

## Outcomes and quality of life in severe traumatic brain injury: A retrospective Single-Center Study of 57 Cases at Hassan II University Hospital, Fez, Morocco

Hamza Didi \*, Abdelkarim Shimi, Mohamed Khatouf, Ali Derkaoui and Abderrahim El Bouazzaoui

*Department of Anesthesia and Intensive Care, Polyvalent ICU Unit A1, Hassan II University Hospital, Fez, Morocco.*

World Journal of Advanced Research and Reviews, 2026, 30(02), 761-769

Publication history: Received on 28 March 2026; revised on 06 May 2026; accepted on 08 May 2026

Article DOI: <https://doi.org/10.30574/wjarr.2026.30.2.1212>

### Abstract

**Background:** Severe traumatic brain injury (sTBI) is a leading cause of death and long-term disability worldwide, disproportionately affecting young adults in low- and middle-income countries (LMICs). Data on neurological outcomes and quality of life after sTBI from North Africa remain scarce. We describe the epidemiological profile, management modalities, in-hospital outcomes, and functional recovery up to 6 months in patients with sTBI admitted to the ICU of Hassan II University Hospital, Fez, Morocco.

**Methods:** A retrospective descriptive and analytical single-center study was conducted in the Polyvalent ICU Unit A1 of Hassan II University Hospital, Fez, Morocco, from March 2018 to May 2021. Patients aged  $\geq 16$  years admitted for isolated or polytraumatic sTBI (Glasgow Coma Scale [GCS]  $\leq 8$  and/or potentially evolving intracranial lesions) were included. Neurological outcome was assessed using the Glasgow Outcome Scale (GOS) at ICU discharge, 3 months, and 6 months. Univariate analysis of prognostic factors was performed using Student's t-test and Chi-square test (significance threshold:  $p < 0.05$ ) with SPSS version 21.

**Results:** Fifty-seven patients were enrolled. Mean age was 32.8 years (range 16–68); 94% were male (sex ratio 18:1). Road traffic accidents (RTAs) accounted for 64.9% of injuries. Mean admission GCS was 7.9 (range 3–14). Cerebral contusions (61.4%) and acute subdural hematomas (56.1%) were the predominant CT findings. Twenty-two patients (38.6%) underwent emergency neurosurgical intervention. In-hospital mortality was 49.1% ( $n=28$ ), primarily attributable to refractory intracranial hypertension (IH, 57%). Among survivors followed at 6 months ( $n=23$ ), 65.2% achieved a good functional recovery (GOS 5). Statistically significant prognostic factors for death were: low admission GCS ( $p=0.028$ ), hemodynamic instability ( $p=0.035$ ), intracranial hypertension ( $p=0.001$ ), subarachnoid hemorrhage (SAH,  $p=0.022$ ), and nosocomial infection ( $p=0.031$ ).

**Conclusion:** sTBI carries a 49.1% in-hospital mortality rate in this Moroccan ICU cohort, yet survivors demonstrate remarkable progressive neurological recovery, with 65.2% achieving good functional outcome at 6 months. Key therapeutic targets include aggressive prevention and management of secondary brain insults, intracranial hypertension, and nosocomial infections. Strengthening prehospital care systems and road safety policies represents the highest-priority preventive intervention in this context.

**Keywords:** Severe Traumatic Brain Injury; TBI; Glasgow Coma Scale; Glasgow Outcome Scale; Secondary Brain Injury; Prognostic Factors; Intensive Care Unit; Morocco

\* Corresponding author: Hamza Didi

## 1. Introduction

Severe traumatic brain injury (sTBI) is defined as a traumatic insult to the brain resulting in a Glasgow Coma Scale (GCS) score  $\leq 8$  and/or potentially evolving intracranial lesions [1]. It constitutes one of the leading causes of trauma-related mortality and is a major source of long-term physical, cognitive, behavioral, and psychosocial sequelae [4, 5]. In high-income countries, an estimated 155,000 TBI cases occur annually in France alone, resulting in approximately 8,000 deaths per year [2]. In low- and middle-income countries (LMICs), including Morocco, road traffic accidents (RTAs) represent the predominant mechanism and epidemiological data remain limited.

