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# Coagulopathy and transfusion requirements in war related penetrating traumatic brain injury. A single centre study in a French role 3 medical treatment facility in Afghanistan

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ABSTRACT

*Introduction:* Traumatic brain injury associated coagulopathy is frequent, either in isolated traumatic brain injury in civilian practice and in combat traumatic brain injury. In war zone, it is a matter of concern because head and neck are the second most frequent site of wartime casualty burden. Data focusing on transfusion requirements in patients with war related TBI coagulopathy are limited.
*Materials and methods:* A descriptive analysis was conducted of 77 penetrating traumatic brain injuries referred to a French role 3 medical treatment facility in Kabul, Afghanistan, deployed on the Kabul International Airport (KaIA), over a 30 months period.
*Results:* On 77 patients, 23 died during the prehospital phase and were not included in the study. Severe traumatic brain injury represented 50% of patients. Explosions were the most common injury mechanism. Extracranial injuries were present in 72% of patients. Traumatic brain injury coagulopathy was diagnosed in 67% of patients at role 3 admission. Red blood cell units (RBCu) were transfused in 39 (72%) patients, French lyophilized plasma (FLYP) in 41 (76%), and fresh whole blood (FWB) in 17 (31%).
*Conclusion:* The results of this study support previous observations of coagulopathy as a frequent complication of traumatic brain injury. The majority of patients with war related penetrating traumatic brain injury presented with extracranial lesions. Most of them required a high level of transfusion capacity.



## Introduction

Brain injury is associated with tissue factor release which activates the coagulation cascade leading to acute coagulopathy, independently of bleeding [1]. This coagulopathy is a key factor of secondary brain injuries development and is associated with a worse outcome [2,3].

The traumatic brain injury (TBI) associated coagulopathy is frequent. A *meta*-analysis howed an overall prevalence of 33% in TBI [3]. Either isolated TBI in civilian practice and combat TBI have been shown to be associated with an increase risk of coagulopathy [4,5]. In a study comparing TBI patients with non-TBI injuries, admission INR was significantly higher in TBI patients, despite no differences in admission status in term of age, base deficit, systolic or diastolic blood pressure, or hemoglobin [5]. In this study, severity of TBI as Glasgow Coma Scale and head AIS score was independently associated with increased coagulopathy as measured by INR.

In war zone, it is a matter of concern because head and neck are the second site of wounds. A systematic review encompassing a total of 19,750 casualties concerning characteristics of battlefield injury from Iraq and Afghanistan showed that the anatomical distribution of wounds was head and neck in 31% of cases, truncal in 27%, and extremity in 39% [6]. This demonstrated a significant increase in the head and neck region compared with previous wars [7]. Moreover, TBI has been shown to be the most frequent single injury type in a large cohort of explosion episodes during the Iraq and Afghanistan wars [8].

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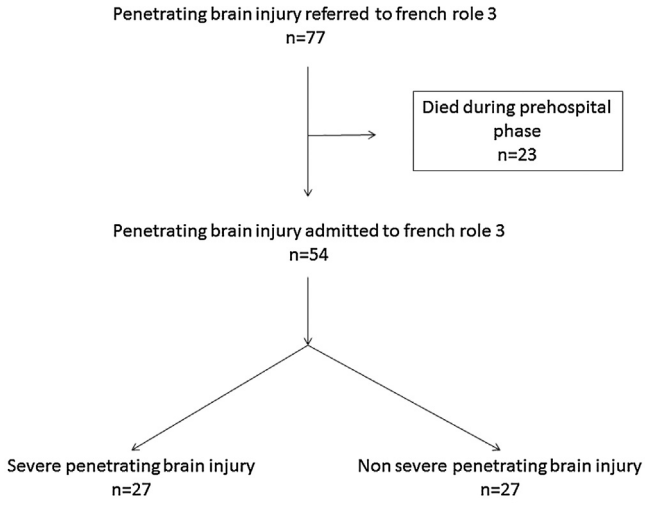


**Fig. 1.** Study's flow chart.

During war time, most of TBI are penetrating. During the 5-year period of the Iraq war, the numbers of penetrating TBIs exceeded closed TBIs by a ratio of 2:1, and during the Afghanistan war, the ratio of penetrating to closed TBIs was approximately 1.3:1 [9].

However, data focusing on transfusion requirements in patients with war related penetrating TBI coagulopathy are limited. Providing critical care including transfusion to TBI patients during war time may be a logistic challenge, that has to be anticipated before deployment. This the reason why we aim to describe the coagulopathy and transfusion requirements in patients with penetrating TBI admitted to a French role 3 medical treatment facility in Afghanistan during a two years period.

## Material and methods

### *Ethical approval*

The study protocol was approved by the Sainte Anne military hospital local ethics committee.

