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Determinants of Outcome in Severe Traumatic Brain Injury Managed in Surgical ICU

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ABSTRACT:

Introduction: Severe traumatic brain injury (sTBI) causes major mortality, mostly after road traffic accidents.

Methods: Retrospective review of 102 sTBI patients admitted to a surgical ICU.

Results: Young males predominated. CT showed mainly contusions and hemorrhages. Mortality was 33%, associated with low GCS, bilateral mydriasis, and severe lesions.

Discussion: Prognosis depends on early recognition of key clinical and radiological severity factors.

Conclusion: Rapid multidisciplinary care and prevention of road accidents are essential.

KEYWORDS: severe traumatic brain injury / glascow coma scale / intracranial hypertension / pupillary reactivity

INTRODUCTION

Severe traumatic brain injuries (STBI) represent a major public health issue due to their frequency, severity, and the heavy socioeconomic burden they generate in low-resource countries as well as globally [1][2]. They result from the transmission of energy to the skull causing immediate primary lesions—contusions, intracranial hemorrhages, diffuse axonal injuries—to which secondary lesions are added, related to hypoxia, hypotension, cerebral edema, and ischemia, responsible for most of the mortality and functional sequelae [3][4]. Understanding the anatomical and pathophysiological mechanisms, particularly the Monro-Kellie doctrine and variations in intracranial pressure within a fixed cranial volume, remains essential to guide monitoring, multimodal surveillance, and early therapeutic strategies [5][6]. Thus, optimal management of sTBI relies on an integrated chain of care beginning in the prehospital phase to prevent systemic and intracranial secondary brain insults that largely determine prognosis.

MATERIALS AND METHODS

We conducted a retrospective descriptive and analytical study of 102 patients admitted for severe traumatic brain injury to the surgical emergency ICU of CHU Ibn Rochd in Casablanca. The study period extended from January 2020 to June 2024. Patients with a Glasgow Coma Scale (GCS) ≤ 8 after stabilization of vital functions were included, whether presenting with isolated sTBI or as part of polytrauma. Non-severe head injuries, deaths occurring in the emergency room, and incomplete files were excluded.

Data collected from medical records included epidemiological characteristics, initial clinical parameters, biological and radiological findings, therapeutic management, and ICU outcome. A standardized sheet was used to unify data collection. Statistical analysis was performed with SPSS 25. Quantitative variables were expressed as means ± standard deviation and compared using Student's t-test; qualitative variables were analyzed using the Chi-square test. Statistical significance was set at $p < 0.05$.

RESULTS

A total of 102 patients with severe traumatic brain injury were included. The mean age was 38.5 ± 16 years, with a strong male predominance (87.3%, sex ratio 6.8). Road traffic accidents were the leading cause (74.5%). Prehospital medical transport was rare (7.8%), and the mean admission delay was 8 ± 4 hours.

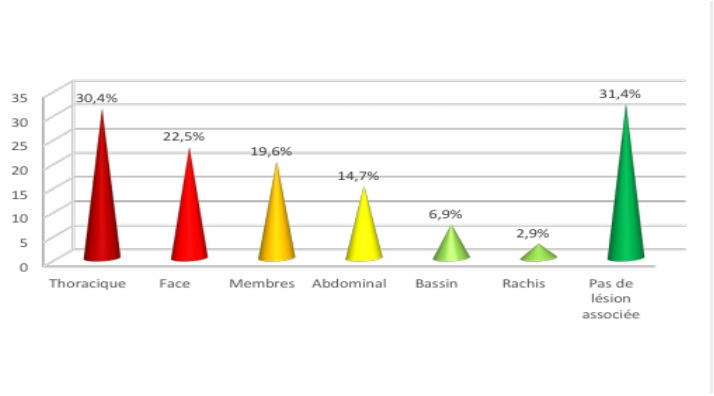
At admission, most patients had a Glasgow score between 6 and 8 (76.5%), while 23.5% had a score < 6. Pupillary assessment revealed 24 cases of anisocoria and 5 cases of bilateral non-reactive mydriasis.

Pupil reactivity	Number	percentage
Symmetric and reactive	52	51%
Tight miosis	21	20.6%
Anisocoria	24	23.5%
Bilateral mydriasis	5	4.9%

Distribution of patients according to pupillary reactivity.

Initial CT scans mainly showed cerebral contusions (54.9%), subarachnoid hemorrhage (38.2%), cerebral edema (31.4%), and subdural hematoma (26.5%). Among the 58 follow-up CT scans, 63.8% showed lesion worsening.

Extracranial injuries were frequent in polytrauma patients, including pulmonary contusions (13.7%), abdominal lesions (14.7%), and pelvic fractures (6.9%).



Distribution of patients according to associated lesions.

Biologically, disturbances in blood glucose, potassium, and urea were significantly associated with poor outcomes.

All patients were mechanically ventilated. Ventilation duration was significantly longer among non-survivors (6.7 vs 4.9 days). The use of catecholamines (26.5%) and osmotherapy was associated with higher mortality. Surgery was performed in 31.4% of patients, mostly neurosurgical.

