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ORIGINAL ARTICLE

Clinical characteristics and prognosis of traumatic head injury following road traffic accidents admitted in ICU "analysis of 694 cases"

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Abstract

Background The aim of the present study is to analyze the clinical and epidemiological characteristics of Traumatic Brain Injury (TBI) following Road Traffic Accidents (RTAs). Moreover, we aim to evaluate the outcome of the TBI victims referred to our medico-surgical Intensive Care Unit (ICU), and to define predictive factors associated with poor prognosis.

Methods A retrospective study over a 4-year period (2009 to 2012) of 694 patients with head injuries, incurred during road traffic accidents, admitted to the Intensive Care Unit (ICU) of a university hospital (Sfax-Tunisia). Basic demographic, clinical, biological, and radiological data were recorded on admission and during the ICU stay.

Results There were 592 males (85.3%), and 102 female patients. The mean age was at 31.8 ± 17.8 years (range 1–91). The mechanism of the accident was detailed in 666 patients (96%). The majority of the victims were motorcycle riders and/or passengers (40.5%), followed by pedestrians (29.1%). Extra-cranial pathology was present in 452 patients (65%). A total of 677 patients (97.6%) required intubation, mechanical ventilation, and sedation. Mean ICU stay was 16 ± 17.4 days. A total of 187 patients (26.9%) died during their hospital stay. The GOS performed within a mean delay of 6 months after hospital discharge was as follows: 198 deaths (28.5%), 13 vegetative state (1.9%), and 349 (50.3%) good recovery and/or moderate disability. A multivariate analysis showed that the factors which correlated with a poor prognosis (mortality and severe disability) were: age > 38 years, Glasgow coma scale score < 8, subdural hematoma, and development of secondary systemic insults (respiratory, circulatory, and metabolic).

Conclusion In Tunisia, traumatic brain injury due to RTAs is a frequent cause of ICU admission, especially among young adults, and is associated with high mortality and morbidity rates. The majority of the victims were motorcycle riders and/or passengers and pedestrians. The factors associated with a poor outcome were: age > 38 years, Glasgow Coma Scale score < 8, subdural hematoma, and development of secondary systemic insults (respiratory, circulatory, and metabolic). As a consequence, prevention is highly warranted.

Keywords Trauma · Head injury · Road traffic accident · Intensive care unit · Multivariate analysis · Prognosis

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Abbreviations

TBI	Traumatic brain injury
ICU	Intensive Care Unit
RTAs	Road traffic accidents
CT	Computed cerebral tomography
GCS	Glasgow coma scale score
SAPSII	Simplified acute physiology score
PRISM	Pediatric risk of mortality
PTS	Pediatric trauma score (PTS)
DAI	Diffuse axonal injury
ICP	Intracranial pressure
SSIs	Secondary systemic insults
GOS	Glasgow outcome scale
SDH	Subdural hematoma

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Introduction

All around the world, one of the prominent external causes of morbidity and mortality, regardless of income or geographic area, is injury. Traumatic brain injury, with an estimated 10 million cases a year worldwide, is a major cause of death and disability among a predominantly young population (aged 15 to 35 years) [1].

Nowadays, the increasing modernization of road traffic, a hike in road traffic accidents (RTAs), becomes exponentially more frequent and serious [1]. Indeed, according to the World Health Organization's (WHO) predictions, in 2030, traffic accidents will be the leading cause of morbidity and mortality of young people worldwide [1]. This morbi-mortality is largely related to the frequency and severity of the brain lesions caused by this type of accidents. Brain trauma is considered to be the leading cause of death following a traffic accident.

On the other hand, the majority of epidemiological studies on head trauma are not exclusively devoted to traffic accident-related head trauma, since they often include other mechanisms, as well (falls, aggressions, etc.).

In Tunisia, nearly 13,000 cases of motor-vehicle crash victims are recorded per year, among whom 1500 die according to the National Guard statistical data [1, 2].

Despite the considerable attention given to health statistics of RTAs, the epidemiological aspects of injuries resulting from such accidents are not fully understood in Tunisia and other developing countries. Moreover, road traffic injuries are one of the leading causes of death, hospitalization, disability, and low socio-economic status. In fact, survivors are susceptible to irreversible neurological damage, which represents an important socio-economic problem [1, 2]. In the Sfax area (South Tunisia), all severe traumatic brain injuries (TBIs) are admitted in the medico-surgical Intensive Care Unit (ICU) where specific monitoring tools (jugular venous saturation, intracranial pressure monitoring, and transcranial Doppler sonography) are not available. The aim of the present study is to analyze the clinical and epidemiological characteristics of traumatic brain injury R-T-A victims, to evaluate their outcome, and to define predictive factors associated with poor prognosis (in-hospital mortality and long-term poor outcomes).