The pathophysiology of sTBI is driven not only by primary mechanical injury but critically by secondary cerebral insults — termed secondary brain injuries of systemic origin (SBISO) or Agressions Cérébrales Secondaires d'Origine Systémique (ACSOS) in French neurocritical literature — including hypoxia, hypotension, hyperthermia, and abnormal carbon dioxide levels, which aggravate primary lesions in the hours and days following trauma [2, 3]. Prevention and early correction of these secondary insults form the cornerstone of ICU management [3].

Neurological outcome assessment in sTBI relies on the Glasgow Outcome Scale (GOS), a validated five-grade instrument widely used to quantify functional recovery [6, 7]. Prognostic modeling, including the internationally validated IMPACT score, allows estimation of mortality and unfavorable outcome at 6 months based on clinical, radiological, and laboratory parameters [19, 22].

This study aimed to evaluate the outcomes and quality of life of sTBI patients managed in the ICU at Hassan II University Hospital (Fez, Morocco), and to identify clinical, neurological, and radiological determinants of functional prognosis at discharge, 3 months, and 6 months.

---

## 2. Methods

### 2.1. Study Design and Setting

We conducted a retrospective, descriptive, analytical, single-center study in the Polyvalent Intensive Care Unit A1 (ICU-A1) of Hassan II University Hospital, a 1,500-bed academic tertiary referral center in Fez, Morocco. The study period extended from March 2018 to May 2021. This manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies.

### 2.2. Inclusion and Exclusion Criteria

We included all patients aged  $\geq 16$  years admitted to ICU-A1 with a diagnosis of isolated or polytraumatic sTBI (GCS  $\leq 8$  and/or evolving intracranial lesions on CT imaging). Exclusion criteria were: age  $< 16$  years; polytrauma without cranial involvement; and patients who died in the emergency department prior to ICU admission.

### 2.3. Outcome Definitions

The primary outcome was in-hospital all-cause mortality. Secondary outcomes included neurological functional recovery assessed by the GOS at ICU discharge, at 3 months, and at 6 months post-injury. GOS scoring was: 1 = death; 2 = persistent vegetative state; 3 = severe disability (conscious but dependent); 4 = moderate disability (independent but disabled); 5 = good recovery. Unfavorable outcome was defined as GOS 1–2; favorable outcome as GOS 3–5. Intracranial hypertension (IH) was defined clinically (anisocoria, Cushing reflex) and by transcranial Doppler criteria (pulsatility index  $> 1.2$  and diastolic velocity  $< 20$  cm/s in the middle cerebral artery), in the absence of direct intracranial pressure monitoring.

### 2.4. Data Collection

Data were extracted retrospectively from inpatient medical records, operative reports, imaging reports, biological results, and the institutional Hosix electronic database. Variables collected included: demographic data (age, sex, mechanism of injury, prehospital transport modality, time to ICU admission); clinical parameters at admission (GCS, pupillary status, hemodynamic and respiratory status, associated injuries); radiological findings (initial and follow-up brain CT, whole-body CT); therapeutic management; complications; prognostic scores (GOS, Marshall, Rotterdam, ISS, IMPACT); and outcomes (discharge, 3-month and 6-month GOS, cause of death).

## 2.5. Statistical Analysis

Data were entered and coded in Microsoft Excel 2019 and analyzed with SPSS version 21. Continuous variables are presented as means with standard deviations (SD). Categorical variables are reported as counts and percentages. Univariate analyses used Student's t-test for continuous variables and the Chi-square test for categorical variables. A p-value <0.05 was considered statistically significant. Multivariate analysis was precluded by the small sample size.

