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Higher mortality in pediatric and adult trauma patients with traumatic coagulopathy, using age-adjusted diagnostic criteria

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ABSTRACT

Background: Acute traumatic coagulopathy has been described in adult trauma patients. Acute traumatic coagulopathy may be associated with higher mortality and morbidity in pediatric trauma patients. We aimed to (1) compare acute traumatic coagulopathy incidence among various age groups, using age-adjusted normal reference values for three tests of coagulation, and (2) compare acute traumatic coagulopathy–associated mortality by age.

Methods: We queried our institutional trauma database for all level 1 and 2 activations with an injury severity score $\geq$ 9 during 2012 to 2017. Demographics, injury information, and coagulation test results were collected. Coagulopathy was defined using published age-specific and assay-specific parameters. Variables were compared among age groups (children, adults, and older adults), and logistic regression was used to determine independent associations with mortality.

Results: A total of 1,983 patients were included with a median injury severity score of 17 and mortality of 12%. Prolonged partial thromboplastin time, prolonged international normalized ratio, and hypofibrinogenemia were all strongly associated with mortality among adults and children, but not among older adults ($P < .001$, $P < .001$, and $P > .01$, respectively). Logistic regression revealed an independent association between prolonged partial thromboplastin time and mortality ($P < .001$).

Conclusion: Prolonged partial thromboplastin time/international normalized ratio and hypofibrinogenemia were common among trauma patients of all ages and were associated with mortality among children and adults, but not older adults, perhaps implicating age-related hemostatic biologic differences.

Introduction

Traumatic injury is a major cause of disability and mortality among all ages and is the leading killer of children and young adults.[1] Although coagulation derangements after traumatic injury have been appreciated for decades, only recently has traumatic (or trauma-induced) coagulopathy been characterized definitively among adults. The pathophysiology is likely multifactorial, with contributions from crystalloid dilution, hypothermia, and intravascular consumption of coagulation factors, among others.[2] However, after controlling for exogenous factors, it appears that ATC persists, suggesting that ATC may be attributed in part to the injury itself and subsequent early immune and hemostatic responses.[3] This is further supported by the findings that mild isolated traumatic brain injuries without major hemorrhage or shock may result in anomalies on coagulation tests immediately after injury.[4]

Different definitions of ATC have been explored in the literature. The majority of published reports rely on prolongation of the conventional coagulation tests on arrival to the emergency department. The most commonly used tests are the international normalized ratio (INR) calculated from prothrombin time (PT) and, to a lesser extent, partial thromboplastin time (PTT). Benefits of using these tests include their ubiquitous use among trauma centers and more than a half century of published experience with their use. Furthermore, such experience has led to a robust set of reports on the normal values among children, which differ from

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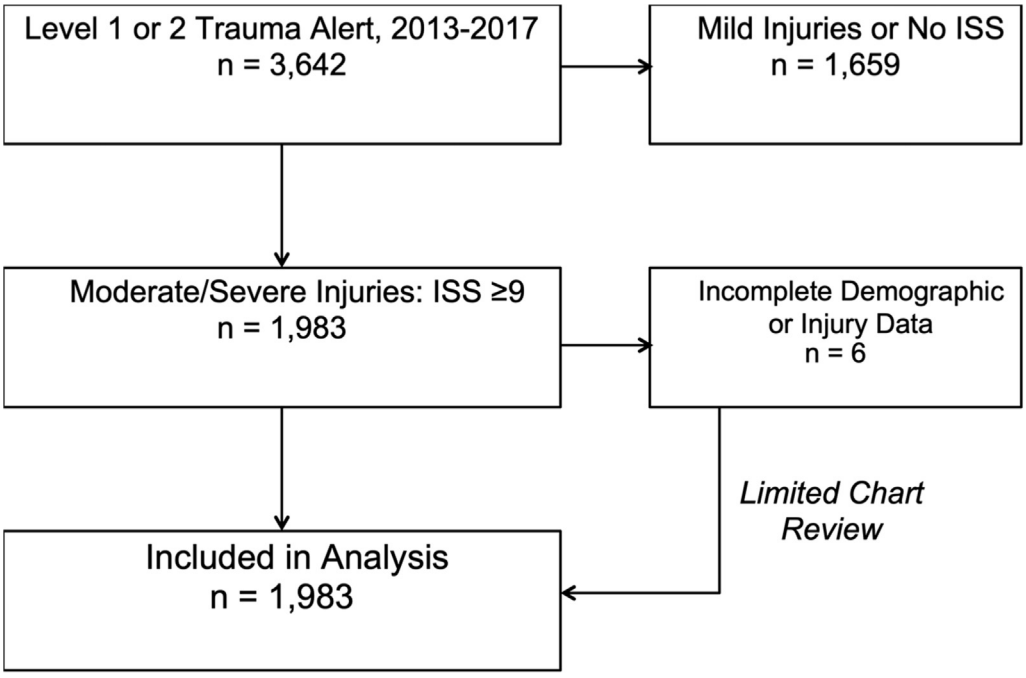