Materials and methods

This study was approved by an internal review board.

Patients

In this retrospective study, were included all consecutive patients with traumatic brain injury related to traffic accidents and admitted to Intensive Care Unit (ICU) of Habib Bourguiba University Hospital over a period of 4 years, from 2009 to 2012. Were excluded all polytrauma cases without associated head trauma.

The data were recorded from the patients' clinical notes with multiple contributors. Our department is a 22-bed medico-surgical ICU in a teaching hospital of 510 beds that serves as a first-line medical center for an urban population of around 2 million inhabitants, and as a referral center for a larger population coming from the south of Tunisia. The total number of admissions in our unit is about 1000 per year. In our department, 3 beds are reserved to pediatric intensive care.

Patients were admitted directly from the scene of the accident within 6 h of injury. They were all examined and scored—according to Glasgow Coma Scale score (GCS)—on arrival, and underwent computed cerebral tomography (CT) scan as soon as feasible.

Methods

The patients' medical files were retrospectively reviewed, and the following data were recorded: age, gender, vital signs (respiratory rate, heart rate, and systolic and diastolic blood pressure), GCS score [3], body temperature in °C, Injury Severity Score (ISS) [4], Simplified Acute Physiology Score (SAPS II) calculated within 24 h after admission [3, 4], Pediatric Risk of Mortality (PRISM) score [5], and Pediatric Trauma Score (PTS) [5]. Were also recorded: mechanism of injury, pupil response, motor deficit, convulsion, presence of shock or arterial hypotension [5], cardiac arrest, fluid intake volume, brain CT scan results, and use of mechanical ventilation and/or catecholamines. The biological parameters measured on admission and during the ICU stay included serum glucose and sodium levels, blood urea and urine specific gravity, arterial blood gases and acid base state, hemoglobin concentration, platelet count, and prothrombinaemia.

Plain radiographic studies of the neck and cranial CT scans were done for all the patients. The CT scan findings were axed on the presence or absence of hematoma (whether extradural, subdural, or intracerebral), cerebral edema, subarachnoid hemorrhage, cerebral contusion, pneumocephalus, intracranial mass lesion, and herniation. In addition, these cranial CT scan results were stratified according to the "Traumatic Coma Data Bank Computed Tomography Classification" for Severe Brain Injury [6].

In all cases, the cerebral CT scan classification was performed by experienced university radiologists.

The neurological status was assessed using the GCS score at the site of the accident and again on hospital arrival before the use of sedative, but after resuscitation: the pre-intubation GCS (used in our analysis). Clinical injury severity was based on GCS score on admission and defined as mild TBI (GCS 14–15); moderate (GCS 9–13); or severe (GCS 3–8).

Our patients were intubated, ventilated, and received sedation with the Fentanyl-Midazolam association, as necessary. The treatment protocol generally applied was sedation and continuous muscle relaxation, normothermia (36–37 °C), and mild hyperventilation ($pCO_2 = 30–35$ mmHg). In our ICU, 15–30° elevation of the upper part of the body was used in all patients.

In our study, assessment of intracranial hypertension was based on clinical findings and imaging results: a second CT scan was performed if the first imaging was performed during the first 3 h after the trauma or if the neurologic status worsened (anisocoria, dysautonomia, seizure, new onset of motor deficit, etc.).

When Cerebral CT Scan showed cerebral edema and/or herniation, mannitol was administered. In our study, mannitol was restricted to patients with signs of brain edema, mass lesions or midline shift, or progressive neurologic deterioration not attributable to extra-cranial causes. The dose used was a bolus of 0.25–1 g/kg body weight, administered intravenously over a period of 15 to 20 min. This dose can be repeated as necessary every 6 h, during 48 h.

However, neither hypertonic saline nor corticosteroids were used in the management of the aforementioned cases. In our practice, anticonvulsants were used only when a patient had developed seizure. However, hypothermia therapy was not used in our study, while decompressive craniectomy was needed as necessary.

Following the protocol in our ICU, the bedhead was kept elevated in all cases and mannitol was used when raised intracranial pressure was suspected or CT showed cerebral edema and/or herniation.

