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A predictive model of early mortality in trauma patients

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Abstract

BACKGROUND: Rapid thrombelastography (rTEG) is a real-time whole-blood viscoelastic coagulation assay. We hypothesized that admission rTEG and clinical data are independent predictors of trauma-related mortality.

METHODS: Prospective observational data (patient demographics, admission vital signs, laboratory studies, and injury characteristics) from trauma patients enrolled within 6 hours of injury were collected. Mann–Whitney *U* test and analysis of variance test assessed significance ($P \leq .05$). Logistic regression analyses determined the association of the studied variables with 24-hour mortality.

RESULTS: Seven hundred ninety-five trauma patients were enrolled, of which 55 died within 24 hours of admission. Admission variables which independently predicted 24-hour mortality were as follows: Glasgow Coma Scale ≤ 8, hemoglobin <11 g/dL, international normalized ratio >1.5, Ly30 $>8\%$, and penetrating injury ($P < .05$). This 5-variable model's area under the receiver operator characteristic curve was .88. The Hosmer–Lemeshow goodness-of-fit test was .90.

CONCLUSIONS: This 5-variable model provides a rapid prediction of 24-hour mortality. The inclusion of rTEG Ly30 demonstrates the association of fibrinolysis with outcome and may support the early use of antifibrinolytic therapies.

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The accuracy of mortality predictive models has paralleled the interpretation of anatomic injury, physiologic components, and laboratory tests. In the 1970s, the American Medical Association's Committee on Medical Aspects of Automotive Safety developed a predictive model utilizing anatomic injuries sustained during motor vehicle collisions.[1,2] The Abbreviated Injury Scale was based on body region and graded injuries on a 1 (minor) through 6 (lethal) scale. This pioneering model was limited and under estimated mortality in the elderly, head injured, and patients with multiple injuries in a single body region or injuries within multiple body regions. The Injury Severity Score (ISS) and new ISS (NISS) overcame these limitations by addressing multiple simultaneous injuries within single and multiple body regions.[3,4] Both the Abbreviated Injury Scale and ISS were primarily developed to predict outcomes in blunt trauma.

The evolution of these early prediction models diverged from solely assessing anatomic injuries to addressing physiologic characteristics, or a combination of the two. The introduction of physiologic parameter models such as

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the Trauma Score (TS), revised TS (rTS), and Physiologic Trauma Score (PTS) accounted for patient characteristics which change secondary to trauma.[5–7] The individual's response and physiologic reserve became factors in mortality prediction. Unlike an anatomic injury, physiologic response is unique to the patient. Inclusion of these measures has improved the accuracy of mortality prediction models.[8] More recently, the Trauma and Injury Severity Score (TRISS), a weighted scoring system utilizing rTS, ISS, and age, has been extensively used as a mortality prediction tool and quality assurance measure.[9,10]

Because of processing latency, the integration of laboratory results into predictive modeling has been difficult. In a trauma setting, clinicians often act upon information which is several hours old. Real-time information more accurately directs patient care. Expedient laboratory tests such as rapid thrombelastography (rTEG), a 5-variable viscoelastic measure of coagulation, is becoming a mainstream tool. rTEG has been able to provide early coagulation assessment and to direct care of trauma patients.[11–13] Given these findings, we proposed that rTEG values and known physiologic parameters can predict 24-hour trauma mortality.

Methods

Prospective observational data from 3 level-1 trauma centers (Oregon Health & Science University, San Francisco General Hospital, and Hermann Memorial Hospital) were obtained. Institutional research board approval was granted at each site. Major trauma patients transported directly to the emergency room meeting the highest level of activation at each of the 3 centers with one or more physiologic or anatomic derangements were eligible (Table 1). Prisoner status, age <18 years, history of prehospital cardio-pulmonary resuscitation, and burns >20% body surface area were excluded.

Table 1 Physiologic and anatomic inclusion criteria

Physiologic	Anatomic
GCS <10	Penetrating torso trauma
SBP <90 mmHg	Groin/neck injury
RR <10 or >29 breaths/min	Amputation proximal to wrist/ankle
HR >120 beats/min	Uncontrolled external hemorrhage
BE < −6 mEq/L	≥2 Long bone fractures
Intubated	Pelvic fracture
	Paraplegia/quadriplegia
	<20% TBSA burn

Major trauma patients meeting the highest level of trauma activation and sustaining one or more physiologic or anatomic derangements were included in the study.