Fig 1. Flowchart presents inclusion and exclusion criteria with the number of included subjects.

adults considerably, owing to normal hemostatic development in the quantity and function of both plasma procoagulant and regulatory factors, which should be considered when interpreting their results.[5,6]

Recent investigations have also reported experiences with the viscoelastic hemostatic assays (ie, thromboelastography and rotational thromboelastometry), which theoretically provide a more rapid and complete picture of hemostasis and thrombotic potential and information about fibrinolysis in the traumatic setting.[7] Some studies have found a relationship between clot strength and early hemorrhage that supports the utility of these devices in the evaluation and resuscitation of acutely injured patients, although a recent Cochrane review of adult trauma patients concluded that more study is required.[8,9]

Therefore, it appears that the conventional coagulation tests will continue to play an important role in the diagnosis and management of ATC among patients of all ages. Despite the robust literature on ATC in adult patients, there are few published reports of the phenomenon and its consequences among children. These studies have not considered age-specific normal thresholds of coagulation tests in defining ATC. In the current study, we aimed to use age-adjusted reference values for conventional coagulation tests to investigate variations in the incidence of ATC by age and the association between ATC and mortality among each age group.

Patients and Methods

At our institution (Duke University Medical Center, Durham, NC), level 1 and 2 trauma codes are typically activated before patient arrival in response to prehospital vital sign or Glasgow Coma Score derangements. We queried our institutional trauma database for all level 1 and 2 trauma team activations for a 5-year period, spanning from January 1, 2013, through and including December 31, 2017, with an injury severity score (ISS) of 9 or greater to capture only those patients with moderate or severe injuries as suggested by Bolorunduro et al.[10] The study was approved by the Duke University Institutional Review Board. The study dates were selected to reflect the longest period possible for which reliable and robust lab data were available. Inclusion criteria were complete records including age, ISS, mortality, and injury mechanism. We conducted a limited chart review on patients with missing mortality or injury data and considered them complete if the data were readily available in the subjective portion of the electronic medical record. Patients with mild injuries (ISS < 9) were excluded (Fig 1).

Patient demographics, injury data, and results from laboratory studies performed in the emergency department were captured and analyzed. Patient disposition from the hospital was also captured and reported. Labs collected included arterial blood gas pH, PaO2, PaCO2, lactate, and hematocrit. Conventional coagulation tests analyzed were PTT, INR, and plasma fibrinogen level. All coagulation studies were conducted by laboratory staff at the hospital's central clinical lab, using an ACL TOP automated analyzer (Instrumentation Laboratory, Bedford, MA). PTT was performed using the SynthASil reagent according to the manufacturer's recommendations (Instrumentation Laboratory).

An occurrence of traumatic coagulopathy was reported using three definitions: prolonged PTT, INR, and fibrinogen.

Table I
Age-adjusted normal ranges for instrumentation laboratory equipment*

	Birth–4 weeks	1–5 months	6–11 months	1–5 years	6–10 years	11–17 years	Adults
PTT (seconds)	35.4 (27.6–45.6)	33.5 (24.8–40.7)	32.4 (25.1-40.7)	31.6 (24–39.2)	31.6 (26.9–38.7)	31.0 (24.6–38.4)	31.7 (27.8–36.3)
INR (seconds)	0.98 (0.83–1.13)	0.97 (0.84–1.17)	0.98 (0.85-1.13)	0.99 (0.86–1.20)	1.04 (0.9–1.30)	1.04 (0.9–1.24)	0.95 (0.81–1.07)
Fibrinogen (g/L)	2.40 (1.36–3)	2.10 (1.41–4.37)	2.30 (1.48-3.67)	2.60 (1.64–4.97)	2.76 (1.71–5.37)	2.48 (1.68–5.29)	2.86 (2.13–4.22)