In our ICU, therapies were directed by repeated CT scan and neurological state and evolution. In patients with extracranial suspected pathology, appropriate investigations were performed and usually a Computed Tomography (CT) body scan was performed. Whenever a diffuse axonal injury (DAI) is suspected, a brain magnetic resonance imaging is performed.

All clinical, biological, and radiological parameters and relevant therapeutic measures were registered on admission and during the ICU stay.

For each patient, daily mean levels of Na, K, and blood sugar were systematically recorded. In addition, we have recorded the development of Secondary Systemic Insults (SSIs) on admission and during ICU stay. SSIs were divided into subgroups of circulatory (hypotension or arterial hypertension) [2, 5, 6], respiratory (hypoxemia, hyper-, hypocapnia) [2, 5, 6], metabolic/ electrolytic SSIs (anemia, hyper- or hypoglycemia, hyponatremia, diabetes insipidus) [2, 5, 6], and hyperthermia. During the ICU stay, the following complications were documented: nosocomial infections [7], pneumonia [8], tract urinary infection [8] meningitis [7], and septicemia [9].

A Glasgow Outcome Scale (GOS) score [10, 11] was established after hospital discharge by the same ICU physician (HC—in all cases). This score was previously used to assess prognosis in head trauma [12]. In all cases, data were recorded via each patient's medical file (Neurosurgery and ICU medical record) after hospital discharge. In case of missing data, concerned patients' families were contacted by phone. Data recovery was thus made possible either by questioning the patients' parents and/or closest relatives, or by re-evaluating the patients themselves. Depending on their GOS score, these patients can be divided into five groups with increasing severity, ranging from good recovery to death as follows [10]:

1. Death (GOS 1).
2. Persistent vegetative state (GOS 2).
3. Severe disability: severe injury with permanent need for help with daily living (GOS3).
4. Moderate disability: disabled but independent (No need for assistance in everyday life).
5. (GOS4).
6. Good recovery: resumption of normal life despite minor deficits (GOS 5).

However, in our study, the patients were divided into two groups: GOS (I): patients with favorable outcome, involving patients having a good recovery and/or moderate disability (GOS 4 and 5). GOS (II): patients with unfavorable outcome, involving patients with severe disability or in a persistent vegetative state, and those who died (GOS 1–3).

Statistical analysis

Categorical data were expressed in proportion and subgroups [survival and death; GOS (I) and GOS (II)] and they were analyzed by the Chi-square test. Continuous variables were expressed as means (± SD) and subgroups evaluated by Student's *t* test. Risk factors were evaluated by univariate and multivariate analyses—by a multiple logistic stepwise regression procedure. Odds ratios were estimated from the *b* coefficients obtained, with respective 95% confidence intervals (CI 95%).

SAPS II, PRISM, PTS, ISS, and GCS Score were used to predict poor outcome [mortality and GOS (II)] and were analyzed using Receiver-Operating Characteristic (ROC)

curves. The area under the ROC curve which was estimated by the method of Hanley and McNeill [13] provides a measure of overall mortality of the test.

For comparable data, a p value less than 0.05 was considered as statistically significant.

Results

Were included in the present study 694 patients, admitted for head trauma secondary to traffic accidents. While 2459 were excluded, as they were admitted for non-traumatic problems, and 264 polytrauma patients without any head trauma (Fig. 1).

There were 592 males (85.3%) and 102 female patients. The mean age was at 31.8 ± 17.8 years (range 1–91). There were 109 (16%) children (age less than 17 years) and 585 adult patients (84%) (Fig. 2).

The demographic and clinical parameters on admission are shown in Table 1. GCS scores were available only for 629 patients. Mean GCS on admission was at 8.7 ± 3.4 (median: 8). According to GCS score, TBI was minor in 69 patients (11.1%), moderate in 225 (35.8%) and severe in 334 (53.1%). On hospital admission, 32 patients had bilateral mydriasis with time duration less than 30 min in all cases.

The mechanism of the accident was precised in 666 patients (96%). The majority of the victims were motorcycle riders and/or passengers on motorcycles (40.5%) followed by pedestrians (29.1%) (Table 2).