BE = base excess; GCS = Glasgow Coma Scale; HR = heart rate; RR = respiratory rate; SBP = systolic blood pressure; TBSA = total body surface area.

Patient demographic data – age, sex, systolic blood pressure (SBP), heart rate (HR), respiratory rate (RR), and Glasgow Coma Scale (GCS) – were obtained. Injury data – mechanism (blunt vs penetrating), ISS, and 24-hour mortality data – were collected. Conventional coagulation parameters (CCP) – prothrombin (PT), partial thromboplastin (PTT), international normalized ratio (INR), fibrinogen (FIB), and platelet count (PLT) – and rTEG data – activated clotting time (ACT), R - time to onset of clotting, K - rate of clot formation, alpha angle - rate of fibrin cross-linking, MA - maximum amplitude or clot strength, and Ly30 - degree of clot lysis 30 minutes after maximal clot strength was achieved - were also acquired. All CCP, rTEG, and hemoglobin (Hgb) labs were drawn upon admission to the emergency department. Collection protocols and lab parameters were standardized among the centers. In accordance with protocol, the TEG machines underwent daily quality control testing with 2 levels of controls.

All data were analyzed to determine if they were normally distributed. All nonparametric data were analyzed using Mann–Whitney U tests and all parametric data were analyzed using analysis of variance tests. Significance was set at $P \leq .05$. Selected data were dichotomized based on their known association with mortality and the manufacturer's coagulopathy parameters (Table 2).[14–16] Variables that were found to be predictive of mortality in univariate analysis were analyzed using a backward conditional logistic regression to determine their independent association with 24-hour mortality. Because of incomplete data, those variables that were found to be independent predictors of 24-hour mortality were reanalyzed using a logistic regression. The model's predictive ability was assessed using the Hosmer–Lemeshow (HL) goodness-of-fit statistic and the area under the receiver operator characteristic (AUROC) curve.[17–19]

Results

There were 795 patients in the study (Table 3). Seven hundred forty patients survived for 24 hours and 55 patients did not. Mortality was associated with a lower GCS and SBP, and a higher ISS. Seventy-four patients did not have a mechanism of injury recorded. Penetrating injury (n = 192) was associated with a higher mortality rate (11% vs 6%; P = .02) and a lower ISS (14 [interquartile range: 5,15] vs 22 [interquartile range: 14,33]; P < .01) as compared with blunt injury (n = 529).

Admission laboratory studies were available on every trauma patient. Twenty-four hour mortality was associated with a larger BE and a lower Hgb and PLT (Table 3). PT, PTT, and INR were prolonged and FIB was decreased in the mortality group. rTEG ACT, R, and K were longer, and the alpha angle and MA were lower in the mortality group. Ly30 was greater in the mortality group, and a larger percentage of patients who died had an Ly30 >8% which is

Table 2 Dichotomous variables for logistic regression

Physiologic	CCP	rTEG
GCS $\leq$8	PT $>$14 sec	ACT $>$118 sec
SBP $\leq$90 mmHg	PTT $>$35 sec	Alpha $<$64°
Hgb $<$11 g/dL	INR $>$1.5	MA $<$52 mm
BE $<-$6 mEq/L	FIB $<$200 mg/dL	Ly30 $>$8 mm
	PLT $<150 \times 10^3/mm^3$	

ACT = activated clotting time; BE = base excess; CCP = conventional coagulation parameters; FIB = fibrinogen; GCS = Glasgow Coma Scale; Hgb = hemoglobin; INR = international normalized ratio; Ly30 = degree of clot lysis 30 minutes after maximal clot strength; MA = maximum amplitude; PLT = platelet count; PT = prothrombin; PTT = partial thromboplastin; rTEG = rapid thrombelastography; SBP = systolic blood pressure.

the limit of the normal range as defined by the manufacturer (28% [n = 12] vs 3% [n = 21]; $P < .01$).

When the demographic, hematologic, and CCP data associated with mortality were analyzed in a backward conditional logistic regression, GCS, SBP, INR, and penetrating injury were found to be independent predictors of mortality (Table 4). Using these 4 variables, the AUROC was .86 and the HL goodness-of-fit was .37 (Fig. 1). When the rTEG parameters were included in an identical analysis, GCS, Hgb, INR, Ly30, and penetrating mechanism of injury were independent predictors of mortality. Using these 5 variables, the AUROC was .88 and the HL goodness-of-fit was .90.

