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ORIGINAL ARTICLE

Efficacy of Thromboelastography (TEG) in Predicting Acute Trauma-Induced Coagulopathy (ATIC) in Isolated Severe Traumatic Brain Injury (iSTBI)

Venencia Albert[1] · Arulselvi Subramanian[1] · Hara Prasad Pati[2] · Deepak Agrawal[3] · Sanjeev Kumar Bhoi[4]

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Abstract To evaluate the efficacy of point-of-care thromboelastography (TEG) to predict acute trauma-induced coagulopathy (ATIC) in isolated severe TBI (iSTBI). We conducted an observational diagnostic cohort. Patients for whom TEG was performed before blood transfusion were stratified by conventional coagulation tests (CCTs) on admission and classified as "ATIC" (prothrombin time $\geq$ 16.70 s; international normalized ratio $\geq$ 1.27; activated partial thromboplastin time $\geq$ 28.80 s) (n = 24) or "no ATIC" (n = 34). Univariate analysis to compare groups, receiver operating characteristic analysis to establish cut-off and diagnostic validation was done. Fifty-eight patients were included [32(25–45) years; 97% male; GCS 6.3 $\pm$ 1.5]. 41% developed ATIC. Compared to no-ATIC, ATIC group had significantly prolonged κ-time (4.6 vs. 2.5 min; $p = 0.01$) and shortened α-angle (40.2° vs. 56.3°; $p = 0.03$). A cut-off for κ-time $\geq$ 3.7 (AUC 0.68 95% CI 0.54–0.82, specificity 70%, sensitivity 63%) and α angle $\leq$ 48.0 (AUC 0.66, 95% CI 0.51–0.81, specificity 67%, sensitivity 67%) was established. The diagnostic accuracy of this cut-off for identifying ATIC, was 55.6% with sensitivity (81.8%) and specificity (14.3%). TEG may be a clinically sensitive test for identifying the underlying coagulopathy following TBI. However confirmation with CCTs is recommended.

Keywords Thromboelastography · Point-of-care systems · Blood coagulation tests · Traumatic Brain injuries

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✉ Arulselvi Subramanian
arulselvi.jpnatc@gmail.com

Venencia Albert
albertvenencia@gmail.com

Hara Prasad Pati
harappati@yahoo.co.in

Deepak Agrawal
drdeepak@gmail.com

Sanjeev Kumar Bhoi
sanjeevbhoi@gmail.com

[1] Department of Laboratory Medicine, Jai Prakash Narayan Apex Trauma Center, AIIMS, New Delhi, India

[2] Department of Hematology, AIIMS, New Delhi, India

[3] Department of Neurosurgery, Jai Prakash Narayan Apex Trauma Center, AIIMS, New Delhi, India

[4] Department of Emergency Medicine, Jai Prakash Narayan Apex Trauma Center, AIIMS, New Delhi, India

Introduction

Acute trauma-induced coagulopathy (ATIC) is an early onset, endogenous blood clotting impairment in response to the injury itself. There is a bleeding-related loss of coagulation factors and platelets [1] which is further exacerbated by fluid resuscitation, transfusion and localized activation of coagulation [2]. ATIC has been widely associated with severity of injury [3], occurs in 10–34% of patients on arrival in hospital and accounts for 40% of all trauma deaths [4].

Traumatic brain injury (TBI) associated coagulopathy can be seen within 1–4 h after injury, despite the lack of severe bleeding and fluid administration. As reported by a meta-analysis, the overall prevalence of TBI-associated coagulopathy is 32.7% and correlates with high risks of

mortality, increased the need for transfusion and longer intensive care unit and hospital stay [5–8]. Early identification and treatment of early coagulopathy are of prime importance in the management of trauma patients, to facilitate the accurate arrangement of blood products and avoid inessential interventions [9].

Current diagnostic tests for ATIC involve conventional coagulation tests (CCTs) that can take from 30 to 90 min before getting results and fail to quantify clotting beyond the initiation, representing only a small portion of the coagulation system. Since CCTs are performed with platelet-poor plasma, the contribution of platelets to clot strength is not measured.

Thromboelastography (TEG) has been reported to be more sensitive than routine assays for the hypercoagulable state posttraumatic injury [10].

TEG uses whole blood and provides a comprehensive assessment of the clotting mechanism i.e. clot initiation, development, stabilization, and dissolution. TEG parameters derived from a single measurement of whole blood coagulation, are a continuum of blood coagulation along with the interactions between all components. Clinically relevant TEG parameters can be obtained within 10 min to guide early treatments [11].