* As Described by Toulon et al.[11]

Table II
Patient characteristics

	All patients ($N = 1,983$) n (%)*	Infant/child ($N = 156$) n (%)*	Adult ($N = 1,524$) n (%)*	Older adult ($N = 303$) n (%)*	Adult versus infant/child P value†	Adult versus older adult P value†
Age (years)					< .001	< .001
Median (IQR)	36.0 (23.0–56.0)	5.0 (2.0–8.0)	33.0 (24.0–47.0)	74.0 (69.0–82.0)		
Mechanism of injury					< .001	< .001
Blunt	1,492 (75.2%)	132 (84.6%)	1,077 (70.7%)	283 (93.4%)		
Penetrating	448 (22.6%)	10 (6.4%)	421 (27.6%)	17 (5.6%)		
Other	43 (2.2%)	14 (9.0%)	26 (1.7%)	3 (1.0%)		
ISS					.04	.36
Median (IQR)	17.0 (10.0–22.0)	12.5 (9.0–19.0)	17.0 (10.0–22.0)	17.0 (11.0–25.0)		

IQR, interquartile range.
* Percentages may not add up to 100 because of rounding or missing values.
† P values for categorical variables are from χ^2 tests. P values for continuous variables are from Satterthwaite t tests.

Age-adjusted normal reference values for the Instrumentation Laboratory ACL TOP system employed at our institution were ascertained from the large, multicenter study of healthy children and adults conducted by Toulon et al.[11] The 2016 publication reported normal values for PTT, INR, and fibrinogen in each of 7 age categories and defined the 95th percent reference ranges according to CLSI C28-A3 guidelines for neonates and infants, young children, toddlers, adolescents, and adults.[12] Using these thresholds, we defined ATC among each patient as prolonged PTT, prolonged INR, or low fibrinogen relative to the age-specific 95% reference interval for all patients (Table I).

To compare the incidence of coagulopathy and outcomes by age, subjects were arranged into 1 of the following 3 groupings by age at the time of injury: infants and children (birth through and including 13 years of age), adults (14 years of age through and including 64 years of age), and older adults (65 years of age and older). These groupings were performed independently of determination of conventional coagulation test normality. The adult age group was used as the reference value for two-way cross-tabulation comparisons of coagulopathy incidence and coagulopathy among those who died in the hospital.

Descriptive statistics were calculated for all variables. Median and interquartile ranges were calculated and reported for age, ISS, and lab values. Satterthwaite t tests or Kruskal-Wallis tests were used to compare continuous variables for infants and children versus adults and older adults versus adults. We uses χ^2 tests or Fisher exact tests to compare categorical variables. Finally, logistic regression models for an outcome of each definition of coagulopathy, and mortality, were used to determine the independence of associations among age, coagulopathy, and mortality. Because of the number of comparisons, $P < .01$ was taken to represent statistical significance, although no formal adjustments for multiple comparisons were made. SAS 9.4 was used for all statistical analyses (SAS Institute, Cary, NC).

