underrecognized clinical entity characterized by CSF circulatory failure and progressive subarachnoid space (SAS) dilation under high intracranial pressure (ICP). Data regarding its incidence, pathophysiology, and management in adult neurocritical care are sparse. Objective: to describe the incidence, risk factors and outcome of EH in adult neurocritical patients.

Methods: prospective study of patients with moderate-to-severe traumatic brain injury (msTBI) or spontaneous subarachnoid hemorrhage (SAH) admitted to intensive care unit (ICU) from January 2024 to June 2025. EH was diagnosed when SAS enlargement was found between two successive cranial CT scans, in the context of intracranial hypertension.

Results: A total of 128 patients were included (78 msTBI, 50 SAH). The overall incidence of EH was 7.8% (10.3% in msTBI vs. 4.0% in SAH). The median time to EH onset was 4.5 days (IQR 4.0–16.5). In the general population, EH development was significantly associated with younger age ($p=0.013$), refractory intracranial hypertension ($p<0.001$), surgical intervention ($p=0.018$), and decompressive craniectomy ($p=0.007$). External hydrocephalus did not resolve spontaneously in any of the cases, requiring treatment with external lumbar drainage in 70%. Patients with EH demonstrated significantly prolonged ICU lengths of stay (34.0 vs. 9.0 days; $p<0.001$) and hospital lengths of stay (46.5 vs. 17.5 days; $p<0.001$).

Conclusions: the first prospective study on EH adult neurocritical patients is presented. EH presents as uncommon but critical and complex driver of secondary neurological deterioration and prolonged hospital resource utilization in adult neurocritical care. Diagnosis requires a high index of suspicion based on clinical and radiological findings. It is associated with refractory intracranial hypertension, generally requiring an external lumbar drainage.

Introduction

External hydrocephalus (EH) is a distinct pathophysiological entity characterized by an alteration in cerebrospinal fluid (CSF) circulatory dynamics, leading to a progressive accumulation of CSF and subsequent expansion of the cortical subarachnoid space (SAS) of the convexity under conditions of elevated intracranial pressure (ICP). While this condition is widely documented and understood within the pediatric population, literature evaluating EH in adult patients remains scarce, and its clinical incidence is likely underestimated in contemporary intensive care settings [1, 2, 3, 4]. In highly prevalent neurocritical care pathologies, such as moderate-to-severe traumatic brain injury (msTBI) and spontaneous subarachnoid hemorrhage (SAH), managing intracranial hypertension (IH) presents a therapeutic challenge. Within this clinical environment, subtle disruptions in CSF flow dynamics frequently escape early detection. Specifically, EH can manifest insidiously delayed and refractory IH accompanied by highly subtle, non-specific tomographic findings. This lack of clear diagnostic signs

hinders timely clinical recognition and has direct, high-stakes implications for targeted therapeutic management [2, 3, 5].

Given the scarcity of available international evidence and the complete absence of local data, it is imperative to evaluate the epidemiological and clinical landscape of EH within our healthcare setting. The aim of this study was to evaluate the incidence of EH in adult neurocritical patients, identifying specific clinical and surgical factors associated with its development, and characterizing its outcome.

Methods

We conducted a prospective, single-center study encompassing all adult neurocritical patients admitted to the intensive care unit (ICU) at Hospital Maciel (Montevideo, Uruguay), from January 2024 to June 2025. The study was approved by the Ethics Committee of Hospital Maciel.

Inclusion criteria consisted of: patients requiring ICU admission, aged 18 or older, diagnosis of msTBI (GCS on admission between 3 and 13) or spontaneous SAH, and at least three cranial CT scans within first 21 days [4]. Patients were excluded if they were < 18 years of age or presented with a primary diagnosis of chronic subdural hematoma.

An individual SAS assessment was performed on each CT scan by two independent investigators (PG and EL). Both investigators had to agree for validate diagnostic consensus. EH was diagnosed when SAS was assessed as being dilated or increased between two successive CT scans, in the context of intracranial hypertension (invasive sustained ICP elevation greater than 20 mmHg over 5 minutes, or two of the following non-invasive neuromonitoring measurements consistent with intracranial hypertension: optic nerve sheath diameter > 6 mm, transcranial Doppler with a pulsatility index > 1.3 or diastolic flow velocity < 20 cm/s, or infrared pupillometry with an NPi <3) [4, 6].