## 2.6. Ethical Considerations

This study was conducted in accordance with the principles of the Declaration of Helsinki. Patient anonymity and data confidentiality were maintained throughout. Data were de-identified prior to analysis. No formal ethics committee submission was required for this retrospective observational study under current national regulations. No funding sources are declared. The authors declare no conflicts of interest.

## 3. Results

### 3.1. Epidemiological Characteristics

Fifty-seven patients were enrolled over the 38-month study period. Mean age was 32.8 years (range 16–68); the 21–40-year age group accounted for 61.9% of patients (Table 1). Male sex predominated markedly, with 54 patients (94%) versus 3 females (6%), yielding a sex ratio of 18:1. RTAs were the leading mechanism (n=37, 64.9%), with motorcyclists constituting 43.2% of RTA cases, followed by pedestrians (37.8%) and car occupants (19%). Falls accounted for 21.1% and assaults for 12.3%.

Pre-existing comorbidities were present in only 6 patients (10.5%). Prehospital care was non-medicalized in 91% of cases (transport by civil protection or regional hospital ambulances); only 5 patients (8.8%) were transferred with medical escort. Mean ICU length of stay was 21.9 days (range 2–61 days). Mean duration of mechanical ventilation was 9.2 days (range 1–33 days).

**Table 1** Baseline epidemiological and clinical characteristics of the study population (N=57)

Variable	n or value	% or range
Mean age (years)	32.8	16–68
Male sex	54	94%
Road traffic accidents (RTAs)	37	64.9%
Falls	12	21.1%
Assaults	7	12.3%
Pre-existing comorbidities	6	10.5%
Mean admission GCS	7.9	3–14
Equal and reactive pupils (PERRL)	37	65%
Anisocoria	15	26.3%
Bilateral unreactive mydriasis	5	8.8%
Hemodynamic instability at admission	23	40%
Respiratory instability at admission	16	28.1%
Associated injuries ( $\geq 1$ lesion)	41	71.9%
Mean ICU length of stay (days)	21.9	2–61
Mean duration of mechanical ventilation (days)	9.2	1–33

GCS: Glasgow Coma Scale; ICU: intensive care unit; PERRL: pupils equal, round, reactive to light; RTA: road traffic accident.

### 3.2. Clinical and Radiological Findings

Hemodynamic instability was present in 23 patients (40%) and respiratory instability in 16 patients (28.1%) at admission. A focal neurological deficit was identified in 4 patients (7%). Forty-one patients (71.9%) presented at least one associated injury; facial (28%) and thoracic (25.8%) injuries were most prevalent.

All patients underwent brain CT imaging. Table 2 details the distribution of intracranial lesions. Cerebral contusion was the predominant finding (61.4%), followed by acute subdural hematoma (SDH, 56.1%), subarachnoid hemorrhage (SAH, 52.6%), skull fracture (52.6%), and epidural hematoma (EDH, 29.8%). Cerebral herniation was present on initial CT in 13 patients (22.8%). Follow-up CT was performed in 54 patients (94.7%) at a mean interval of 20 hours; it demonstrated lesion progression in 41%, stability in 52%, and improvement in 7%.

**Table 2** Distribution of intracranial CT lesions on initial scan (N=57)

Intracranial CT Lesion	n	Frequency
Cerebral contusion	35	61.4%
Subdural hematoma (SDH)	32	56.1%
Subarachnoid hemorrhage (SAH)	30	52.6%
Skull fracture (including depressed: 19.3%)	30	52.6%
Epidural hematoma (EDH)	17	29.8%
Cerebral herniation	13	22.8%
Intraventricular hemorrhage (IVH)	9	15.8%
Cerebral edema	11	19.3%
Intracerebral hematoma (ICH)	7	12.3%
Normal CT scan	1	1.8%

SDH: subdural hematoma; EDH: epidural hematoma; SAH: subarachnoid hemorrhage; IVH: intraventricular hemorrhage; ICH: intracerebral hematoma.