Table III
Lab results

	All patients (N = 1,983) n (%)*	Infant/child ($N = 156$) n (%)*	Adult ($N = 1,524$) n (%)*	Older adult ($N = 303$) n (%)*	Adult versus infant/child P value†	Adult versus older adult P value†
pH					.04	< .001
n	1,721	95	1,368	258		
Median (IQR)	7.3 (7.2–7.4)	7.3 (7.2–7.3)	7.3 (7.2–7.4)	7.3 (7.3–7.4)		
PaO2					.99	.12
n	1,639	92	1,302	245		
Median (IQR)	44.0 (29.0–82.0)	48.0 (37.0–76.0)	44.0 (29.0–85.0)	40.0 (28.0–74.0)		
PaCO2					.01	.42
n	1,524	84	1,218	222		
Median (IQR)	46.0 (40.0–53.0)	48.5 (40.0–58.5)	46.0 (39.0–53.0)	46.5 (40.0–54.0)		
Lactate					.67	< .001
n	1,510	80	1,214	216		
Median (IQR)	2.6 (1.7–3.9)	2.4 (1.6–3.6)	2.7 (1.7–4.0)	2.1 (1.4–3.1)		
Base excess					.10	< .001
n	1,725	93	1,374	258		
Median (IQR)	−3.0 (−6.0–−1.0)	−5.0 (−7.0–−3.0)	−4.0 (−7.0–−1.0)	−1.0 (−4.0–1.0)		
HCT					< .001	< .001
n	1,922	146	1,478	298		
Median (IQR)	38.3 (34.0–42.0)	34.0 (30.2–36.0)	39.0 (35.0–42.2)	37.0 (32.0–41.0)		
PTT					< .001	.40
n	1,771	123	1,374	274		
Median (IQR)	26.2 (23.8–9.1)	28.2 (26.3–32.3)	26.0 (23.7–28.8)	26.2 (23.8–29.2)		
INR					.07	< .001
n	1,870	130	1,447	293		
Median (IQR)	1.0 (1.0–1.1)	1.1 (1.0–1.2)	1.0 (1.0–1.1)	1.0 (1.0–1.1)		
Fibrinogen					< .001	< .001
n	1,271	83	996	192		
Median (IQR)	226.0 (181.0–287.0)	185.0 (158.0–231.0)	221.0 (178.0–274.0)	287.0 (230.0–361.0)		

IQR, interquartile range.
* Percentages may not add up to 100 because of rounding or missing values.
† P values for categorical variables are from χ^2 tests. P values for continuous variables are from Satterthwaite t tests.

Table IV
Patient outcomes

	All patients ($N = 1,983$) n (%)*	Infant/child ($N = 156$) n (%)*	Adult ($N = 1,524$) n (%)*	Older adult ($N = 303$) n (%)*	Adult versus infant/child P value†	Adult versus older adult P value†
Disposition from hospital					< .001	< .001
Home	1,233 (62.2%)	126 (80.8%)	1,025 (67.3%)	82 (27.1%)		
Skilled nursing or rehab facility	476 (24.0%)	14 (9.0%)	322 (21.1%)	140 (46.2%)		
Deceased or hospice	249 (12.6%)	14 (9.0%)	159 (10.4%)	76 (25.1%)		
Other acute care facility	25 (1.3%)	2 (1.3%)	18 (1.2%)	5 (1.7%)		
Mortality					.78	< .001
Alive	1,747 (88.1%)	142 (91.0%)	1,369 (89.8%)	236 (77.9%)		
Dead	236 (11.9%)	14 (9.0%)	155 (10.2%)	67 (22.1%)		
PTT compared with age-adjusted reference					.07	.07
Prolonged (coagulopathic)	110 (5.5%)	12 (7.7%)	75 (4.9%)	23 (7.6%)		
Low/normal	1,661 (83.8%)	111 (71.2%)	1,299 (85.2%)	251 (82.8%)		
INR compared with age-adjusted reference					< .001	.001
Prolonged (coagulopathic)	735 (37.1%)	20 (12.8%)	569 (37.3%)	146 (48.2%)		
Low/normal	1,135 (57.2%)	110 (70.5%)	878 (57.6%)	147 (48.5%)		
Fibrinogen compared with age-adjusted reference					.03	< .001
Abnormal low (coagulopathic)	483 (24.4%)	25 (16.0%)	423 (27.8%)	35 (11.6%)		
High/normal	788 (39.7%)	58 (37.2%)	573 (37.6%)	157 (51.8%)		

* Percentages may not add up to 100 because of rounding or missing values.
† P values are from Fisher's exact tests.

Results

A total of 1,983 subjects who presented as level 1 or 2 trauma alerts with ISS $\geq$ 9 had complete demographics and injury data and were included in the analysis (100%). There were 156 patients under 14 years of age (7.9% of the sample overall) and 303 older adults 65 years of age or older (15.3% of the sample overall) with a median sample age of 36 years (Table II). Overall ISS was 17 and mortality in the sample was 11.9%, making the sample generally representative of a severely injured cohort. We observed no statistically significant differences in ISS among age groups, although penetrating trauma was more common among adults compared with extremes of age ($P < .001$ for both cases).