There are no pathognomonic neuroimaging signs of acute EH. Both CT and magnetic resonance imaging (MRI) can be used, although from a practical point of view, CT scan is more readily available for neurocritical patients. Different signs have been associated with acute EH, including: wider cortical sulci, greater width of the Sylvian fissure, patent basal cisterns, cortical vein sign (visualization of cortical veins crossing the widened subarachnoid space, an important feature distinguishing EH from subdural hygroma, in which veins are displaced rather than crossing the collection). The key observation in the diagnosis of EH is the progressive enlargement of the SAS, paradoxical in a context of raised ICP [3]. Figure 1 shows an example of external EH in neuroimaging and a diagram illustrating the differences between EH and subdural hygroma.

Refractory intracranial hypertension was defined as ICP remaining above 25 mmHg despite first-tier measures (ventricular CSF drainage, osmotherapy, moderate hyperventilation, as well as sedation and neuromuscular blockade).

The variables recorded were age, sex, Simplified Acute Physiologic Score 3 (SAPS 3) and GCS score on admission, Hunt and Hess and modified Fisher scale (SAH), Marshall CT classification, ICP monitoring, presence of ventriculomegaly, need for neurosurgery or decompressive craniectomy, measures used for the management of intracranial hypertension, ICU and hospital length of stay, and Glasgow Outcome Scale Extended (GOSE) and mortality at discharge from the ICU and hospital.

Statistical Analysis

Nominal variables are presented as absolute frequencies or percentages. Continuous variables are presented as medians with interquartile ranges (IQR, 25-75) because most data did not present a normal distribution. Comparative analyses between patients who developed EH and those who did not were performed utilizing Fisher's exact test for categorical variables. The Mann-Whitney U test was deployed for continuous variables. In all cases, $p < 0.05$ was considered significant. SPSS version 23.0 was used for the statistical analyses.

Results

A final cohort of 128 patients meeting all inclusion criteria was analyzed; 78 patients (60.9%) carried a diagnosis of msTBI, and 50 patients (39.1%) presented with SAH. The demographic and clinical characteristics of the global study population are shown in table 1.

The overall clinical incidence of EH across the entire cohort was 7.8%. The incidence was more than two-fold higher within the msTBI group compared to the SAH group (10.3% [8/78] vs. 4.0% [2/50], respectively) (Table 1).

The median time from initial neurological injury to the definitive diagnosis of EH was 4.5 days (IQR 4.0–16.5). Spontaneous resolution of the extra-axial fluid collections did not occur in any patient (0.0%). Active therapeutic intervention via external lumbar drainage (ELD) was required in 70.0% of the affected individuals. The raw mortality rate within the EH subgroup was 30.0% (n=3), with a neuro-specific death driving the clinical outcome in two-thirds of those cases.

When comparing patients who developed EH against those who did not, several physiological, surgical, and resource-utilization differences emerged (Table 2). Patients developing EH were significantly younger than their non-EH counterparts (median 27.5 vs. 44.0 years; p=0.013). Patients with EH had a significantly higher incidence of refractory intracranial hypertension compared to the group without HE. (80 % vs 12.7 %, p<0.001). Furthermore, patients who developed EH exhibited a significantly higher requirement for neurosurgical procedures (60.0% vs. 22.9%; p=0.018) and, specifically, decompressive craniectomy (DC) (50.0% vs. 11.9%; p=0.007). The median ICU length of stay for EH patients was nearly four times longer than for non-EH patients (34.0 vs. 9.0 days; p<0.001), and their total hospital stay was similarly prolonged (46.5 vs. 17.5 days; p<0.001).

Within the isolated msTBI, statistically significant factors associated with development of EH included: a clinical course marked by refractory ICH (75.0% vs. 10.0%; p<0.001), neurosurgical intervention (62.5% vs. 22.9%; p=0.029), and the performance of a decompressive craniectomy (50.0% vs. 14.3%; p=0.031). Both ICU stay (34.0 vs. 7.5 days; p<0.001) and overall hospital stay (51.0 vs. 17.5 days; p<0.001) were extensively prolonged in the EH group (Table 3).

