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Targeting Resuscitation to Normalization of Coagulating Status: Hyper and Hypocoagulability After Severe Injury are Both Associated with Increased Mortality

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Abstract

Introduction—The prevalence and impact of hypercoagulability(hypo) in severely injured patients early after injury remains unclear. We hypothesize that the predominant phenotype of postinjury coagulopathy is hypercoagulability(hyper) and it is associated with increased mortality.

Material and Methods—Blood samples from 141 healthy volunteers assayed with thrombelastography(TEG) were used to identify thresholds of hypo and hypercoagulability(above 95th/below the 5thpercentile) in four TEG indices. These cutoffs were subsequently evaluated in severely injured trauma patients(ISS>15) from two level 1 trauma centers.

Results—2,540 patients with a median ISS of 25 were analyzed. Normal TEG was present in 36% of patients. Hyper was found in 38% of patients, with mixed(11%) and hypo(15%) being less common. Compared to normal coagulation patients and after controlling for age, sex, blood pressure, and injury hyper(0.013), mixed($p<0.001$) and hypo($p<0.001$) were all independent predictors of mortality.

Conclusion—These data support the ongoing need for goal directed resuscitation in trauma patients, it appears the optimal resuscitation strategy should be targeted towards normalization of coagulation status as both early hyper and hypocoagulability are associated with increased mortality.

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Keywords

trauma induced coagulopathy; fibrinolysis; massive transfusion; trauma; hypercoagulability

Introduction

The fear of hypocoagulability(hypo) and treating patients aggressively with pre-emptive hemostatic blood products originated from the 1980's (1) where the early use of plasma was advocated in selective patients. This association of adverse outcomes and coagulopathy was further suggested by work in the early 2000's which identified an elevated INR was prevalent in roughly a quarter of high level trauma activations and associated with a four-fold increase in mortality (2). Subsequently, high ratios of plasma and platelets to red blood cells (RBC) transfusions have been associated with improved survival in retrospective analyses (3, 4). While randomization of patients to 1:1:1 of platelets: plasma: RBCs improved early (0–3hours) and hemorrhage-related mortality, it was not superior to 1:1:2 in improving 30-day mortality (5).

With the use of viscoelastic assays in trauma, distinct phenotypes of coagulopathy have been appreciated (6) and suggests that personalized resuscitation efforts may have benefit. A retrospective analysis suggests that thrombelastography (TEG) based resuscitation is superior to ratio driven resuscitation (7). The survival benefit of TEG based resuscitation has recently been validated in a randomized controlled trial that demonstrated a 50% reduction in mortality (8). However, the causality for improvement in mortality in this study remains unclear, as TEG guided resuscitation was associated with lower plasma and platelet transfusions in the first several hours after injury. While the focus of trauma and coagulation early after injury has been predominantly on hypocoagulability, hypercoagulable (hyper) states has received less attention. Hypercoagulability following injury is common when measure by TEG (9), and the development of hypercoagulability after injury is associated with thrombotic complications (10). Recent analysis of the fibrinolytic system has identified that impaired clot degradation (fibrinolysis shutdown) is associated with increased mortality (11, 12). However, the threshold for defining fibrinolysis shutdown was based on a risk of mortality, and not defined by a healthy volunteer population. Furthermore, the overall impact of other TEG measurements to detect hypercoagulability and mortality have not been assessed previously. We hypothesized that the predominant phenotype of postinjury coagulopathy is hypercoagulability and is associated with increased mortality, largely attributable to impaired fibrinolysis.

Material and Methods

Acutely injured patients enrolled in studies under IRB approved protocols from 2012–2014 from the University of Colorado Denver/Denver Health Medical Center and University of Texas Houston/Memorial Hermann were included in this study. Patients meeting trauma activation criteria were included if they had an injury severity score (ISS) greater than 15, were directly transferred from the injury scene to the emergency department, and had a rapid TEG drawn within one hour postinjury. Patients taking anticoagulant medication including

warfarin or direct factor inhibitors were excluded from the study. In addition, patients who received antifibrinolytics before TEG were excluded.