TBI was isolated in 242 patients (35%). However, extracranial pathology (polytrauma) was present in 452 patients (65%), including fracture of long-bones (30.5%), injury of

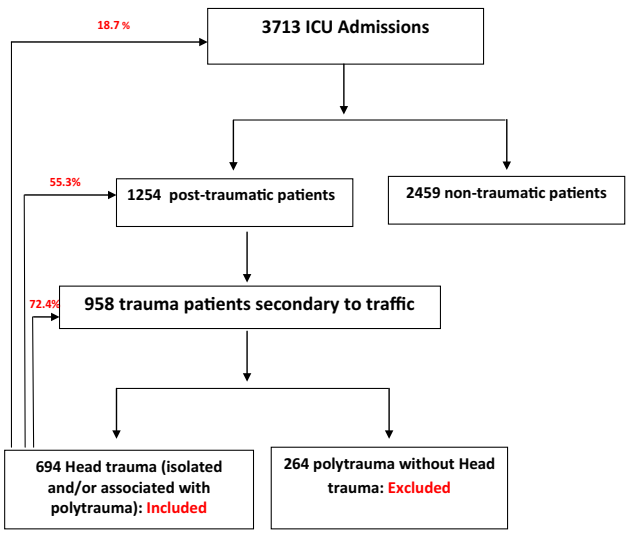

Fig. 1 Study flow chart

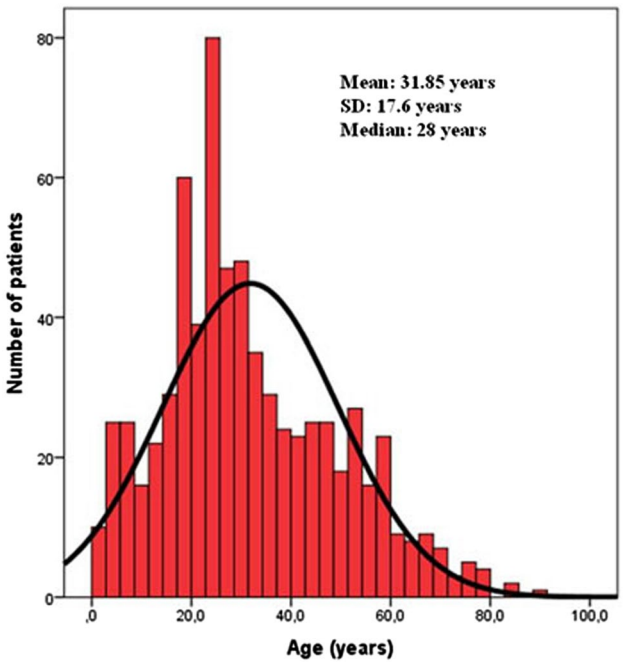

Fig. 2 Repartition of patients according to their age on ICU admission

Table 1 Demographic and clinical parameters on admission of all study population

Parameters	Results	%
Age (years)	31.9 ± 17.7	
Sex M/F	592/102	
SAPS II	37.8 ± 12.6	
GCS score	8.7 ± 3.4	
ISS score	34.7 ± 9.8	
PRISM score	12.7 ± 7.6	
HR (beats/min)	94.5 ± 23.8	
SBP (mmHg)	116 ± 30	
Respiratory distress	121	17.4
Shock	138	20
Cardiac arrest	5	0.7
Glasgow coma scale score	8.8 ± 3.4	
Anisocoria	158	22.8
Bilateral mydriasis	32	4.6
Motor deficit	48	6.9
Convulsion	55	7.9
Other injury	452	65
Pathological antecedent	152	22

HR heart rhythm, *SBP* systolic blood pressure, *GCS* Glasgow Coma Scale

Table 2 Repartition of all population according to the mechanism of accident

Host type	Number	Percentage (%)
Motorcycle riders and/or Passengers on motorcycles	270	40.5
Drivers of four-wheeled vehicles	105	15.8
Passengers on four-wheeled vehicles	86	12.9
Pedestrian	194	29.1
Open truck occupants	11	1.7
Total	666	100

the chest (44.7%), abdomen (15.7%), spine (16.3%), and of the pelvis (11.5%).

Four hundred and five patients (58.4%) had a fracture of the skull, and ninety-six patients (13.8%) had depressed skull fractures with brain contusions. A total of 677 patients (97.6%) required intubation, mechanical ventilation, and sedation on admission according to protocol. The mean duration of mechanical ventilation was 10.4 ± 9.8 days. Fluid resuscitation (0.9% normal saline) was used in 551 patients (79.4%), while 450 patients (64.8%) required blood (and/or its derivatives) transfusion. Moreover, catecholamines were used in 365 cases (52.6%).