Within the spontaneous SAH sub-cohort (n=50), only 2 patients developed EH (4.0%). In this subgroup, patients developing EH were significantly younger (median 30.5 vs. 58.0 years; p=0.031), experienced significantly higher rates of refractory IH (100.0% vs. 16.7%; p=0.037), and required therapeutic external lumbar drainage (ELD) placement more frequently (50.0% vs. 0.0%; p=0.040) (Table 4).

Discussion

To our knowledge, this study represents the first prospective study on adult external hydrocephalus within the neurocritical care literature [4]. Our findings demonstrate that EH is a clinical reality in adult neurocritical patients, occurring in 7.8% of TBI and SAH patients. Crucially, the incidence reaches 10.3% among patients presenting with msTBI, confirming that extra-axial CSF accumulation is a substantial and frequently unrecognized driver of secondary neurological progression in this group of patients [2, 4]. Data on patients with aneurysmal SAH are even more scarce. In their retrospective study, Yoshimoto et al. detected the presence of subdural fluid collections in 38% of cases, although 81% of these patients presented with concomitant ventriculomegaly [5].

The historical trajectory of EH highlights why it remains underdiagnosed in adult populations. For decades following Walter Dandy's foundational 1946 description, EH was considered a pediatric condition [7]. It was not until the landmark reports by Cardoso and Schubert in 1996 that the condition was formally identified in adults, a classification subsequently reinforced by the characterizations of Manet et al. and Gergelé et al [2, 4, 8]. In the only published review of EH in adults, 15 studies were identified, mainly retrospective and case series, with a limited methodological quality [3].

Our observed incidence of 10.3% in msTBI, while lower than the 30.4% reported by Gergelé et al. in the largest patient series published to date, underlines a critical clinical truth: a significant portion of patients suffering severe intracranial injury develop extra-axial fluid collections that are frequently misclassified or overlooked [3, 4]. This discrepancy in absolute incidence could be due to variations in neuromonitoring and imaging protocols across the different institutions. In this regard, it is important to note that the study by Gergele et al. included only patients with TBI and invasive ICP monitoring [4].

An interesting finding of our study is the statistically significant association between EH development and younger age (p=0.013 in the global cohort). This finding contrasts with the report by Gergelé et al., who described a trend toward older age in the group with EH [4] . The difference could be explained by the composition of our cohort, which included a significant proportion of young patients with msTBI, the group that accounted for the majority of EH cases. On the other hand, this unequal age distribution could

be explained by the fact that younger adults possess minimal to no pre-existing cerebral atrophy, resulting in a highly tight, non-compliant intracranial compartment with minimal intrinsic tolerance for extra-axial fluid accumulation. Conversely, cerebral atrophy in older adults could provide a pressure-free structural buffer that allows for the expansion of the extracerebral space without resulting in mechanical tissue distortion or intracranial hypertension. This could also explain the greater recognition of EH in the pediatric population [1].

Pathophysiologically, the evolution of acute EH in adults relies on three primary factors: intracranial hemorrhage, arachnoid membrane tearing, and a subsequent mismatch between CSF synthesis and arachnoid granulation clearance [3, 5, 9]. In both severe neurotrauma and aneurysmal rupture, the deposition of cellular debris, erythrocytes, and fibrin breakdown products leads to mechanical clogging of the arachnoid villi. This downstream obstruction reduces overall CSF outflow conductance, triggering upstream accumulation within the cortical SAS. Simultaneously, mechanical structural tears in the arachnoid tract—inflicted either by the primary kinetic impact or subsequent surgical corridors— may act as a valve device leading to the extra-axial trapping of CSF, a mechanism it shares with the subdural hygroma according to some authors [1, 10, 11, 12, 13, 14]. Rekate et al has described that an increase in SAS thickness of more than 2 mm is associated with a volume increase of approximately 100 cc, a fact that explains the rise in ICP in patients with EH [15].