Findings from arterial blood gas analysis performed in the emergency department, including lactate and base deficit, generally indicated that adult patients were more likely to arrive physiologically deranged; however, hematocrit was significantly lower in both the pediatric and older adult samples compared with adults, even without adjustment for an expected higher hematocrit among very young children and neonates ($P < .001$ in both cases [Table III]). Of note, before any adjustment for age, there were significant differences in PTT, INR, and fibrinogen on arrival. PTT was significantly prolonged among children compared with adults ($P < .001$), and INR was significantly prolonged among older adults compared with adults ($P < .001$). Plasma fibrinogen level was significantly lower among children compared with adults and

Table V
Cross tabulations of mortality rate among patients with conventional coagulation test by age group

	PTT*		Total	P value
	Low/normal PTT	Prolonged PTT		
Infant/child	1/111 (0.9%)	10/12 (83.3%)	14/156 (9.0%)	< .001
Adult	69/1,299 (5.3%)	38/75 (50.7%)	155/1,524 (10.2%)	< .001
Older adult	54/251 (21.5%)	9/23 (39.1%)	67/303 (22.1%)	.05
Total	124/1,661 (7.5%)	57/110 (51.8%)	236/1,983 (11.9%)	
	INR†		Total	P value
	Low/normal INR	Prolonged INR		
Infant/child	3/110 (2.7%)	8/20 (40.0%)	14/156 (9.0%)	< .001
Adult	42/878 (4.8%)	66/569 (11.6%)	155/1,524 (10.2%)	< .001
Older adult	32/147 (21.8%)	30/146 (20.5%)	67/303 (22.1%)	.80
Total	77/1,135 (6.8%)	104/735 (14.1%)	236/1,983 (11.9%)	
	Fibrinogen‡		Total	P value
	High/normal fibrinogen	Hypofibrinogenemia		
Infant/child	2/58 (3.4%)	7/25 (28.0%)	14/156 (9.0%)	<0.001
Adult	40/573 (7.0%)	56/423 (13.2%)	155/1524 (10.2%)	<0.001
Older adult	39/157 (24.8%)	10/35 (28.6%)	67/303 (22.1%)	0.65
Total	81/788 (10.3%)	73/483 (15.1%)	236/1983 (11.9%)	

* A total of 23% of patients who died were missing PTT results. P values are from χ^2 tests of mortality and PTT result within each age group. Cochran-Mantel-Haenszel P value < .001.
† A total of 23% of patients who died were missing INR results. P values are from χ^2 tests of mortality and INR result within each age group. Cochran-Mantel-Haenszel P value < .001.
‡ A total of 35% of patients who died were missing fibrinogen results. P values are from χ^2 tests of mortality and fibrinogen result within each age group. Cochran-Mantel-Haenszel P value < .001.