Two hundred and thirty patients (33%) required craniotomy and neurosurgery, while 193 (27%) needed extra-cranial surgery. In our study, we have not excluded any patients from treatment due to neurological deterioration at admission. All patients were treated according to the protocol previously described.

The results of brain CT scan are presented in Table 3. Subarachnoid hemorrhage was observed in 526 patients (75.8%), subdural hematoma in 314 (45.2%), cerebral edema in 31%, and extradural hematoma in 186 (26.8%) patients.

Table 3 Results of cerebral CT scan

CT scan signs	Number	Percentage (%)
Subarachnoid hemorrhage	526	75.8
Fracture of skull	405	58.4
Cerebral contusion	515	74.2
Cerebral edema	217	31
Diffuse axonal injury	342	49.3
Extradural hematoma	186	26.8
Subdural hematoma	314	45.2
Pneumocephalus	143	20.6
Mass lesion	258	37.2
Brain herniation	161	23
Cerebral trunk injury	68	9.8
Depressed skull fracture	96	13.8
Normal CT scan	24	3.4

MRI was performed in 180 cases (26%); it showed Diffuse Axonal Injuries (DAIs) in 149 (82.8%) ones.

During the ICU stay, 679 patients (97.8%) developed secondary systemic insults (SSIs), as detailed in Table 4.

Several complications were also observed. Three hundred and twenty-eight patients (47.3%) developed nosocomial infections; pneumonia in 264 (38%), tract urinary infection in 70 (10%), meningitis in 26 (3.7%), septicemia in 90 (13%), and inner ear infection or conjunctivitis in 15 (2.1%) patients.

Moreover, 94 patients (13.5%) had thromboembolic complications. Acute renal failure was observed in 55 patients (7.9%), fat embolism in 4 (0.7%), pressure ulcer stage III or IV in 92 (13.3%), osteoma in 27 (3.9%), and gastro-intestinal hemorrhage in 8 (1.1%) patients.

The mean ICU stay was 16 ± 17.4 days. A total of 187 patients (26.9%) died during their hospital stay. Brain herniation was the main cause (66%) of early mortality.

A good correlation between the SAPSII score and the mortality was found in adult patients, since SAPSII value ≥ 40 was associated with death with a sensitivity at 68%, a specificity at 78%, and an area under the ROC curve at 0.78 (Fig. 3).

Moreover, a good correlation between the PRISM score and mortality was found in children, since PRISM value ≥ 14 was associated with death with a sensitivity at 66%, a specificity at 75%, and an area under the ROC curve at 0.78 (Fig. 4).

A univariate analysis showed that the factors associated with in-hospital mortality were: age ($p < 0.001$), SAPSII score ($p < 0.001$), ISS score ($p = 0.010$), presence of shock ($p < 0.001$), and bilateral mydriasis ($p < 0.001$) (Table 5). The factors associated with in-hospital mortality in multivariate analysis are featured in Table 6.

The GOS performed within a mean delay at 34 ± 24 months after hospital discharge (range 1 and 86 months;

Table 4 Frequency of secondary systemic insults among patients

Type of secondary systemic insults	Number (%)
Hyperthermia	482 (69.5)
Disseminated intravascular coagulation	368 (53)
Hyponatremia	99 (14.3)
Arterial hypotension	375 (54)
Arterial hypertension	71 (10.2%)
Hyperglycemia	253 (36.5)
Hypoxemia	118 (17)
Hypercapnia (> 45 mmHg)	123 (17.7)
Hypocapnia	180 (25.9)
Hypoglycemia (< 2.8 mmol/l)	12 (1.7)
Anemia (Hb < 8.5 g/dl)	301 (43.4)
Diabetes insipidus	21 (3%)

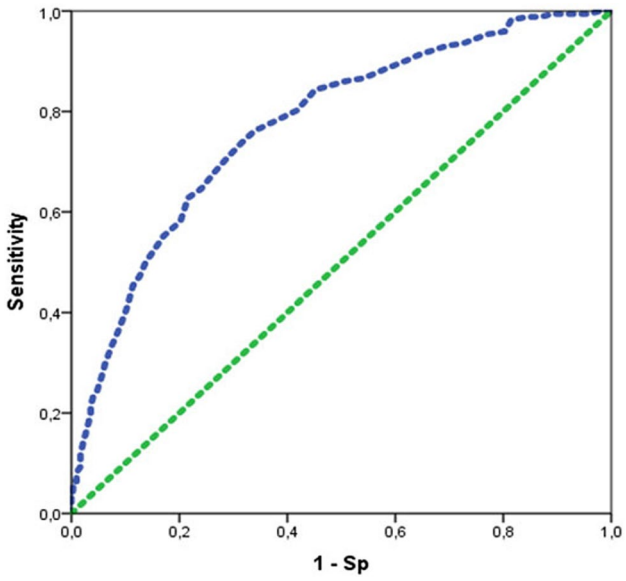

Fig. 3 Receiver-operating characteristic curve (ROC curve) for ability of SAPSII score to predict the mortality. The area under the curve was 0.78 indicating a good capability of the model to discriminate between survivors and non-survivors