Patient demographics, injury patterns, and blood product use was prospectively recorded in all patients. The primary outcome was in-hospital mortality. Secondary outcomes included the cause of mortality (determined by senior investigators EEM and BAC based on morbidity and mortality meetings and clinical pathology reports if available), survival time (from injury to death), and massive transfusion (defined as greater than or equal 10 units of RBC within 6 hours postinjury).

Trained professional research assistants assayed blood with the TEG 5000 Hemostatic Analyzer (Heamonetics, Braintree, MA). Blood was obtained in 2.7 ml citrated tubes (Vacutainer, Becton-Dickison, Franklin Lakes NJ) and assayed after re-calcification. The following four rapid TEG (rTEG) measurements were recorded: T-ACT (seconds), angle (α , degrees), maximum amplitude (MA, mm), and lysis 30 minutes after MA (LY30, %).

Patients were categorized as hypercoagulable, hypocoagulable, or normal with all four rTEG variables. Coagulation status was based off of the 5th or 95th percentile of the values of a rapid TEG obtain in healthy volunteers (n=141) in a separate study. Hypercoagulability was defined as T-ACT <5th percentile (97 sec), angle >95th percentile (77 degrees), MA >95th percentile (70), and LY30 < 5th percentile (0.4%). Hypocoagulability was defined as T-ACT >95th percentile (>136 sec), angle <5th percentile (<64 degrees), MA <5th percentile (<55 mm), LY30 >95th percentile (4.8%). TEG variables within the 5th and 95th percentile were considered normal. From the composite of a patients score they were classified as hypercoagulable (if they had one or more hyper TEG variables and no hypo) hypocoagulable (if they had one or more hypo and no hyper), mixed (if had a hyper and hypo measurements), or normal (if all 4 TEG variables normal)

SPSS version 22 (IBM, Armonk, NY, USA) was used for statistical analysis. rTEG measurements are presented as median and interquartile values. Comparisons between groups were contrasted with chi square for categorical variables, and Kruskal-Wallis for continuous variables with adjustment for multiple comparisons. Binary logistic regression was used to identify variables associated with each coagulation pattern. Logistic regression was also used to predict mortality based on these coagulation phenotypes and control for confounders included age, sex, and, injury severity. Kaplan Meir curves were generated to contrast survival times and contrasted between cohorts with a log rank test.

Results

Patient Population

2,540 patients were included in the analysis. They were predominantly male (74%) with a median age of 38 years (26–55). This population of severely injured patients had a median ISS of 25 (21–33) and predominantly sustained blunt injury (83%), with a massive transfusion rate of 8.3%, and an overall mortality rate of 22%. The demographic and injury patterns of the patients stratified by coagulation status are listed in table 1. Logistic regression variables associated with hypercoagulable patients included older age ($p<0.001$),

female ($p<0.001$), higher systolic blood pressure ($p=0.001$), and lower chest abbreviated injury severity score (AIS $p=0.020$). Hypocoagulable patients were associated with a lower systolic blood pressure in the ED ($p<0.001$) lower GCS ($p=0.001$), a higher rate of penetrating injuries ($p=0.046$), and a higher ISS ($p=0.002$). Mixed coagulopathy was associated with an older age ($p=0.001$), higher heart rate in the ED ($p=0.007$), lower GCS ($p<0.001$), and lower chest AIS ($p=0.007$).

Coagulation Patterns

Compared to the healthy volunteer population, the distribution of all rTEG measurements were statistically different (T-ACT $p<0.001$, angle $P<0.001$, MA <0.001 , LY30 <0.001). Normal four rTEG coagulation was present in 36% of patients. Hypercoagulability was the most common, found in 38% of patients, with mixed (11%) and hypo (15%) being less common. The specific rTEG abnormality of each stratum are listed in Table 2a (hyper) and 2b (hypo). In patients who were hypercoagulable the LY30 was the most common abnormality prevalent in 67% of patients. The same trend was appreciated in mixed coagulopathy patients in which 87% of patients had an abnormally low LY30. In hypocoagulable patients, abnormalities of all four rTEG measurements had a similar prevalence (roughly 50%), but in the mixed cohort the same pattern existed except LY30 was only abnormally high in 10% of patients.