Our data also reveal a significant association between surgical manipulation, specifically decompressive craniectomy, and EH development. Decompressive craniectomy alters craniospinal pressure gradients and disrupts normal CSF dynamics, and may predispose to redistribution of CSF toward the cortical subarachnoid space. When combined with impaired arachnoid villi absorption, this alteration of the biomechanical profile accelerates the progressive accumulation of CSF in the subarachnoid space, predisposing to or exacerbating external hydrocephalus [3, 14].

Differentiating EH from subdural hygroma remains a central diagnostic hurdle in neurocritical patients [3, 4, 11, 16]. While both conditions present as hypodense extra-axial fluid collections on plain CT scans, they differ fundamentally in mechanical location and pressure profiles: EH represents a pathological volume expansion within the anatomically preserved subarachnoid space, whereas hygromas occupy a pathologically created subdural cleavage plane. Crucially, true EH is defined by the presence of elevated ICP, whereas simple subdural hygromas characteristically manifest with normal or low baseline pressures. Our data strongly reflect this clinical distinction: 80 % of patients who developed EH required intensive management for refractory IH ($p<0.001$), emphasizing that EH is a hypertensive, space-occupying process rather than a passive phenomenon. Magnetic resonance imaging could play an important role in the diagnosis of acute EH, as it allows for the identification of increased SAS volume, one of the most critical signs for EH diagnosing [17]. Recently, two different MRI sequences (Time-SLIP and SPACE) have shown utility in detecting acute EH by evaluating the spatial distribution and calculating the volume of intracranial CSF [18, 19].

A key finding of the present study is the significantly higher incidence of refractory IH in patients with EH compared to those who did not present EH (80 % vs 12.7 %, p < 0.001). This can be explained by the fact that the first- and second-tier measures commonly used to control ICP are ineffective in cases of EH. Patients with IH due to EH represent a subgroup for whom external lumbar drainage (ELD) may be a particularly effective measure [2, 3, 20, 21]. This response to ELD can be explained by the fact that CSF drains directly from the cortical SAS via the spinal SAS, which has a larger volume than the ventricular space [20, 22]. Furthermore, according to Mayras et al., the decrease in ICP following the removal of a small amount of CSF strongly suggests a diagnosis of acute EH, a scenario considered unlikely otherwise [23]. In our patient population, an ELD was placed in 70% of patients with HE, leading to ICP control in all cases.

While the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC) recommended avoiding lumbar drains due to theoretical risks of transtentorial herniation, these guidelines do not account for the specific subgroup of patients with refractory intracranial hypertension presenting with acute EH [24, 25]. Along these same lines, the recent French guidelines on the neurosurgical management of TBI recommend to treat EH with ELD [26].

Finally, the impact of EH on resource utilization is profound. Patients developing EH experienced highly prolonged ICU (34.0 vs. 9.0 days) and hospital stays (46.5 vs. 17.5 days), which is consistent with the findings of Gergele et al. [4]. This massive extension of critical care requirements highlights EH as a major driver of post-injury morbidity and healthcare costs. Interestingly, despite these prolonged courses, all-cause mortality and 6-month functional outcomes (GOSE 1–4) did not significantly differ between groups. This coluld indicate that, while EH delays recovery and complicates the acute ICU course, invasive targeted treatments—such as ELD—can mitigate the risk of permanent neurological damage, matching the long-term outcomes of patients with less complex recovery trajectories.

This study has limitations. First, its unicenter design. Second, the small sample size within the EH subgroup, particularly the SAH arm. These factors limit the ability to perform robust multivariate regression analysis to isolate independent predictors. Third, the diagnosis of intracranial hypertension was made invasively in 80% of our patients with EH, while detection was performed non-invasively in the remainder. Although invasive ICP measurement is the gold standard, in cases where this is not available, noninvasive neuromonitoring tools can be used to estimate ICP, such as transorbital ultrasonographic measurement of the optic nerve sheath diameter, transcranial doppler ultrasonography, and ICP waveform pulse morphology monitoring through cranial cavity micro-expansions [6, 15, 27].

Conclusions

The first prospective study on EH in adult neurocritical care patients is presented. EH is a critical and complex driver of secondary neurological deterioration and prolonged hospital resource utilization in this patient population. Early recognition based on a high index of clinical suspicion and serial neuroimaging is key. External lumbar drainage appears to be an effective measure in the management of refractory IH

due to EH. Prospective, multi-center studies are needed to better define standardized screening and management algorithms for adult EH.