Comments

We developed a 5-variable 24-hour mortality prediction model based on biochemical and physiologic data available within 30 minutes of admission to the resuscitation bay. The inclusion of Ly30 demonstrates the association of fibrinolysis with mortality suggesting a potential benefit of administering tranexamic acid (TXA) during resuscitation. The 4-variable model, which did not include rTEG data, produced a similar AUROC profile, but its ability to predict the observed results was poorly calibrated and significantly inferior based on the HL goodness-of-fit. This discrepancy may have been a product of meeting the fibrinolysis threshold, which was seen significantly more frequently in the mortality group.

The ISS was validated in 1974 as a predictor of mortality after motor vehicle trauma.[3] Its limitation, not accounting for multiple injuries to single body region, was overcome by the NISS.[4] In a 4-year study at 2 level-1 trauma centers, the NISS possessed a larger AUROC and a better goodness-of-fit as compared to ISS calculations on the same population. Conversion to a NISS system should have been seamless; however, it has not been widely employed.

Table 3 Demographic and admission laboratory data for 795 trauma patients

	Survived (n = 740)	Died (n = 55)	P value
Age (years old)	38 (26, 52)	39 (25, 61)	.39
GCS	15 (3, 15)	3 (3, 3)	$<$.01
SBP (mmHg)	130 (108, 148)	109 (78, 137)	$<$.01
HR (beats per minute)	96 (81, 115)	101 (76, 129)	.4
RR (respirations per minute)	20 (16, 24)	18 (16, 22)	.23
ISS	19 (10, 29)	28 (25, 38)	$<$.01
Base excess (mEq/L)	$-$2.7 ($-$6.0, .5)	$-$6.6 ($-$12.2, 3.0)	$<$.01
Hgb (mg/dL)	13.6 (12.2, 14.8)	11.0 (9.7, 13.2)	$<$.01
PLT ($\times 10^6$/L)	235 (190, 284)	178 (223, 143)	$<$.01
PT (sec)	13.9 (13.2, 14.9)	17.2 (15.7, 20.4)	$<$.01
PTT (sec)	27.2 (24.6, 30.4)	38.3 (31.9, 51.9)	$<$.01
INR	1.1 (1.0, 1.2)	1.4 (1.3, 1.7)	$<$.01
Fibrinogen (mg/dL)	264 (215, 317)	199 (128, 297)	$<$.01
ACT (sec)	113 (105, 128)	121 (113, 144)	$<$.01
R (sec)	42 (36, 48)	48 (42, 60)	$<$.01
Alpha (degrees)	74 (70, 77.0)	69 (55, 73)	$<$.01
K (sec)	84 (66, 108)	114 (75, 192)	$<$.01
MA (mm)	63 (59, 67)	58 (49, 63)	$<$.01
Ly30 (%)	1.1 (.2, 2.4)	2.1 (.2, 15)	$<$.01

$P \leq .05$.

GCS = Glasgow Coma Scale; Hgb = hemoglobin; HR = heart rate; INR = international normalized ratio; ISS = Injury Severity Score; PLT = platelet count; PT = prothrombin; PTT = partial thromboplastin; ACT = activated clotting time; Alpha = rate of fibrin cross-linking; K = rate of clot formation; Ly30 = degree of clot lysis 30 minutes after maximal clot strength; MA = maximum amplitude; R = time to onset of clotting; RR = respiratory rate; SBP = systolic blood pressure.

Table 4 Backward conditional regression models using solely conventional coagulation parameters and with the inclusion of rapid thrombelastography parameters

	OR (95% CI)	*P* value	HL
Model with CCP			
GCS ≤9	11.1 (4.0–30.8)	<.01	.37
SBP <100 (mmHg)	2.5 (1.0–5.9)	.05	
INR >1.5	9.6 (4.1–22.6)	<.01	
Penetrating	5.4 (2.4–12.0)	<.01	
Model with rTEG			
GCS ≤9	8.0 (2.8–22.7)	<.01	.90
INR >1.5	7.4 (3.0–18.3)	<.01	
Hgb <11 (mg/dL)	4.0 (1.7–9.1)	<.01	
Ly30 >8 (%)	3.7 (1.2–12.1)	.03	
Penetrating	5.0 (2.2–11.4)	<.01	

P ≤ .05.