Outcomes

The rate of massive transfusion between coagulation pattern was different ($p<0.001$). Normal coagulation and hyper patients both had a MT rate of 5.7%, while mixed had a rate of 12.1% and hypo had the highest rate at 18.2%. The mortality also differed between phenotypes ($p<0.001$ Figure 1). Compared to normal coagulation patients and after controlling for age, sex, SBP, and injury severity hyper (0.013), mixed ($p<0.001$) and hypo ($p<0.001$) were all independent predictors of mortality. Compared to normal coagulation, survival times were significantly different between groups (hyper $p<0.001$, mixed $p<0.001$, hypo $p<0.001$ Figure 2). The cause of mortality differed between coagulation phenotypes ($p<0.001$ Figure 3). Hypocoagulable patients represented 46% of all hemorrhagic deaths. Hypercoagulable patients represented 36% of all traumatic brain injuries and 39% of all patients that died from a septic/thrombotic/organ failure related complication.

Discussion

In our study of 2,540 severely injured patients at two high-volume level-1 trauma centers, hypercoagulability is not only the most common presentation, but is associated with increased mortality compared to patients with normal coagulation status. The LY30 measurement was the most commonly abnormal value in hypercoagulable patients, while hypocoagulability did not have a defining rTEG measurement. Hypocoagulability and mixed coagulopathy, while less frequent, were more lethal than the hypercoagulability presentation. Where hypocoagulable patients had the highest incidence of hemorrhagic deaths, hypercoagulable patients had a high percentage of mortality related to traumatic brain injury and septic/thrombotic/organ failure.

A hypercoagulable state following injury has previously been reported to be common (9, 13) but has not previously been directly or specifically associated with increased mortality. However, inhibition of fibrinolysis (inability to breakdown clot) has been associated with increased mortality by several investigators (11, 14). To our knowledge this is the first paper to take into consideration the entire TEG profile to characterize patient's overall coagulation status. Prior literature has partitioned TEG measurements to assess the individual risk of each component with adverse outcomes and has been predominantly focused on hypocoagulability (15, 16). However, it is known that there are different drivers of coagulation changes following trauma, and it has been demonstrated that clot formation is independent of fibrinolysis (6, 17). A more global picture of a patient's TEG profile discriminated outcomes and risk of mortality, and supports that normal coagulation following injury is associated with the lowest mortality.

When trauma patients present to the hospital they are responding to an injury pattern to block ongoing hemorrhage but not cause microvasculature thrombosis to occlude. Animal work supports that tissue injury promotes a stronger clot (12, 18). Conversely, shock promotes a weaker clot (19) prone to fibrinolysis (12). The protective effect of hypocoagulation during profound shock has been previously reported by Hardaway et al. in which both heparin (20) and fibrinolytics (21) improved mortality in animals that underwent a near lethal model of shock. Further evidence of the protective role of hypocoagulation following shock is evident with the findings that eliminating protein C is 100% lethal in mice undergoing hemorrhagic shock due to diffuse microvascular thrombosis (19). These mechanistically explain why trauma patients can present to the hospital with a mix of hyper and hypocoagulable TEG profiles, as severe trauma is often a combination of tissue injury and shock.

The clinical relevance of this perplexing early state of mixed coagulopathy is that the majority of these patients will transition to a hypercoagulable state. Solid organ injured patient can become hypercoagulable within 48 hours of injury (10, 22). Furthermore, the duration that a patient remains in a hypercoagulable state, the higher risk of mortality (23). Animal work has demonstrated that tissue injury causes fibrinolysis shutdown, while hemorrhagic shock increases fibrinolysis (12, 24). These early changes may represent a response to injury in attempt to attain hemostasis but retain blood flow to organs, but prolongation of deranged coagulation may be pathologic. Therefore, a resuscitation strategy designed to the patient in a normal range of coagulation is logical. This was appreciated in the 1960's in which it was suggested that coagulation is a physiologic process never intended to reach a physiologic endpoint (25). This attempt to correct hypercoagulability following injury is not new, as heparin based venous thromboembolic prophylaxis is advocated to start with 48 hours of injury (26, 27). However, this may not be an adequate mechanism to bring patients back to a normal coagulation state, as even increased dosed heparin based prophylaxis does not correct a post injury TEG detected hypercoagulability (28).