Declarations

The manuscript complies with all instructions to authors.

Author contributions:

- Pedro Grille: conception, design, analysis of data, drafting, reviewing, final approval.

- Emiliano Lecor: data collection, analysis of data, drafting, reviewing.

- Pablo Bouzas: analysis of data, reviewing, final approval

The authorship requirements have been met and the final manuscript was approved by all authors.

This manuscript has not been published elsewhere and is not under consideration by another journal

The authors confirm adherence to ethical guidelines and was approved by Ethics Committee of Hospital Maciel, ASSE, Montevideo, Uruguay.

Informed consent was obtained from all individual participants, or surrogates, included in the study

The authors have no conflicts of interest.

Artificial Intelligence statement: No artificial intelligence was used in this manuscript.

A reporting checklist (STROBE Statement) was used.

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Tables

Table 1. Baseline Clinical and Demographic Characteristics of the General Population

Variable	Result (n=128)
Male sex, n (%)	84 (65.6%)
Age (years)	44.0 (26.8 – 56.2)
SAPS 3 score	46.0 (37.8 – 55.0)
Primary diagnosis: • mstbi • spontaneous SAH	 78 (60.9%) 50 (39.1%)
Global incidence of EH	10 (7.8%)
• msTBI cohort (n=78)	8 / 78 (10.3%)
• spontaneous SAH cohort (n=50)	2 / 50 (4.0%)
Admission GCS score	8.0 (6.0 – 12.2)
ICU length of stay (days)	11.0 (5.0 – 19.0)
Hospital length of stay	19.0 (8.0 – 33.0)
6-month GOSE score	6.0 (1.0 – 7.0)
Mortality	36 (28.1%)

GCS: Glasgow Coma Scale; ICU: intensive care unit; msTBI: moderate to severe traumatic brain injury; SAH: subarachnoid hemorrhage; SAPS: Simplified Acute Physiology Score.

Table 2. Comparative Matrix Between Patients With and Without External Hydrocephalus in the global population.

Variable	With EH (n=10)	Without EH (n=118)	p-value
Male sex	9 (90.0%)	75 (63.6%)	0.163
Age (years)	27.5 (20.5 – 40.5)	44.0 (27.0 – 57.8)	0.013
SAPS 3 score	53.0 (45.8 – 60.2)	45.0 (37.2 – 54.5)	0.257
Admission GCS	7.0 (3.5 – 8.0)	8.0 (6.0 – 12.8)	0.200
Invasive ICP monitoring	8 (80.0%)	23 (19.5%)	< 0.001
Refractory IH	8 (80.0%)	15 (12.7%)	< 0.001
Neurosurgery	6 (60.0%)	27 (22.9%)	0.018
Decompressive craniectomy	5 (50.0%)	14 (11.9%)	0.007
Ventriculomegalia	1 (10.0%)	17 (14.4%)	1.000
ICU length of stay (days)	34.0 (20.8 – 41.5)	9.0 (5.0 – 17.0)	< 0.001
Hospital length of stay (days)	46.5 (33.2 – 53.0)	17.5 (7.2 – 29.0)	< 0.001
Unfavorable outcome (GOSE 1–4)	4 (40.0%)	45 (38.1%)	1.000
Mortality	3 (30.0%)	33 (28.0%)	1.000

GCS: Glasgow Coma Scale; GOSE: Glasgow Outcome Scale Extended; ICU: intensive care unit; IH: intracranial hypertension; msTBI: moderate to severe traumatic brain injury; SAH: subarachnoid hemorrhage; SAPS: Simplified Acute Physiology Score.

Table 3. Comparative Subgroup Matrix for Moderate-to-Severe Traumatic Brain Injury Patients.