CCP = conventional coagulation parameters; CI = confidence interval; GCS = Glasgow Coma Scale; Hgb = hemoglobin; HL = Hosmer–Lemeshow; INR = international normalized ratio; Ly30 = degree of clot lysis 30 minutes after maximal clot strength; OR = odds ratio.

These early models were solely based on anatomic trauma data; the inclusion of physiologic parameters increased their predictive ability. Derived from the Triage Index, a prehospital assessment tool, Champion's TS utilized the following physiologic parameters: SBP, RR, capillary refill, respiratory expansion, and GCS.[5,20] These readily measurable parameters made the TS a desirable tool; however, capillary refill and respiratory expansion were difficult to assess in the field. The exclusion of these variables lead to the rTS, a 3-variable model which was easier to apply, and gave a larger weighting to the GCS score.[6] This resulted in a more accurate characterization of head injured patients. The rTS was the basis for the TRISS, a comprehensive model which also incorporates ISS and age.[9] This methodology offered a consistent mortality prediction model and was used in the Major Trauma Outcome Study as the standard for patient and treatment evaluation measures.[10] The TRISS, however, was restricted by the latency to calculate ISS upon admission. The ISS was not known until an operative intervention or autopsy was performed.

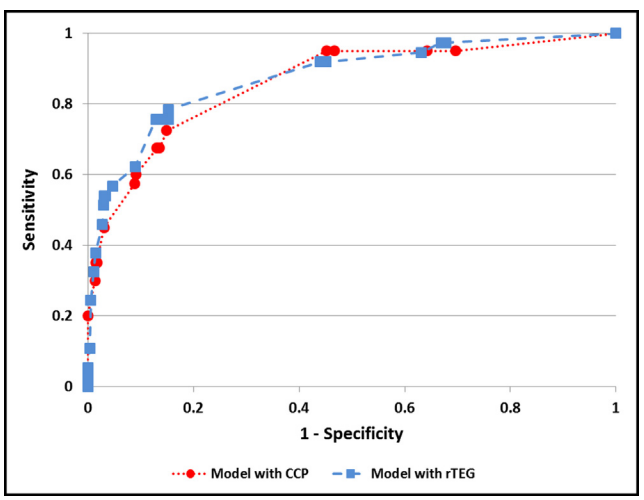

Figure 1 Receiver operator curves for the 4-variable and 5-variable models.

The expediency of admission laboratory work resulted in the development of the PTS, a model which combined the systemic inflammatory response syndrome score, GCS, and age.[7] The presence of systemic inflammatory response syndrome has been found to be an independent predictor of mortality and intensive care unit admission.[21,22] In a logistic regression analysis using data from over 9,000 trauma patients, no difference was seen between PTS and TRISS.[7] Its feasibility, ease of calculation, and equivalence to TRISS made PTS an attractive mortality model.

Our institution recently published a simple model for massive transfusion (MT).[23] This study was a retrospective cohort analysis of 558 combat casualties received at 2 combat support hospitals in Iraq. Two hundred forty-seven patients received an MT, and 311 did not. Patient variables associated with an MT underwent a stepwise logistic regression. Those found to be independent predictors of an MT, the Schreiber Score (SS), were the following: hemoglobin ≤11 g/dL, INR >1.5, and penetrating mechanism of injury. This simple scoring system is a validated tool for early goal-directed resuscitation in trauma patients.[24]

The SS, combined with Ly30 and GCS, is our 24-hour mortality prediction model. This extended Schreiber Score (eSS) segues from an MT to a mortality model. Similar to TRISS and rTS, the inclusion of GCS may allow for an increased accountability for head injured patients. The inclusion of Ly30, a measure of fibrinolysis, underscores the utility of TEG, and the potential benefit of TXA, a lysine analogue which inhibits fibrinolysis by competitively binding to plasminogen and plasmin, thus preventing degradation of fibrin. TXA has been associated with decreased mortality in civilian and military settings and has become a front line agent for battle field injuries.[25,26] The Clinical Randomization of an Antifibrinolytic in Significant Hemorrhage-2 trial was a prospective study