### 3.3. Therapeutic Management

All patients received invasive mechanical ventilation following orotracheal intubation. Deep sedation-analgesia was systematic (midazolam, fentanyl, propofol; mean duration 6.7 days, range 1–18). Osmotherapy (mannitol and/or hypertonic saline) was administered to 29 patients (50.9%) for clinically diagnosed or transcranial Doppler-detected intracranial hypertension. Blood transfusion was required in 20 patients (35.1%). Mechanical thromboprophylaxis (compression stockings) was systematic; pharmacological prophylaxis (enoxaparin 4,000 IU/day) was initiated at 3–5 days based on individual hemorrhagic and thrombotic risk. Antiepileptic prophylaxis (phenobarbital) was universal.

Twenty-two patients (38.6%) underwent emergency neurosurgical intervention at a mean delay of 3 hours: hematoma evacuation in 21 cases (95.5%), decompressive craniectomy in 1 case, and external ventricular drainage (EVD) in 2 cases. Thoracic drainage was performed in 9 patients (15.8%) for pneumo/hemothorax.

### 3.4. Complications

Table 3 summarizes in-hospital complications. Intracranial hypertension (IH) was the most frequent complication (61.4%), present in 89% of patients who died. Nosocomial infections occurred in 42.1% of patients, predominantly ventilator-associated pneumonia (VAP, n=20). Pressure injuries were documented in 38.6% of patients. Acute respiratory distress syndrome (ARDS) complicated the course in 9 patients (15.8%), and refractory status epilepticus (RSE) was diagnosed in 6 patients (10.5%) by electroencephalography.

**Table 3** In-hospital complications (N=57)

Complication	n	Frequency
Intracranial hypertension (IH)	35	61.4%
Nosocomial infections (including VAP n=20)	24	42.1%
Pressure injuries (decubitus ulcers)	22	38.6%
Shock (septic 70%, cardiogenic 30%)	10	17.5%
Acute respiratory distress syndrome (ARDS)	9	15.8%
Refractory status epilepticus (RSE)	6	10.5%

IH: intracranial hypertension; VAP: ventilator-associated pneumonia; ARDS: acute respiratory distress syndrome.

### 3.5. Mortality and Neurological Outcome (GOS)

In-hospital mortality was 49.1% (n=28); mean time to death was 13.4 days (range 2–63 days). Causes of death were: refractory IH in 16 patients (57%), brain death in 6 (21.4%), septic shock in 5 (17.9%), and undetermined cause in 1 (3.6%).

Among the 29 survivors, mean GOS at ICU discharge was 2.3: GOS 2 in 1 patient (3.4%), GOS 3 in 11 (37.9%), GOS 4 in 16 (55.2%), and GOS 5 in 1 (3.4%). Follow-up was performed at 3 months in 27 survivors (47.4%) and at 6 months in 23 survivors (40.4%). A progressive and marked improvement in GOS was observed over time: at 6 months, 65.2% of followed patients (15/23) achieved good recovery (GOS 5). Table 4 details the temporal GOS distribution.

**Table 4** Glasgow Outcome Scale (GOS) distribution at ICU discharge, 3 months, and 6 months in survivors

GOS Grade	Discharge (n=29)	3 months (n=27)	6 months (n=23)	Trend
1 (Death)	0	0	0	—
2 (Vegetative state)	1 (3.4%)	1 (3.7%)	0 (0%)	↓
3 (Severe disability)	11 (37.9%)	3 (11.1%)	3 (13.0%)	↓↓
4 (Moderate disability)	16 (55.2%)	15 (55.6%)	6 (26.1%)	↓
5 (Good recovery)	1 (3.4%)	8 (29.6%)	15 (65.2%)	↑↑↑

GOS: Glasgow Outcome Scale. ↑/↓: increase/decrease in proportion relative to ICU discharge. Loss to follow-up: 2 patients at 3 months, 6 patients at 6 months.