TEG has emerged as a popular point-of-care device for monitoring ATIC, hypercoagulable tendencies and hyperfibrinolysis.

We previously evaluated the utility of thromboelastography in a trauma care setup, compared to CCTs and observed that TEG can give highly specific results depicting the underlying coagulopathy, following trauma [12].

This prospective study aimed to establish a clinically significant cut-off for TEG parameters to identify ATIC in isolated severe TBI patients and to validate the established TEG definition to determine whether TEG can replace traditional coagulation assays in the emergency department.

Methodology

Study Design and Setting

For this prospective diagnostic study 107 severe TBI patients were screened in the emergency department of our level I trauma care center for a period of 16 months. TBI was diagnosed based on admission head computed tomography (CT) findings. Patients with associated extracranial injuries (n = 22), clinical evidence of brain death (n = 3) and secondary admissions (n = 24) were excluded from the study. 58 isolated severe TBI patients were enrolled in the study based on Glasgow Coma Scale (GCS) of ≤ 8, age group 16–65 years and blood withdrawn for analysis < 12 h of injury, prior to fluid/blood transfusion. One time sampling was done in the emergency department and investigations were performed.

Sample Collection and Analysis

Complete blood count was done on Sysmex XE 2100 hematology analyzer (Sysmex, Kobe, Japan). CCTs was quantified using STA compact automated coagulation analyzer (Diagnostica Stago, France) using sodium citrate-anticoagulated blood. TEG was performed on TEM-A, automated thromboelastometer (Framar Biomedica, Rome) within 2 h of blood collection. All TEG parameters were recorded from a graphical tracing: r-time (1.8–14.2 min), κ-time (0.7–7.3 min), α-angle (27.3°–72.3°), maximum amplitude (32.1–87.9 mm; MA) and lysis at 30 min (LY30, %).

Variables

Clinical demographic data at baseline was recorded. ATIC was defined as INR ≥ 1.27 and/or PT ≥ 16.7 s and/or aPTT ≥ 28.8 s at hospital admission, based on the study by Greuters et al. [13], and our institutional current clinical practice and experience. Patients were classified as 'no-ATIC' and 'ATIC' groups. We have previously established a normal reference value for TEG parameters [14]. Hypocoagulability was defined as prolonged R & κ-time and shortened α-angle & MA. Hyper-coagulability was defined as shortened R & κ-time and prolonged α-angle & MA [15].

Statistical Methods

Statistical analysis was performed for the comparison of TEG parameters between 'no-ATIC' and 'ATIC' groups. Continuous variables were expressed as mean ± standard deviation or median (interquartile range) and categorical variables were represented as frequency (percentage). The analysis was performed using STATA 11.0 statistical software (USA). Univariate analysis of the continuous variables between two groups was done using *t* test/Wilcoxon's rank sum test and categorical data were analyzed using Pearson Chi Square test/Fishers test. Spearman's correlation coefficient (rho) was applied to measure the strength and direction of the association between TEG versus CCTs parameters. A p value of < 0.05 was considered to be statistically significant. Receiver operating characteristic curves (ROC) were constructed to generate optimal cut-offs for significant TEG parameters. Diagnostic accuracy was performed for evaluation of discriminatory power of the established cut-offs on the validation

cohort. Measures of diagnostic accuracy calculated to quantify the ability of TEG to discriminate between and/or predict ATIC were (1) *sensitivity* (percentage of all patients with disease present who have a positive test) and *specificity* (percentage of all patients without disease who have a negative test) (2) *positive and negative predictive value* (PPV, NPV). The predictive value of a test is a measure (%) of the times that the value (positive or negative) is the true value; i.e., the percent of all positive tests that are true positives is PPV and NPV is the proportion of negative test results that are the true negative.

Results

Patient Characteristics

Median (IQR) age of the study cohort was 32 (25–45) years and 96% were males. Twenty four (41%) had ATIC following TBI. Age and sex did not vary between the ATIC and no-ATIC group. The mean time taken from injury to admission was 3 h. GCS was lower in ATIC compared to no-ATIC, yet statistically insignificant [6 (5–7) vs. 7 (5–8); p 0.65] (Table 1).

Computed tomography (CT) scan, revealed various acute traumatic aetiologies, including 19 (33%) with intraventricular hemorrhage and SAH, 24 (42.1%) with SDH, and 6 (10.5%) EDH. The groups are statically comparable for CT abnormalities ($p < 0.05$).