involving over 20,000 civilian trauma patients admitted to 274 hospitals in 40 countries.[25] Patients who received TXA within the 1st hour after trauma experienced decreased mortality due to hemorrhage (TXA [5.3%] vs placebo [7.7%]; $P < .0001$). This benefit was also noted for those patients who received TXA within 1 to 3 hours of their injuries (TXA [4.8%] vs placebo [6.1%]; $P = .03$). This landmark investigation was limited by the use of clinical findings as inclusion criteria, study patients who may have not been actively bleeding, and a large study population may have led to a type II error. The Military Application of Tranexamic Acid in Trauma Emergency Resuscitation study was a retrospective observational study involving 896 combat traumas who received at least 1 unit of packed red blood cells.[26] Those receiving TXA (17.4%) had a lower mortality rate than those who did not (23.9%; $P = .03$). Even though Military Application of Tranexamic Acid in Trauma Emergency Resuscitation study was limited by missing 30-day outcome data from host nation patients and the inability to discern the cause or timing of death, it was able to demonstrate improvement of coagulopathy and survival in those receiving TXA. Ly30, TXA, and mortality are intimately connected. The SS evolving into the eSS represents a seamless transition in patient outcome prediction and a continuum of care.

There were limitations to our study. This was a prospective study performed at 3 geographically separated institutions. The patient populations, injuries seen, and practice patterns may have varied. A patient's medical history was not known at the time of enrollment, therefore medications or pre-existing conditions which could affect coagulation were not known. There was no accountability or standardization for prehospital or hospital interventions. Centralized processing of data was not performed, therefore some patient data were incomplete. Finally, the study variables' covariance between each other may have introduced a level of bias into the regression analysis.

We have developed a novel 5-variable 24-hour mortality prediction model. It incorporates our previous MT model, SS, with GCS, a variable which increased the previous models' predictive ability by accounting for head injuries and severe shock, and Ly30, a measure of thrombolysis. Our model demonstrates a high level of functionality by exploiting variables which are readily available within the trauma bay and has the potential to be used as a real-time predictive score in the treatment of trauma patients.

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Discussion

Matthew J. Martin, M.D.: One of the most difficult tasks in trauma care, and in all of medicine, is being able to separate concepts that meaningfully change the way we do business from transient fads or trends that eventually fizzle out and die. This paper touches on several current trends that are being widely implemented and discussed, but are still awaiting that ultimate test of time: these include damage control resuscitation, tranexamic acid, and the use of thromboelastography (TEG) in the initial trauma evaluation. For the purposes of full disclosure, I will admit that I am the senior author on another paper being presented at this meeting that focuses on the utility of rotational thromboelastometry, or ROTEM, which is essentially a TEG with a European accent.

I have several comments and questions for the authors, and as always I look forward to Dr. Schreiber's responses and insights:

1. The first comment is really a philosophical question - although we talk about various scoring systems for predicting everything from the need for blood transfusion to whether the patient will ultimately live or die, does anyone actually use any of these scoring systems in the emergent clinical setting? I take comfort in the fact that these incredibly complex and multi-variate decision processes still require a human brain capable of considering both hard data and the more nebulous "gestalt" from an injured patient. Are you using this scoring system at OHSU, and if so, how?

2. A major criticism of scoring systems such as ISS and TRISS is that they are not useful or even available at the initial evaluation, when hard decisions need to be made. Physiologic variables like heart rate and blood pressure are immediately available, and with modern point of care devices most lab values can be obtained within minutes of patient arrival. How useful is a test like LY30 that requires 30 minutes in addition to the time required to obtain the sample, start the test, and report the result?

3. As your data shows, there is really no difference in the area under the ROC curves for the 4-variable model versus the model with additional TEG data. What is the benefit of adding the LY30, or other TEG measures, in terms of mortality prediction? How does adding this variable significantly improve the goodness of fit of the model, but not the predictive ability?

4. Finally, it must be stated that the jump from the study results to the conclusion that early TXA use is indicated is entirely speculative. The CRASH-2 and MATTERS studies did not use any measure of fibrinolysis as an indication for TXA administration. Is an elevated LY30 an absolute indication for TXA, and more importantly, is a normal LY30 enough reason to withhold giving TXA?

Only time will tell whether the use of TEG or ROTEM in trauma resuscitation becomes a standard of care or a passing fad. The most important factor in making that determination will be the continued collection and reporting of robust data and outcomes as demonstrated by Dr. Schreiber and colleagues at Oregon Health & Science University.