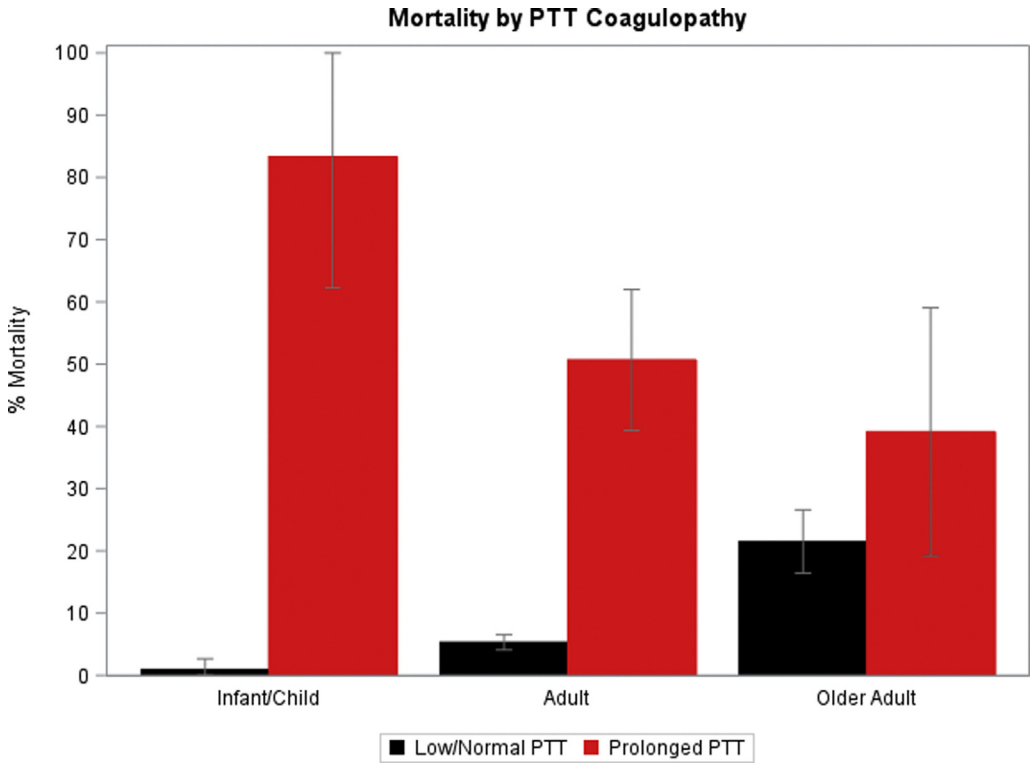

Fig 2. Mortality rate by age group and deranged PTT. A statistically significant ($P < .01$) difference exists in mortality between those with normal PTT and prolonged PTT for pediatric and adult groups, but not among older adults.

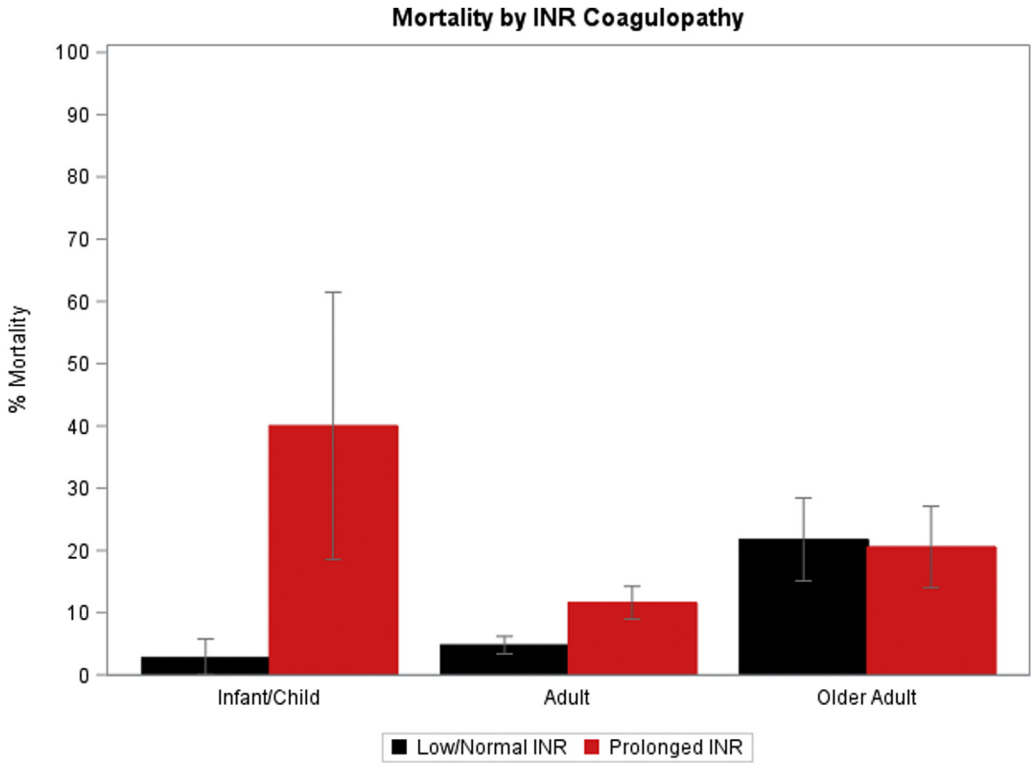

Fig 3. Mortality rate by age group and deranged INR. A statistically significant ($P < .01$) difference exists in mortality between those with normal INR and prolonged INR for pediatric and adult groups, but not among older adults.