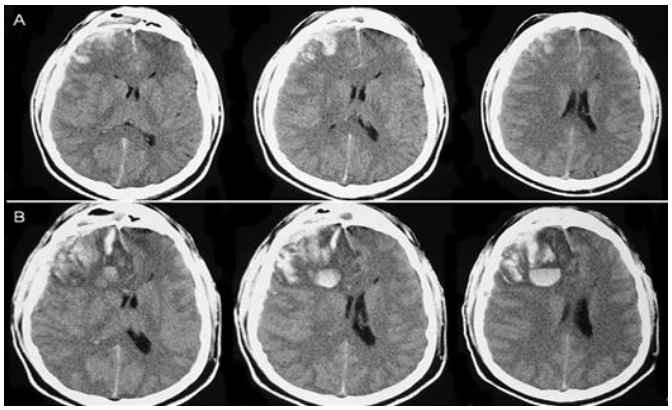
Complications occurred in 56.9% of patients, mainly ventilator-associated pneumonia (36.3%). Mean ICU stay was 10 days. Overall mortality reached 33.3%, mainly due to neurological deterioration.

Multivariate analysis identified the following prognostic determinants: low initial GCS, bilateral non-reactive mydriasis, mean arterial pressure ≤ 85 mmHg, SpO_2 < 92%, subarachnoid hemorrhage, cerebral edema, brain herniation, metabolic disturbances (potassium, urea, glucose), need for catecholamines, osmotherapy, prolonged ventilation, and occurrence of intracranial hypertension or septic shock.

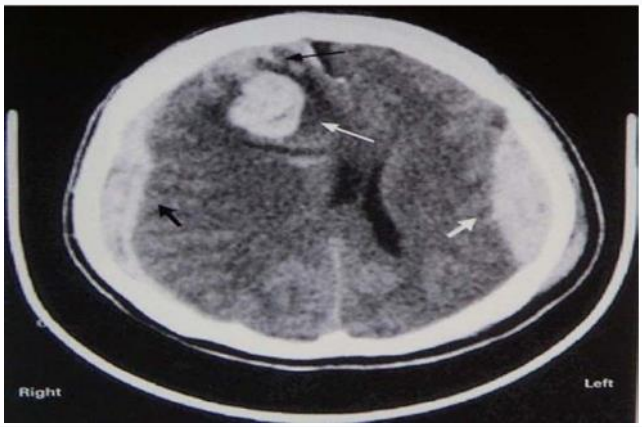
DISCUSSION

Severe traumatic brain injuries represent a major challenge in intensive care because they combine immediate primary lesions with a cascade of secondary injuries responsible for most morbidity and mortality. Secondary injuries are dominated by hypotension and hypoxia, whose negative impact on cerebral perfusion, intracranial pressure (ICP), and neurological prognosis is well established [7][8][9].

A precise understanding of cranial and brain anatomy and physiopathology, particularly the Monro-Kellie doctrine and intracranial volume dynamics, is essential to interpret CT findings and guide initial management [10][11][12]. Clinical assessment relies mainly on the Glasgow score, pupil examination, and hemodynamic stability—key elements in identifying high-risk patients. CT scan is the reference exam in initial evaluation, enabling detection of operable lesions such as extradural or subdural hematomas, hemorrhagic contusions, or signs of intracranial hypertension, thereby guiding neurosurgical strategy [13][14][15].



Time evolution of a hemorrhagic contusion between H2 (A) and H 16 (B) post-traumatic



A 71-year-old man, victim of a road traffic accident, presented with a subdural hematoma, an extradural hematoma, an intraparenchymal hematoma, and a meningeal hemorrhage.

Management principles aim to prevent secondary brain insults by maintaining adequate oxygenation and blood pressure to ensure optimal cerebral perfusion pressure, and by rapidly correcting metabolic abnormalities such as hyperglycemia, hypocalcemia, or electrolyte disorders, whose prognostic impact is well documented [8][16]. Neurophysiological monitoring is central: ICP measurement, transcranial Doppler, jugular venous oxygen saturation, brain tissue oxygen pressure ($PtiO_2$), continuous EEG, and sometimes cerebral microdialysis allow tailored treatment according to cerebral physiology and ischemic risk [17].

Management of intracranial hypertension relies primarily on osmotherapy using mannitol or hypertonic saline. Mannitol acts through both osmotic and volume-expanding mechanisms, whereas hypertonic saline provides a faster and more sustained reduction in ICP in some studies, particularly in refractory cases [18].

Controlled hyperventilation may be used transiently in acute ICP surges with signs of herniation, but prolonged use is discouraged due to the risk of cerebral hypoperfusion. In refractory cases, invasive approaches such as external ventricular drainage or decompressive craniectomy become necessary. Corticosteroids are contraindicated due to lack of benefit and increased complications [13]. Anticonvulsant prophylaxis is recommended only during the first week and in high-risk patients.

Despite progress, systemic and nosocomial complications—ventilator-associated pneumonia, urinary infections, post-traumatic meningitis, ARDS, and severe metabolic disorders—remain major causes of deterioration and mortality, primarily neurological despite maximal therapy. Optimal management therefore requires an integrated approach combining early diagnosis, prevention of secondary insults, continuous multimodal monitoring, and hierarchical therapeutic strategies involving medical and neurosurgical interventions. These measures significantly improve survival and functional outcomes in a condition with persistently severe prognosis.

CONCLUSION

The management of severe traumatic brain injury requires close interdisciplinary collaboration and specialized training of emergency teams to reduce secondary complications and optimize outcomes. Additionally, strong preventive policies led by health authorities, including effective programs targeting road traffic accidents, are essential to reduce the primary cause of these injuries.

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