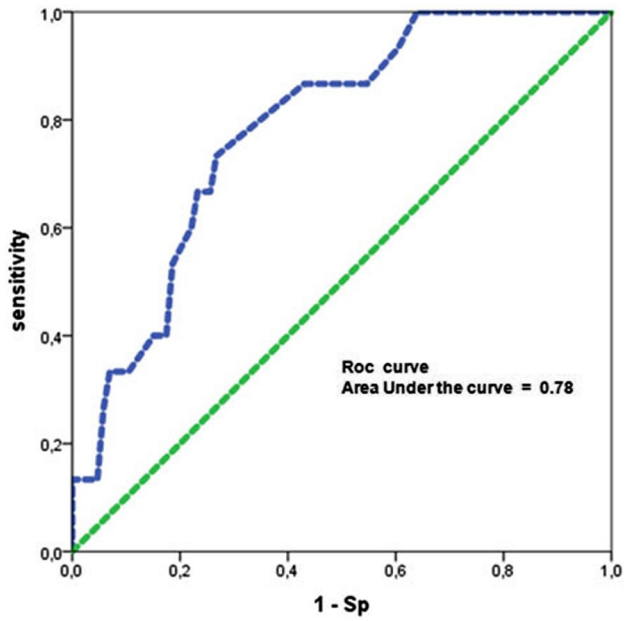

Fig. 4 Receiver-operating characteristic curve (ROC curve) for ability of PRISM to predict the mortality. The area under the curve was 0.78 indicating a good capability of the model to discriminate between survivors and non-survivors

median: 36 months) was as follows: 198 deaths (28.5%), 13 vegetative state (1.9%), and 349 (50.3%) good recovery and/or moderate disability (Table 7).

On the other hand; the factors associated with unfavorable outcome (GOS II) were as follows: age ≥ 38 years, shock on ICU admission, and pupillary abnormalities. Table 8 features all the factors associated with GOS II.

Discussion

Our study confirms that Road Traffic Injury (RTI) is a major public health concern as it is responsible for an increasing number of morbi-mortality cases worldwide [14, 15].

In our study, despite the severity of trauma in our population, and the non-availability of sophisticated monitoring tools in our ICU, the in-hospital mortality rate (26.9%) seems to be within the world standards [1, 3, 5, 11, 12]. Moreover, the GOS score established within a mean delay at 34 ± 24 months after hospital discharge was as follows: 198 deaths (28.5%), 13 vegetative state (1.9%), and 349 (50.3%) good recovery and/or moderate disability. In our study, we have not excluded any patients from treatment due to neurological deterioration at admission. All patients were treated according to the protocol described.

In Tunisia, almost 13 000 cases of motor-vehicle crash victims are registered per year with a mortality rate of about 1500 according to the National Guard Statistical Data [1, 2]. In the present study, like a previous study published 12 years ago [2], the majority of RTA victims were mainly young in their productive and creative years, with a male preponderance. The reason for this high incidence of RTAs in males lies in their high activity levels and participation in high-risk activities such as aggressive driving/riding and over-speeding without wearing any protective gears. In fact, in our study, the majority of victims were motorcycle riders and/or passengers (40.5%), followed by pedestrians (29.1%). Moreover, the low percentage of female victims can be explained by the fact that women do not ride motorcycles or drive cars as frequently as men do. Besides, women use seatbelts more frequently than men. According to the National Guard Statistical Data, seatbelts are used by only 22% of the motorists, and only 60% of motorbike drivers wear helmets in Tunisia [1, 2].

Moreover, the study period coincides with the Tunisian and Libyan revolutions (January and February 2011, respectively). Over-speeding, poor road and/or vehicle condition, the non-use of helmets by motorcyclists or seatbelts by drivers and passengers, and uncontrolled driving may explain the severity of injury—and thereby the prognosis—in our sample. The above-mentioned reasons may also, partially, explain the high frequency of motor-vehicle crashes as the main etiology of severe head injury in our country.