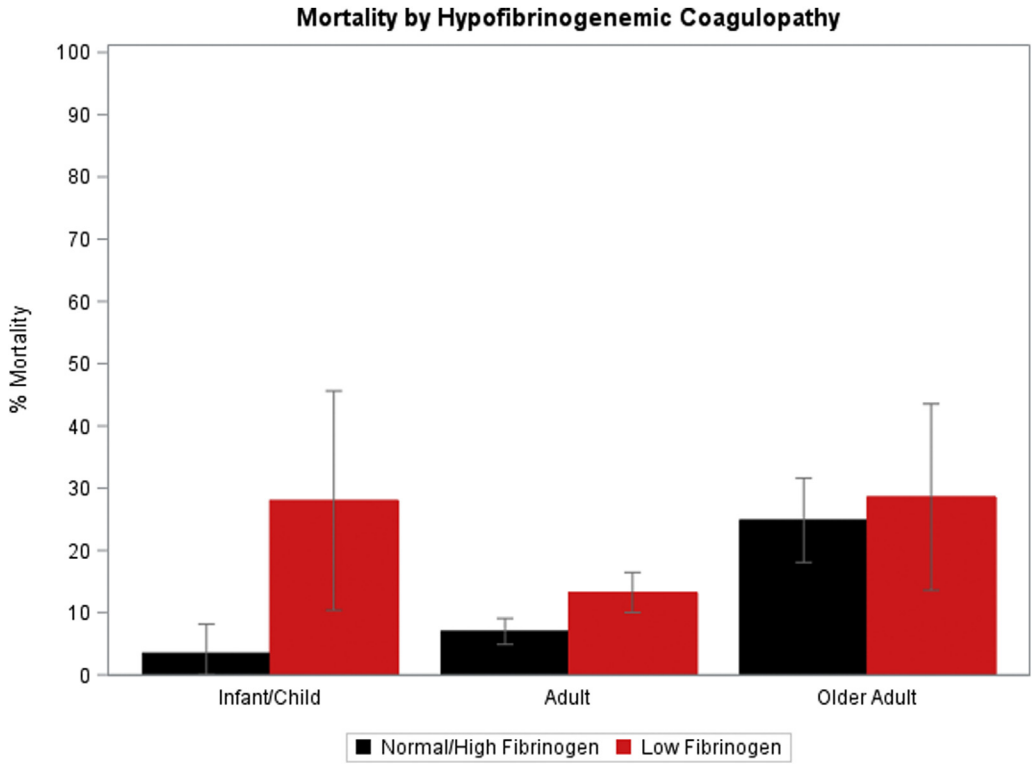

Fig 4. Mortality rate by age group and hypofibrinogenemia. A statistically significant ($P < .01$) difference exists in mortality between those with normal or high fibrinogen and hypofibrinogenemia for pediatric and adult groups, but not among older adults.

significantly higher among older adults compared with adults ($P <$ .001 in both cases). Older adults were more likely and children less likely to be discharged to a skilled nursing facility compared with adults (Table IV).

After adjustment of reference values for age, PTT was prolonged among 7.7% of children, 4.9% of adults, and 7.6% of older adults (not significantly different; $P = .07$ in both cases). INR was prolonged among 12.8% of children, 37.3% of adults, and 48.2% of older adults (significantly higher among adults compared with children and highest among older adults; $P = .001$ for children versus adults and $P < .001$ for older adults versus adults). Finally, fibrinogen was low among 16.0% of children, 27.8% of adults, and 11.6% of older adults (lower among adults compared with both extremes of age; $P = .03$ for children versus adults and $P < .001$ for older adults versus adults).

Analysis of unadjusted association between coagulopathy and age among patients with inhospital mortality demonstrated a greater proportion of deaths among patients with abnormal labs across age groups. There was a particularly strong association with PTT, in which the rate of coagulopathy was more than 50% among those with inhospital mortality (Table V). In serial comparisons of mortality rate by age group comparing those with coagulopathy versus those without, PTT, INR, and fibrinogen anomalies were significantly different in children and adults but not older adults (Figs 2–4). Multivariable logistic regression for outcomes of prolonged PTT/INR and hypofibrinogenemia revealed that age had no significant independent association with prolonged PTT ($P = .23$), but age group was statistically significantly associated with abnormal INR and hypofibrinogenemia (Tables VI–VIII, $P < .001$ in both cases).

Finally, in a logistic regression model with outcome of mortality, prolonged PTT was independently associated with mortality, along with ISS and older age (Table IX, $P < .001$). Despite associations with mortality in unadjusted comparisons, prolonged INR and hypofibrinogenemia were not significantly associated with mortality ($P = .43$ and .53, respectively).