### 3.6. Prognostic Scores

Marshall CT classification was dominated by class V (surgically evacuated mass lesions, 31.6%) and class VI (unevacuated mass lesions, 49.1%). Mortality by Marshall class was 44.4% for class V and 71% for class VI. Mean Rotterdam score was 3.2 (range 1–5); mortality reached 92% for Rotterdam score 5. Mean ISS was 11.6; 87.7% of patients had an ISS of 25–39. The mean IMPACT-predicted 6-month mortality probability was 32.1% (range 4.9–62.9%); the mean probability of unfavorable outcome at 6 months was 52.3% (range 8.7–86.5%).

### 3.7. Univariate Analysis of Prognostic Factors

Table 5 presents the results of the univariate analysis comparing survivors and non-survivors. Statistically significant factors associated with in-hospital mortality were: lower admission GCS (survivors 8.72±2.25 vs. non-survivors 7.21±2.78; p=0.028), hemodynamic instability at admission (p=0.035), occurrence of IH during hospitalization (p=0.001), SAH on initial CT (p=0.022), and nosocomial infection (p=0.031). Age, sex, hypoxia at admission, and EDH were not statistically significant. Multivariate analysis was not feasible given the sample size.

**Table 5** Univariate analysis of prognostic factors: survivors vs. non-survivors

Variable	Survivors (n=29)	Non-survivors (n=28)	p-value
Mean age (years)	30.32 ± 8.68	36.04 ± 13.90	0.072 (NS)
Male sex	26/29	28/28	0.237 (NS)
Admission GCS (mean ± SD)	8.72 ± 2.25	7.21 ± 2.78	0.028 *
Hemodynamic instability	7/29	10/28	0.035 *
Hypoxia at admission	5/29	11/28	0.082 (NS)
Intracranial hypertension (IH)	10/29	25/28	0.001 ***
Subarachnoid hemorrhage (SAH)	13/29	17/28	0.022 *
Nosocomial infection	16/29	7/28	0.031 *
Epidural hematoma (EDH)	10/29	7/28	0.565 (NS)

\*p<0.05; \*\*\*p<0.001; NS: not significant. GCS: Glasgow Coma Scale; SD: standard deviation; IH: intracranial hypertension; SAH: subarachnoid hemorrhage; EDH: epidural hematoma.

## 4. Discussion

### 4.1. Epidemiology: The Young Male RTA Pattern

The epidemiological profile of our cohort — mean age 32.8 years, 94% male, 64.9% RTA-related — is consistent with sTBI data from other Moroccan and African series [11–15] and reflects the disproportionate exposure of young men to road traffic risks in LMICs. This pattern contrasts with European and North American cohorts where sTBI increasingly affects older populations through falls, with a higher burden of pre-existing anticoagulation and cardiovascular comorbidities [16].

The near-total absence of medicalized prehospital care (91% non-medicalized transport) is a critical system-level vulnerability in our context. Evidence consistently demonstrates that prehospital hypotension (systolic BP <90 mmHg) and hypoxia (SpO<sub>2</sub> <90%) — both preventable with medicalized transport — independently double the risk of death and poor neurological outcome in sTBI [17, 18]. Addressing this gap represents the single highest-impact intervention available at the health system level in Morocco.

### 4.2. Clinical Presentation and Radiological Findings

A mean admission GCS of 7.9 reflects the severity of our patient population. The mortality gradient across GCS strata — 75% for GCS ≤5, 50% for GCS 6–8, 37.5% for GCS 9–12 — is concordant with IMPACT data and international series [19, 22]. Pupillary abnormalities (anisocoria 26.3%, bilateral unreactive mydriasis 8.8%) indicated frequent mass effect or impending herniation at admission.