Overall incidence of disseminated intravascular coagulation (DIC) was 3 (5.26%) and 32 (56.1%) had non-overt DIC. Patients who developed ATIC had higher incidence of DIC (p 0.01).

Correlations Between Thromboelastography and Individual Conventional Coagulation Assays

Table 1 presents the comparison of laboratory investigations between ATIC and no-ATIC group.

All TEG parameters were within normal range except MA (reduced absolute clot strength) following TBI. In comparison to no-ATIC, ATIC patients had prolonged r-time [5.7 (3.8–8.2) vs. 6.8 (4.3–10.0); $p = 0.49$], significantly prolonged κ-time [2.5 (1.5–3.7) vs. 4.6 (1.8–8.1); $p = 0.01$], significantly shortened α-angle [56.3 (43.6–63.3) vs. 40.2 (27.3–52.9); $p = 0.03$].

In ATIC group 45% were hypercoagulable and 37% were hypocoagulable based on TEG results. Based on CCTs 16.7% patients had established coagulopathy in the ATIC group and none in no-ATIC. However, TEG identified 30% patients as hypercoagulable and 36% as hypocoagulable in the no-ATIC group (Table 1).

Weak and insignificant association between the individual CCTs and TEG parameters and a significant negative moderate association between fibrinogen levels and κ-time ($r = -0.4$; $p = 0.03$) were observed.

A cut-off of ≥ 3.7 s (sensitivity 63% and specificity 70%) for κ-time and $\leq 48.0°$ (sensitivity 67% and specificity 67%) for α-angle was established for identification of ATIC in isolated severe TBI patients by TEG (Fig. 1).

Validation Cohort

Median (IQR) age was 35 (29–42) years, 87% were male and mean time from injury to admission was 2 h. On applying the CCT based definition 24 (61.5%) patients were classified as 'ATIC' and 15 (33.5%) as 'no-ATIC'.

Patients were also stratified as 'high' and 'low' based on the cut-offs established for κ-time and α-angle.

On comparing the identification of ATIC by CCTs by TEG cut-offs we observed 20 patients as true positive, 12 were false positive, whereas three were true negative and four were false negative; sensitivity 63% (95% CI 45.25–77.07), specificity 43% (95% CI 15.82–74.95), 83% (95% CI 64.15–93.32) PPV and 20% (95% CI 7.047–45.19) NPV was observed. The efficiency of a test result was observed to be 59% (95% CI 43.42–72.92) (Table 2).

Discussion

While the accuracy of thromboelastography is known in other settings [16], the aim of the present study was to know if the TEG could be used to accurately identify ATIC with isolated severe TBI.

Twenty four (41%) isolated severe TBI patients were identified as ATIC based on the CCTs in this study. Our results demonstrated that brain injury affects the overall clot stability and quality (shortened MA) in all patients, reflecting poor platelet number, platelet dysfunction and the interaction with fibrin, indicating thrombocytopenia and hyperfibrinogenemia. Clot initiation time (r-time), the speed at which fibrin build up and cross-linking takes place (κ-time and α-angle), were within normal range. Significant prolongation in fibrin formation (κ-time) and shortening of clot amplification (α angle) was seen in patients with ATIC compared to no-ATIC, suggesting an early state of hypo-coagulability following TBI. The clinical cut-off for κ-time ≥ 3.7 min and α angle $\leq 48.0°$, to detect ATIC with a high diagnostic sensitivity of 63% and moderate specificity of 43% was established.

Coagulopathy is an independent predictor of unfavorable outcome in isolated TBI [6]. In the pre-intervention

Table 1 Comparison of demographic and laboratory data between patients with ATIC and no-ATIC group