Various outcome measures are used in prognostic analysis for traumatic brain injury. These include mortality and global outcome measures, e.g., measures of GOS.

Table 5 Factors correlated with in-hospital death in univariate analysis

Parameters	Survivors (%)	Deaths (%)	*p*
Age (years)	29.3 ± 16	38.6 ± 20	< 0.001
SAPS II score	34.3 ± 10.8	46.6 ± 12.6	< 0.001
ISS score	34 ± 9.7	36 ± 9.9	0.010
PRISM score	11.4 ± 6	19.7 ± 9	< 0.001
GCS score	9.3 ± 3.3	7.3 ± 3.2	< 0.001
Shock	78 (15.4)	62 (33.2)	< 0.001
Respiratory distress	42	79	0.034
Isolated TBI (242 patients)	176 (72.7%)	66 (27.3%)	0.65
Glasgow Coma Scale score	9.3 ± 3.3	7.3 ± 3.2	< 0.001
Bilateral mydriasis (32 patients)	13 (2.6)	19 (10.2)	0.001
Anisocoria	94 (18.5)	64 (34.2)	< 0.001
Cerebral edema	129 (25.4)	88 (47)	< 0.001
Subarachnoid hemorrhage	115 (22.9)	102 (55)	< 0.001
Subdural hematoma	210 (41.4)	104 (55.6)	0.001
Cerebral contusion	356 (70.2)	159 (85)	< 0.001
Mass lesion	144 (28.4)	114(61)	< 0.001
Brain herniation	80	81	< 0.001
Prothrombinaemia < 60%	409 (80)	168 (90)	< 0.001
Platelets count < 150,000 cels/ml	132 (26)	89 (47)	< 0.001
Disseminated intravascular coagulation	237	131	< 0.001
Serum glucose > 10 mmol/l	147 (29)	106 (56)	< 0.001

Table 6 Risk factors associated with in-hospital death in trauma brain injury (obtained by logistic regression)

Parameters	*p*	Odds ratio	95% CI
Age > 38 years	< 0.001	3	1.8–5.1
Glasgow coma scale score < 8	0.004	2.2	1.3–3.8
Bilateral mydriasis	0.002	2.3	1.3–3.9
Arterial hypotension	0.001	2.6	1.5–4.6
Hypoxemia	0.014	2.1	1.2-4
Subdural hematoma	0.005	3.2	1.4–7.1
Mass lesion	0.003	2.7	1.4-5
Type IV lesion AMC	0.032	4.4	1.1–17.1
Anemia (Hb < 8.5 g/dl)	0.046	1.8	1–3.1
Glycaemia > 8.5 mmol	< 0.001	3.1	1.8–5

AMC: according to Marshall classification

The measurement of outcome is fundamental to the effective evaluation of the clinical management of any illness. The GOS is widely used for assessing outcome after HI and offers the opportunity to diagnose functional disability or cognitive deficit [11, 16–18]. The mortality rate in our study is a little higher than what is reported in other studies [19–21]. The factors associated with poor outcome (Mortality and GOS II) were age > 38 years, Glasgow coma scale score < 8, subdural hematoma, and the development of Secondary Systemic Insults (respiratory, circulatory, and metabolic).

Age is a strong prognostic factor following traumatic brain injury, with discrepancies defining the critical prognostic age threshold [1–4, 20, 21]. Mortality is higher at the extreme ages of the life [6, 20–23]. Younger patients

Table 7 Outcome of all patients according to the Glasgow outcome scale (GOS)

Classification	GCS on admission	Number of patients	Percentage
Death (GOS 1)	7.3 ± 3.3	198	28.5
Persistent vegetative state (GOS 2)	6.3 ± 2.2	13	1.9
Severe disability (GOS 3)	8.7 ± 3.4	134	19.3
Moderate disability (GOS 4)	9.4 ± 3.4	185	26.7
Good recovery (GOS 5)	10.1 ± 3	164	23.6

GCS Glasgow coma scale

Table 8 Risk factors associated with GOS II (obtained by logistic regression)

Parameters	*p*	Odds ratio	95% CI
Age > 38 years	< 0.001	3.7	2.2–6.1
shock on ICU admission	0.006	2.4	1.3–4.3
Glasgow coma scale score	0.008	1.9	1.2–3.1
Pupillary abnormalities	0.031	1.8	1.1–3
Coma duration > 5 days	0.001	2.4	1.4–3.9
Cerebral edema	0.002	2.2	1.3–3.7
Subdural hematoma	0.002	2.6	1.4–4.9
Diffuse axonal injury	0.001	2.3	1.4–3.7
Anemia (Hb < 11.5 g/dl)	0.029	1.7	1.1–2.8
Glycaemia > 8.5 mmol	< 0.001	2.8	1.8–4.5
Catecholamine's use	0.002	2.1	1.3–3.4

have better chances of survival, tolerate longer periods of coma or decerebration, and have fewer life-threatening complications.