**Table 1**
Patients' neurological, hemodynamic, and respiratory conditions on arrival at hospital.

| | |
|---|---|
| Age | 22.6 (11.8)[a] |
| Gender, n (%) | 43 (80%) |
| Male | 11 (20%) |
| Female | |
| Combatants, n (%) | 20 (27%) |
| Children (age <15) n (%) | 12 (22%) |
| GCS score | 6 (3–12)[b] |
| Blood pressure, mmHg | 82 (24)[a] |
| Heart rate,/min | 99 (27)[a] |
| SpO2, % | 100 (98.7–100)[b] |
| Intubated patients, n (%) | 26 (48%) |
| Temperature, °C | 36.6 (35.6–37.2)[b] |
| Time from injury to hospital, min | 155 (104–250)[b] |
| Mydriasis or anisocory, n (%) | 7 (13%) |
| Associated injuries, n (%) | 39 (72%) |
| Face and neck, n (%) | 15 (28%) |
| Thorax, n (%) | 18 (33%) |
| Abdomen, n (%) | 6 (11%) |
| Peripheric, n (%) | 24 (44%) |
| Admission to a role 2 prior role 3, n(%) | 10 (19%) |

[a] Mean (standard deviation).
[b] Median (interquartile range).

**Table 2**
Evolution of coagulation parameters.

| | at hospital arrival | at ICU admission | *p* value |
|---|---|---|---|
| Hemoglobin, g/dL | 10.8 (3.0) | 10.4 (2.3) | 0.15* |
| PT, % | 54.9 (14.9) | 59.2 (16.0) | 0.23* |
| aPTT, s | 30 (28.5–36) | 31.5 (30–36.8) | 0.66** |
| Platelets, $10^3/mm^3$ | 243 (80) | 219 (109) | 0.25* |
| Fibrinogen, g/L | 2.1 (1.5–2.4) | 2.6 (2.0–3.0) | 0.08** |
| Coagulopathy, n (%) | 36 (67%) | 38 (70%) | 0.48*** |

*paired Student *t*-test
**Wilcoxon Signed Rank Sum test
*** McNemar's Chi-squared test with continuity correction

### *Study location and period*

The study was performed in a French role 3 medical treatment facility in Kabul, Afghanistan, deployed on the Kabul International Airport (KaIA).

The study period was from February 2010 to August 2012.

### *Patients' inclusion*

All patients referred to role 3 for TBI were considered. A total of 77 patients were included in the registry during study period. Twenty three patients died during the pre-hospital phase, and were not included. Finally, 54 patients were analysed.

### *Definitions*

Severe brain injury was defined as a Glasgow Coma Scale (GCS) score inferior to 9. Coagulopathy was defined as an activated partial thromboplastin time (aPTT) >34s, and/or a prothrombin time (PT) >1.5N, and/or a platelets count <100000/$mm^3$ [9].

### *Collected data*

Data were extracted from a prospectively recorded neurosurgical registry. Collected data were patients' age, gender, injury mechanism, evacuation delay, role 2 admission prior to role 3 admission, admission status (Glasgow coma scale, blood pressure, heart rate, temperature, tracheal intubation), associated injury and localization, hemoglobin, prothrombin time, aPTT, platelets count, need for transfusion, number of transfused red blood cell (RBC) unit, french lyophilized plasma (FLYP) unit, whole blood unit. Hospital death and length of stay were also recorded.

**Table 3**
comparison of patients with severe TBI and without severe TBI at admission.

| | Severe TBI n = 27 | Non severe TBI n = 27 | *p* value |
|---|---|---|---|
| Age | 23.4 (11.6) | 21.8 (12.7) | 0.66* |
| Evacuation delay, min | 143 (99–295) | 155 (105–250) | 0.94** |
| Coagulopathy at admission, % | 19 (70) | 17 (63) | 0.77*** |
| Hb level (g/dL) | 10.6 (3.6) | 11 (2.7) | 0.67* |
| Platelets($10^3/mm^3$) | 209 (76) | 271 (83) | 0.01* |
| PT | 50.8 (13.7) | 57.8 (17.8) | 0.15* |
| aPTT (sec) | 32 (28–40) | 20 (28–32) | 0.61** |
| Fibrinogen(g/L) | 1.5 (1.3–2.2) | 2.2 (1.7–2.5) | 0.81** |
| MAP (mmHg) | 82 (29) | 83 (18) | 0.81* |
| SpO2 (%) | 100 (97–100) | 100 (99–100) | 0.16** |
| HR (bpm) | 104 (32) | 92 (22) | 0.13* |
| Associated injuries (n, %) | 19 (70%) | 20 (74%) | 1.00*** |

*Student *t*-test.
**Mann and Whitney test.
***Chi2 test with Yates correction.