Clinical parameters	No ATIC (n = 34)	ATIC (n = 24)	*p* value
Age (years)	35 (25–40)	30.5 (25.5–47.5)	0.37
Sex[b]			
Male	34 (100)	22 (91.6)	0.17
Female	0	2 (8.4)	
Mode of injury[b]			
RTA	20 (60.6)	14 (58.4)	0.77
Fall	5 (15.2)	5 (20.8)	
Assault	2 (6.1)	0	
Miscellaneous	6 (54.5)	5 (20.8)	
Time taken from injury to admission (h)[a]	3.0 (1.5–5.5)	3.0 (2.0–4.5)	0.65
Mechanical ventilation[b]			
No	14 (42.4)	12 (50.0)	0.60
Yes	19 (61.3)	12 (50.0)	
Systolic BP (mm/Hg)	142.3 ± 28.65	132.5 ± 24.06	0.18
Glasgow coma scale	7 (5–8)	6 (5–7)	0.69
Haemoglobin (g/dL)	13.3 ± 2.15	12.3 ± 2.25	0.07
Red blood cell count (× 10*6 cumm)	4.5 ± 0.78	4.1 ± 0.83	0.07
White blood cell count (× 10*3/cumm)[a]	12.9 (10.3–16.6)	12.0 (9.8–15.8)	0.65
Platelet count (× 10*3/cumm)[a]	195.0 (142.0–244.0)	170.0 (125.0–225.5)	0.27
Prothrombin time (s)	13.7 ± 0.9	17.6 ± 4.3	< 0.0001
Activated partial thromboplastin time (s)[a]	24.5 (22.2–25.7)	30.8 (29.5–36.6)	< 0.0001
International normalized ratio[a]	1.0 (0.9–1.1)	1.3 (1.2–1.5)	< 0.0001
D-Dimer (μg/mL)[b]			
< 0.5	12 (66.7)	6 (50.0)	0.42
1–4	5 (27.8)	3 (25.0)	
4–8	1 (5.6)	3 (25.)	
Coagulopathy[b]			
No	**33 (100.0)**	**20 (83.3)**	**0.02**
Yes	**0**	**4 (16.7)**	
R-time (min)[a]	5.7 (3.8–8.2)	6.8 (4.3–10.0)	0.49
κ-time (min)[a]	**2.5 (1.5–3.7)**	**4.6 (1.8–8.1)**	**0.01**
α-angle (°)[a]	**56.3 (43.6–63.3)**	**40.2 (27.3–52.9)**	**0.03**
Maximum amplitude (mm)[a]	30.9 (0–61.4)	29 (12.9–50)	0.49
Lysis 30[a]	0 (0–9.6)	0 (0.8–35)	0.95
TEG coagulation status[b]			
Hypo-coagulable	12 (36.4)	9 (37.5)	0.36
Normocoagulable	11 (33.3)	4 (16.7)	
Hyper-coagulable	10 (30.3)	11 (45.8)	

Continuous variables are reported as mean ± SD or [a]median (interquartile range); [b]categorical variables are reported in terms of frequency (percentage). Bold font depicts significant *p* value

RTA road traffic accident, *BP* blood pressure

stage a transient hypercoagulable state exists, that progresses to a hypercoagulable state in the later stages [17].

Previous studies have used TEG to identify an early hypo-coagulable state after TBI [18, 19]. Massaro et al., identified and reported high MA values compared to control at 24 h after TBI (GCS < 12) [20]. TEG has also been suggested as a screening and monitoring device for deep venous thrombosis and pulmonary embolism following brain injury [21]. Whereas standard plasma-based CCTs fail to identify a hypercoagulable state in vitro and provide a functional assessment of hemostatic mechanisms in vivo [22].

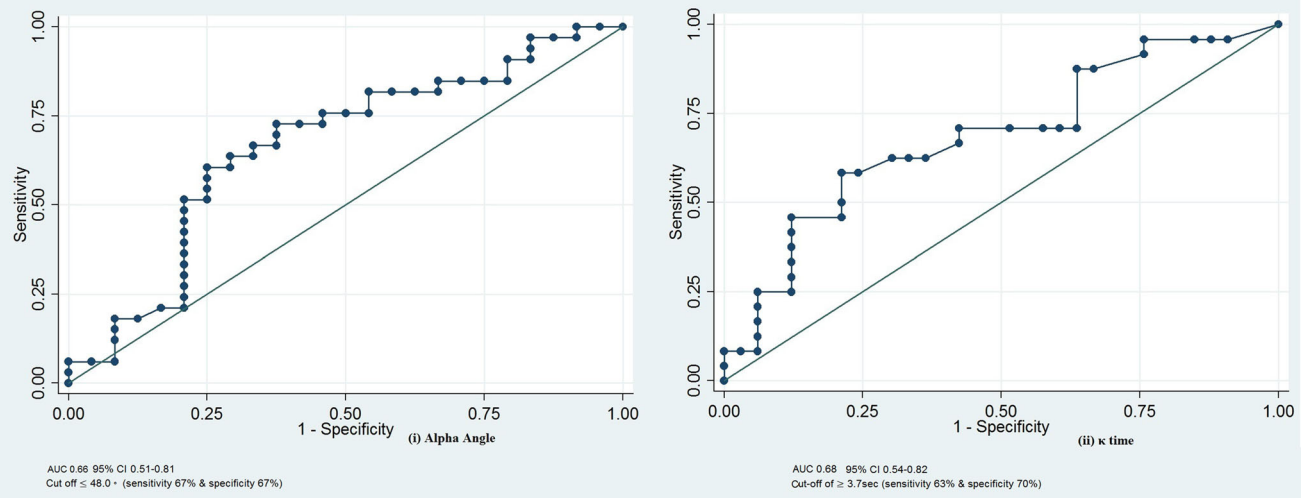

Fig. 1 Receiver operating characteristics curve for thromboelastograph parameters: κ-time and α angle