The predominance of cerebral contusions (61.4%) and SDH (56.1%) reflects the high-kinetic-energy acceleration/deceleration mechanism characteristic of motorcycle accidents [20]. The 71.9% rate of associated injuries highlights the polytraumatic nature of most sTBI cases in our cohort, requiring simultaneous management of extracranial hemorrhage and cerebral injury — a complex challenge in a setting with limited trauma team resources.

### 4.3. Mortality: Context-Specific Determinants

The in-hospital mortality of 49.1% exceeds the 30–37% reported in high-income country ICU series [16, 19] but is comparable to other Moroccan and African centers (Table 6). This excess mortality is multifactorial: delayed or absent prehospital medical management, limited intracranial pressure (ICP) monitoring (not available in our unit during the study period), higher proportion of severely injured patients (ISS 25–39 in 87.7%), and delayed access to neurosurgical intervention in some cases.

Refractory IH was responsible for 57% of deaths — a proportion consistent with the international literature [3, 16] — confirming that IH management is the primary lever for mortality reduction. The absence of direct ICP monitoring in

our unit represents a significant limitation: ICP-guided therapy has been shown to improve outcomes by enabling precise titration of osmotherapy, sedation, and decompressive craniectomy thresholds [3, 16].

**Table 6** Comparison of in-hospital sTBI mortality: our series vs. selected published data

Study (Country)	Belachi (Morocco)	Haddar (Morocco)	Assamadi (Morocco)	EU / USA series	Present series (Fez)
Year	2013	2016	2016	Multicenter	2021
In-hospital mortality	~50%	43.2%	64.8%	30–37%	49.1%
Mean age (years)	NR	NR	NR	47–60	32.8
RTA (% of etiology)	>60%	>50%	>50%	Varies	64.9%
ICP monitoring	NR	NR	NR	Standard	Not available

NR: not reported; RTA: road traffic accident; ICP: intracranial pressure. EU/USA data from Brain Trauma Foundation multicenter analyses.

#### 4.4. Neurological Recovery: A Progressive and Encouraging Trajectory

The most compelling finding of this study is the marked progressive improvement in GOS among survivors over 6 months: from 3.4% achieving GOS 5 at ICU discharge, to 29.6% at 3 months, and 65.2% at 6 months. This trajectory aligns with established neuroscience evidence demonstrating that neuroplasticity and functional recovery in sTBI can continue for 12–24 months post-injury [6, 21]. It provides a strong clinical rationale for sustained therapeutic effort and family support during the acute phase, even in patients with initially poor GOS scores.

These findings also highlight a critical gap in the Moroccan health system: the absence of structured neurorehabilitation pathways. The majority of patients who achieved GOS 5 at 6 months were initially discharged with GOS 3–4, suggesting that organized rehabilitation services would substantially improve functional outcomes. Prospective studies using extended GOS (GOSE) and patient-reported quality of life measures (SF-36, QOLIBRI) are needed to more comprehensively characterize long-term outcomes.

An important limitation of the follow-up data is the substantial loss to follow-up: only 40.4% of survivors were assessed at 6 months. This attrition likely introduced a favorable survival bias, as patients with severe sequelae may have been less accessible for follow-up. The true proportion of good recovery may therefore be somewhat lower than our data suggest.

#### 4.5. Prognostic Factors: Alignment with IMPACT Model

The five significant prognostic factors identified in our univariate analysis — admission GCS, hemodynamic instability, IH, SAH, and nosocomial infection — are broadly concordant with the IMPACT model predictors, which include age, motor GCS component, pupillary reactivity, hypotension, hypoxia, Marshall classification, SAH, and EDH [19, 22].

The dominant prognostic weight of IH ( $p=0.001$ , present in 89% of non-survivors) underscores its central role as both a mechanism of secondary injury and a treatment target. Our IH management protocol — deepened sedation, osmotherapy with mannitol or hypertonic saline, and decompressive craniectomy as a last resort — follows international guidelines [3], but was significantly constrained by the absence of direct ICP monitoring.