Variable	With EH (n=8)	Wthout EH (n=70)	p-value
Male sex	8 (100.0%)	56 (80.0%)	0.338
Age (years)	27.5 (21.5 – 37.8)	31.0 (23.0 – 45.8)	0.429
SAPS3 score	53.0 (49.2 – 59.2)	46.0 (40.2 – 54.5)	0.161
Admission GCS	5.5 (3.0 – 8.0)	8.0 (6.0 – 8.0)	0.089
Invasive ICP monitoring	7 (87.5%)	13 (18.6%)	< 0.001
Traumatic SAH	7 (87.5%)	43 (61.4%)	0.247
Marshall CT classification	4.0 (2.8 – 5.0)	2.0 (2.0 – 4.0)	0.072
Intracranial hypertension	8 (100.0%)	17 (24.3%)	< 0.001
Refractory IH	6 (75.0%)	7 (10.0%)	< 0.001
Ventriculomegalia	0 (0.0%)	2 (2.9%)	1.000
Neurosurgery	5 (62.5%)	16 (22.9%)	0.029
Decompressive craniectomy	4 (50.0%)	10 (14.3%)	0.031
ICU length of stay (days)	34.0 (19.5 – 42.2)	7.5 (4.0 – 16.0)	< 0.001
Hospital length of stay (days)	51.0 (35.8 – 54.2)	17.5 (7.0 – 27.0)	< 0.001
Unfavorable outcome (GOSE 1–4)	3 (37.5%)	22 (31.4%)	0.706
Mortality	2 (25.0%)	15 (21.4%)	1.000

GCS: Glasgow Coma Scale; GOSE: Glasgow Outcome Scale Extended; ICU: intensive care unit; IH: intracranial hypertension; msTBI: moderate to severe traumatic brain injury; SAH: subarachnoid hemorrhage; SAPS: Simplified Acute Physiology Score.

Table 4. Comparative Subgroup Matrix for Spontaneous Subarachnoid Hemorrhage Patients

Variable	With EH (n=2)	Without EH (n=48)	p-value
Male sex	1 (50.0%)	19 (39.6%)	1.000
Age (years)	30.5 (24.8 – 36.2)	58.0 (47.8 – 64.5)	0.031
SAPS 3 score	43.5 (34.8 – 52.2)	41.5 (34.0 – 53.5)	0.729
Invasive ICP monitoring	1 (50.0%)	10 (20.8%)	0.395
Hunt and Hess score	3.0 (2.5 – 3.5)	3.0 (2.0 – 4.0)	0.859
Modified Fisher scale score	3.0 (3.0 – 3.0)	4.0 (3.0 – 4.0)	0.185
Prophylactic ELD	1 (50.0%)	22 (45.8%)	1.000
Therapeutic ELD	1 (50.0%)	0 (0.0%)	0.040
Intracranial hypertension	2 (100.0%)	12 (25.0%)	0.074
Refractory IH	2 (100.0%)	8 (16.7%)	0.037
Ventriculomegalia	1 (50.0%)	15 (31.2%)	0.542
Neurosurgery	1 (50.0%)	11 (22.9%)	0.426
Decompressive craniectomy	1 (50.0%)	4 (8.3%)	0.192
ICU length of stay (days)	31.5 (27.2 – 35.8)	11.0 (7.0 – 19.5)	0.087
Hospital length of stay (days)	33.5 (28.2 – 38.8)	17.5 (8.0 – 31.2)	0.298
Unfavorable outcome (GOSE 1–4)	1 (50.0%)	23 (48.9%)	1.000
Mortality	1 (50.0%)	18 (37.5%)	1.000

ELD: external lumbar drainage; GCS: Glasgow Coma Scale; GOSE: Glasgow Outcome Scale Extended; ICU: intensive care unit; IH: intracranial hypertension; msTBI: moderate to severe traumatic brain injury; SAH: subarachnoid hemorrhage; SAPS: Simplified Acute Physiology Score.