Table 2 Diagnostic performance of point-of-care thromboelastography to predict acute trauma induced coagulopathy

Characteristics (95% CI)	K-time ≥ 3.7 min	α angle ≤ 48.0°	K-time ≥ 3.7 min, α angle ≤ 48.0°
Sensitivity	64% (45.83–79.29)	62% (44–77.31)	63% (45.25–77.07)
Specificity	46% (21.27–71.99)	40% (16.82–68.73)	43% (15.82–74.95)
Positive predictive value	75% (55.1–88)	75% (55.1–88)	83% (64.15–93.32)
Negative predictive value	33% (43.42–72.92)	27% (10.9–51.95)	20% (7.047–45.19)
Diagnostic accuracy	59% (43.42–72.92)	56% (40.98–70.7)	59% (43.42–72.92)

The difference in the sensitivity between CCTs and TEG in trauma set up, have been previously reported [23, 24]. Doran's study reported that TEG identified coagulopathy in 64% compared to only 10% identified by abnormal CCTs [24]. Similarly, Jeger et al. [23] reported a moderate correlation of CCTs with rapid thromboelastography (r-TEG), they established a cut-off for α-angle 74.7° to identify transfusion requirement with high sensitivity (84%) than CCTs but less specificity (57%).

TEG is more suited for the identification and management of TIC, owing to the cornucopia of information it provides regarding the physical properties of the impaired clotting mechanism and its high diagnostic sensitivity. However, we observed low specificity and statistically weak correlations with CCTs which could be due to two reasons in our study, as TEG was performed (1) using citrated instead of non-citrated whole blood in the study and (2) without any additional coagulation activator. Kashuk and colleagues [25] reported a higher correlation between non-citrated TEG values with CCTs compared to citrated TEG results. Suggesting that non-citrated samples may be more accurate. However, they also stated that comparing a static (conventional) with a dynamic test (TEG) is obscure.

Another study by Ågren and Wikman [26] showed poor agreement between CCTs and TEG in patients with significant risk of bleeding undergoing major surgery.

Similarly, Kornblith et al., in 2014 reported suboptimal correlations between k-time and α-angle and fibrinogen levels following trauma [27].

Cotton et al., in 2011 demonstrated that r-TEG values for activated clotting time (ACT), k-time and r-time correlated strongly with PT and aPTT, whereas α-angle and MA showed moderate correlation with PT, aPTT and platelet count in relation to coagulopathy [28]. In another study, the correlations of ACT and r-time were moderate with PT and INR and strong with aPTT following TBI [29]. Another study in 2005 a higher prevalence of coagulopathy by r-TEG alpha and MA was observed in isolated TBI patients, however, no difference in the prevalence of coagulopathy by CCTs values was detected. They report that TBI coagulopathy is contingent upon platelet and fibrinogen interaction [30].

Although in this study we did not see the association of TEG identified coagulopathy with patient outcome and

transfusion requirements, recent literature [31] suggests that TEG-guided identification of early coagulation defects and can prompt clinicians on the optimum transfusion strategy. Currently the TEG-guided protocol that are being used, appear to improve survival while reducing overall product use.

Future evidence based TEG studies for diagnosing trauma coagulation defects in order to direct individualized blood products resuscitation is recommended.

Conclusion

Despite its small sample size, the study has clearly demonstrated 41% patients with isolated severe TBI develop a hypo-coagulable state immediately following TBI. These patients have significant impairment of clot stability and quality, as identified by thromboelastography. Although the efficacy of classical coagulation test has been well established, in emergency trauma situations that require immediate corrective measures. TEG's ability to assess the spectrum of different coagulopathies in whole blood renders it to be ideal for rapidly identifying ATIC and transfusion guidance. However in our study, we observed low specificity, therefore TEG cannot replace the conventional coagulation assays for identifying ATIC but may potentially be clinically sensitive in depicting the underlying coagulopathy following brain trauma.

Compliance with Ethical Standards

Conflict of interest All authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the All India Institute of Medical Sciences Ethics Committee [Ref. IESC/T-431/30.11.2012,OT-1/27.01.2016] and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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

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