The association between nosocomial infection and mortality ( $p=0.031$ ) deserves specific attention. sTBI patients are uniquely vulnerable to hospital-acquired infections due to trauma-induced immunosuppression, prolonged mechanical ventilation, central venous catheterization, and aspiration. VAP occurred in 20 of 57 patients (35.1%), a rate exceeding most European ICU benchmarks (10–25%) [16], reflecting the resource constraints of care delivery in our setting and the priority of infection prevention bundles.

The non-significant association of EDH with mortality ( $p=0.565$ ) is consistent with IMPACT findings, where the presence of EDH is a relatively favorable CT marker compared to other lesions, owing to its reversibility when promptly evacuated [19]. The 41% rate of CT lesion progression in our follow-up series also illustrates the dynamic nature of sTBI and justifies routine early repeat imaging.

#### 4.6. Strengths and Limitations

The principal strength of this study is its provision of the first sequential neurological outcome data (discharge, 3 months, 6 months) for sTBI patients managed in the ICU of Hassan II University Hospital, including a systematic univariate prognostic factor analysis and benchmarking against international comparators. It contributes original evidence from a North African academic center underrepresented in the global sTBI literature.

Key limitations include: the retrospective design with its inherent selection and information biases; the relatively small sample size (n=57), limiting statistical power and precluding multivariate analysis; the absence of direct ICP monitoring; the substantial loss to follow-up at 6 months (59.6% of survivors); the absence of standardized neurorehabilitation pathways; and the lack of extended GOS or patient-reported quality of life instruments. A prospective multicenter design with standardized data collection, ICP monitoring, and structured follow-up would be required to overcome these limitations.

---

#### 5. Conclusion

Severe traumatic brain injury in this Moroccan ICU cohort is characterized by a 49.1% in-hospital mortality, primarily driven by refractory intracranial hypertension. It predominantly affects young adult males involved in road traffic accidents, most of whom receive no prehospital medical care. The five statistically significant prognostic factors — low GCS, hemodynamic instability, intracranial hypertension, subarachnoid hemorrhage, and nosocomial infection — define actionable therapeutic targets within current resource constraints.

Notwithstanding the high acute mortality, survivors demonstrate a striking progressive neurological recovery, with 65.2% achieving good functional outcome (GOS 5) at 6 months. This trajectory underscores the value of sustained intensive care effort and the urgent need for structured neurorehabilitation services in Morocco. Priority system-level investments should target: medicalization of prehospital transport, implementation of ICP monitoring, VAP and catheter-related infection prevention bundles, and national road safety policy. Prospective multicenter Moroccan studies are needed to generate region-specific prognostic benchmarks and validate therapeutic protocols in this population.

---

#### Compliance with ethical standards

##### *Disclosure of conflict of interest*

No conflict of interest to be disclosed.

##### *Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

---

#### References

- [1] Savitsky B, Givon A, Rozenfeld M, Radomislensky I, Peleg K. Traumatic brain injury: It is all about definition. *Brain Inj.* 2016;30(12):1537–1544. doi:10.1080/02699052.2016.1187290.
- [2] Mrozek S, Srairi M, Geeraerts T. Traumatisme crânien grave à la phase aiguë. *J Eur Urgences Réanimation.* 2017;29(2):112–119. doi:10.1016/j.jeurea.2017.08.005.
- [3] Brain Trauma Foundation. Guidelines for the Management of Severe Traumatic Brain Injury, 4th Edition. *Neurosurgery.* 2017;80(1):6–15. doi:10.1227/NEU.0000000000001432.
- [4] Masson F. Santé publique et traumatismes crâniens graves. *Ann Fr Anesth Réanim.* 2000;19:261–269.
- [5] Perel P, Wasserberg J, Bhatt MA, et al. A systematic review of prognostic factors after severe traumatic brain injury. *Br J Neurosurg.* 2006;20(5):286–294. doi:10.1080/02688690600949363.
- [6] Jennett B, Bond M. Assessment of outcome after severe brain damage. *Lancet.* 1975;1(7905):480–484. doi:10.1016/s0140-6736(75)92830-5.
- [7] Teasdale GM, Pettigrew LE, Wilson JT, Murray G, Jennett B. Analyzing outcome of treatment of severe head injury: a review and update on advancing the use of the Glasgow Outcome Scale. *J Neurotrauma.* 1998;15(8):587–597. doi:10.1089/neu.1998.15.587.