These results were confirmed by a recent retrospective study including 244 patients [22]. In this study, 144 patients were found to have severe TBI. Moderate TBI and mild TBI were found in 38 and 62 patients, respectively [22]. Its authors concluded, in a stepwise manner centered on a 40-year threshold—independent of other prognostic factors, that the older the patient, the less favorable the prognosis, at 6 months. Our study showed a highly significant relationship between age and prognosis. Indeed, age > 38 years was associated with a poor outcome (Mortality and GOS II).

CT scanning provides an objective assessment of the structural damage of the brain following traumatic brain injury. It is also assumed to reflect the seriousness of head injuries and predict the course and outcome. In fact, both the CT classification and individual CT characteristics are important predictors of outcome in TBI [22]. The influence of the type of cerebral lesions on cranial pressure and mortality has been evaluated variously in the literature [1, 2, 5, 24]. However, some lesions appear to carry a poor prognosis. In fact, the prognosis of TBI patients with subdural hematoma (SDH) remains poor, with a high mortality rate despite intensive treatment [25, 26]. Some SDHs are caused by blood leaking from hemorrhagic contusions, and traumatic subarachnoid hemorrhage that extends to the subdural space due to tears of the arachnoid membrane. In other cases, SDHs are caused by rupture of emissary veins. Excessive movement of the brain causes rupture of these vessels, which are attached to the skull. As a consequence, SDH is often associated with other lesions such as contusion and subarachnoid hemorrhage [27]. Thus, the ischemic damage, which is caused by raised intracranial pressure (ICP) producing impaired cerebral perfusion, is an important contributor to the neurological injury associated with SDH. The seriousness of SDH was confirmed in a large study of severe TBI cases (GCS < 8) from seven trauma centers in USA [27]. Subdural hematoma was found to be accompanied by the highest mortality rate (ranging from 40 to 90%), and was frequently associated with other lesions [27, 28]. In our study, the presence of a subdural hematoma also correlated with poor prognosis.

The negative influence of the SSIs to the brain is well documented. In our study, like other series [1, 2, 5, 6], the development of SSIs was associated with a poor prognosis in double analyses. In fact, hypoxemia, shock, anemia, and hyperglycemia were associated with poor outcome. These correlations are well documented in the literature [1, 2, 5, 6].

Despite the retrospective nature of this study, we were able to define some clinical and epidemiological characteristics of traumatic brain injury secondary to road traffic accidents. Moreover, we have revealed simple measures indicative of a poor short-term outcome based on easily measurable clinical, biochemical, and laboratory parameters which can be used at the scene of accident (clinical), and in the emergency department of any hospital with appropriate facilities.

Finally, it is worth mentioning that our studied population was characterized by its severity. In fact, extra-cranial pathology was present in 452 patients (65%), 677 (97.6%) required intubation, mechanical ventilation and sedation on admission, 230 (33%) needed craniotomy and neurosurgery, and 193 (27%) underwent extra-cranial surgery. This can be explained by the severity of road traffic accidents in our country. That is why prevention is highly warranted. Indeed, campaigns to increase seatbelt and helmet use in our country ought to be promoted as frequently as it takes to decrease serious head injuries. It goes without saying that strict regulation and enforcement of speed limits, and appropriate road engineering—with road signs and evidently displayed speed limits to guide road users, can lead to a significant reduction of the number and the severity of RTAs in our country.

Conclusions

TBIs secondary to RTAs are a frequent cause of ICU admission, especially among young adults, and are associated with high morbi-mortality rates.

Factors associated with poor outcome (Mortality and GOS II) were Age > 38 years, Glasgow coma scale score < 8, subdural hematoma, and development of SSIs (respiratory, circulatory, and metabolic). Hence, prevention is highly warranted in our country and elsewhere in the world!

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Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

Availability of data and materials The data sets were analyzed during the current study available from the corresponding author on reasonable request.

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