Figures

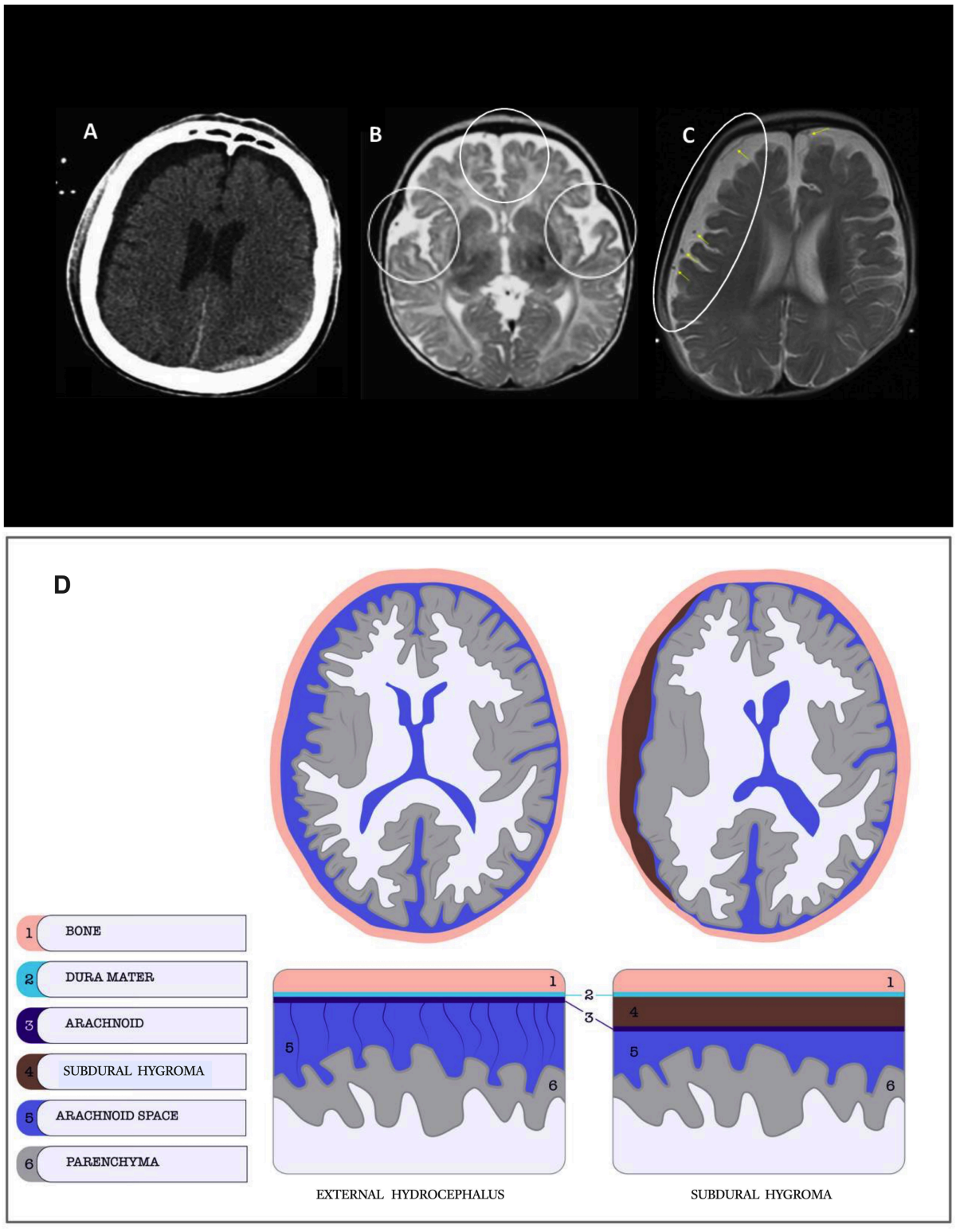


Figure 1

A: adult patient with moderate TBI and traumatic subarachnoid hemorrhage showing bilateral frontoparietal enlargement of the subarachnoid space with normal ventricular size, consistent with acute external hydrocephalus. Note the

cortical sulci widening. B: adult patient with aSAH following surgical clipping,

who developed postoperative external hydrocephalus. C: MRI shows enlargement of the interhemispheric and bilateral sylvian fissures (circles), with cortical vessels crossing the widened subarachnoid space (cortical vein sign).

D: diagram showing difference between external hydrocephalus and subdural hygroma. In external hydrocephalus, the cerebrospinal fluid (CSF) diffuses into the cortical subarachnoid space which becomes dilated. In subdural hygroma, the CSF collects in the subdural space which is virtual (with permission of Grille et al.) [3].