There are several limitations of this study worth discussing. First, this was a study of only severely injured trauma patients (ISS>15) and only those from two trauma centers. As well, this was retrospective data abstracted from institutional databases, and not from direct,

prospective observation. In addition, this study used a statistical probability to define hyper and hypocoagulability as the extremes of the four TEG parameters based on a large healthy volunteer population. Previous descriptions of the hypercoagulability were based on manufactures recommendations (9, 22) or a small sample of healthy controls (28). However, the reference range for injured, non-bleeding trauma patients may be vastly different from healthy volunteers, which may partially explain the difference in recommended treatment thresholds released by the manufacturer and those of level-1 trauma centers experienced in TEG-based resuscitation. While there is no direct evidence that transfusing hypercoagulable trauma patients with hemostatic blood products increases mortality in trauma, researchers have shown that unguided platelet transfusions to patients with intracranial hemorrhage have been associated with increased mortality (29). This may explain why empiric high ratios of platelets and plasma to RBC in trauma (5) may not be as efficacious as goal directed resuscitation with TEG (8).

While there are continued efforts to refine the thresholds for transfusions of hypocoagulability with TEG (30) new efforts should also focus on what defines early hypercoagulability for potential therapeutic interventions. The current study found that hypercoagulability early after injury is associated with increased mortality in trauma. Future robust, prospective mechanistic studies are needed to identify if this represents a biomarker of poor outcomes versus a mechanism for increased mortality.

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Figure 1.

Figure 2.

Figure 3.

Table 1

Patient demographics and injury patterns

	Normal	Hyper	Mixed	Hypo	P value
Age (years)	34 (23 – 49)	45 (29 – 59)	43 (25 – 60)	33 (23 – 50)	<0.001
Sex (male)	78%	69%	74%	79%	<0.001
ED SBP	118 (95 – 138)	120 (100 – 142)	112 (86 – 140)	105 (82 – 130)	<0.001
ED HR	97 (79 – 117)	96 (80 – 115)	105 (80 – 121)	100 (80 – 120)	0.028
ED GCS	9 (3 – 15)	6 (3 – 15)	3 (3 – 15)	3 (3 – 14)	<0.001
Blunt	81%	88%	81%	79%	<0.001
ISS	25 (19 – 30)	25 (20 – 30)	26 (22 – 22)	29 (22 – 36)	<0.001
AIS Head	3 (0 – 4)	3 (0 – 4)	3 (0 – 5)	3 (0 – 5)	<0.001
AIS Chest	3 (0 – 3)	2 (0 – 3)	2 (0 – 3)	3 (0 – 3)	0.001
AIS Abd	0 (0 – 3)	0 (0 – 2)	0 (0 – 3)	0 (0 – 3)	0.238

Hyper= hypercoagulable; Hypo= hypocoagulable; ED = emergency department; SBP = systolic blood pressure; GCS = Glasgow coma score; ISS = injury severity score; AIS = abbreviated injury severity score; Abd = abdomen

Table 2A

Percent of rTEG measurements in hypercoagulable range

	T - ACT	Angle	MA	LY30
Normal	0%	0%	0%	0%
Hyper	9%	38%	29%	67%
Mixed	6%	11%	2%	86%
Hypo	0%	0%	0%	0%
Overall	4%	16%	12%	34%

Hyper= hypercoagulable; Hypo= hypocoagulable; T-ACT= TEG activated clotting time; MA = maximum amplitude; LY30 = lysis at 30 minutes

Table 2B

Percent of rTEG measurements in hypocoagulable range

	T - ACT	Angle	MA	LY30
Normal	0%	0%	0%	0%
Hyper	0%	0%	0%	0%
Mixed	45%	48 %	57 %	10 %
Hypo	50%	52 %	49 %	5 0%
Overall	13%	11 %	14 %	9 %

Hyper= hypercoagulable; Hypo= hypocoagulable; T-ACT= TEG activated clotting time; MA = maximum amplitude; LY30 = lysis at 30 minutes