- [8] Wilson JT, Pettigrew LE, Teasdale GM. Structured interviews for the Glasgow Outcome Scale and the extended Glasgow Outcome Scale. *J Neurotrauma*. 1998;15(8):573–585.
- [9] Dikmen SS, Machamer JE, Powell JM, Temkin NR. Outcome 3 to 5 years after moderate to severe TBI. *Arch Phys Med Rehabil*. 2003;84(10):1449–1457. doi:10.1016/s0003-9993(03)00287-9.
- [10] Lingsma HF, Roozenbeek B, Steyerberg EW, Murray GD, Maas AI. Early prognosis in traumatic brain injury: from prophecies to predictions. *Lancet Neurol*. 2010;9(5):543–554. doi:10.1016/S1474-4422(10)70065-X.
- [11] Belachi S, Aissaoui A, El Khamlichi A. Cranioencephalic traumatic injuries operated at the CHU of Fez: report of 144 cases. *Afr J Neurol Sci*. 2013.
- [12] Haddar R. Severe traumatic brain injury in the ICU — CHU Hassan II, Fez [Doctoral Thesis in Medicine]. Fez: Sidi Mohammed Ben Abdellah University; 2016.
- [13] Belkaich A. Outcome and quality of life in severe traumatic brain injury [Doctoral Thesis in Medicine]. Rabat: Mohammed V University; 2018.
- [14] Khallouk A. Prognostic factors of severe traumatic brain injury in the ICU [Doctoral Thesis in Medicine]. Fez: Sidi Mohammed Ben Abdellah University; 2019.
- [15] Aabydi MA. Severe traumatic brain injury and prognostic scoring. *Ann Fr Anesth Réanim*. 2010;29(2).
- [16] Stocchetti N, Maas AI. Traumatic intracranial hypertension. *N Engl J Med*. 2014;370(22):2121–2130. doi:10.1056/NEJMra1208518.
- [17] Badjatia N, Carney N, Crocco TJ, et al. Guidelines for prehospital management of traumatic brain injury, 2nd edition. *Prehosp Emerg Care*. 2008;12(Suppl 1):S1–S52. doi:10.1080/10903120701732052.
- [18] Spaite DW, Hu C, Bobrow BJ, et al. Mortality and prehospital blood pressure in patients with major traumatic brain injury: implications for the hypotension threshold. *JAMA Surg*. 2017;152(4):360–368. doi:10.1001/jamasurg.2016.4686.
- [19] Steyerberg EW, Mushkudiani N, Perel P, et al. Predicting outcome after traumatic brain injury: development and international validation of prognostic scores based on admission characteristics. *PLoS Med*. 2008;5(8):e165. doi:10.1371/journal.pmed.0050165.
- [20] Rosenfeld JV, Maas AI, Bragge P, Morganti-Kossmann MC, Manley GT, Gruen RL. Early management of severe traumatic brain injury. *Lancet*. 2012;380(9847):1088–1098. doi:10.1016/S0140-6736(12)60864-2.
- [21] Maas AI, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. *Lancet Neurol*. 2008;7(8):728–741. doi:10.1016/S1474-4422(08)70164-9.
- [22] Murray GD, Butcher I, McHugh GS, et al. Multivariable prognostic analysis in traumatic brain injury: results from the IMPACT study. *J Neurotrauma*. 2007;24(2):329–337. doi:10.1089/neu.2006.0035.