Table VI
Logistic regression predicting abnormal PTT (N = 1,769)

	Odds ratio (95% CI)	*P* value	Overall *P* value
Age group			.23
Adult	Reference		
Infant/child	1.23 (0.60–2.54)	.57	
Older adult	1.57 (0.93–2.65)	.09	
Mechanism of injury			< .001
Blunt	Reference		
Penetrating	1.57 (0.96–2.59)	.07	
Other	5.77 (2.26–14.74)	< .001	
ISS	1.05 (1.04–1.07)	< .001	< .001
HCT	0.92 (0.89–0.95)	< .001	< .001

CI, confidence interval.

Table VII
Logistic regression predicting abnormal INR (N = 1,868)

	Odds ratio (95% CI)	*P* value	Overall *P* value
Age group			< .001
Adult	Reference		
Infant/child	0.16 (0.10–0.27)	< .001	
Older adult	1.41 (1.07–1.85)	.02	
Mechanism of injury			< .001
Blunt	Reference		
Penetrating	1.68 (1.32–2.15)	< .001	
Other	1.46 (0.71–2.99)	.30	
ISS	1.03 (1.02–1.04)	< .001	< .001
HCT	0.90 (0.88–0.92)	< .001	< .001

CI, confidence interval.

Table VIII
Logistic regression predicting abnormal fibrinogen (N = 1,270)

	Odds ratio (95% CI)	*P* value	Overall *P* value
Age group			< .001
Adult	Reference		
Infant/child	0.43 (0.25–0.73)	.002	
Older adult	0.25 (0.17–0.38)	< .001	
Mechanism of injury			< .001
Blunt	Reference		
Penetrating	1.93 (1.46–2.56)	< .001	
Other	1.68 (0.75–3.80)	.21	
ISS	1.05 (1.04–1.06)	< .001	< .001
HCT	0.93 (0.91–0.95)	< .001	< .001

CI, confidence interval.

Discussion

ATC is an often-described and often-encountered entity among severely injured adult patients.[13] Multiple definitions have been suggested, and its prevalence and consequences in the realm of pediatric trauma have only recently been queried.[14,15] The myriad definitions and associated clinical manifestations (which include hemorrhage, thrombosis, and death) have led to numerous hypotheses about its underlying mechanisms. In addition to exogenous influences from resuscitation and shock, the intrinsic factors that have been implicated include consumption of plasma coagulation factors, hyperfibrinolysis, and activation of protein C.[16] In an analysis of clinical and hematologic factors among significantly injured pediatric patients, Leeper et al[17] demonstrated that poor clot strength and global coagulation factor depletion were associated with mortality and transfusion requirement.

In the current study, we chose to consider any patient with prolongation of clotting tests or hypofibrinogenemia to be coagulopathic, to provide the broadest sample of ATC possible, including those with early and subtle evidence of coagulopathy. This includes both patients with those intrinsic mediators of ATC (protein C activation, consumption of coagulation factors without overt hemorrhage), and those with exogenous contributors (crystalloid administration, major hemorrhage). Although this may introduce some heterogeneity into the definition, the fundamental lesion associated with ATC occurs at the time of tissue injury and will be present in all patients with coagulation test derangements, unless there is a spurious lab value, preexisting coagulopathy, or other coincidental derangement.

Table IX
Logistic regression predicting death (N = 1,187)

	Odds ratio (95% CI)	*P* value	Overall *P* value
Age group			< .001
Adult	Reference		
Infant/child	0.78 (0.29–2.11)	.63	
Older adult	5.14 (3.11–8.49)	< .001	
PTT			< .001
Low/normal	Reference		
Abnormal high	9.00 (4.97–16.31)	< .001	
INR			.43
Low/normal	Reference		
Abnormal high	0.83 (0.53–1.31)	.43	
Fibrinogen			.53
High/normal	Reference		
Abnormal low	0.86 (0.53–1.38)	.53	
Mechanism of injury			.07
Blunt	Reference		
Penetrating	1.71 (1.02–2.88)	.04	
Other	2.43 (0.65–9.03)	.19	
ISS	1.11 (1.08–1.13)	< .001	< .001
HCT	1.01 (0.98–1.05)	.39	.39

CI, confidence interval.

It has been established that the concentrations and activities of plasma coagulation factors and inhibitors change throughout life.[5] Normal hemostasis is currently understood as a dynamic biologic system with redundant mechanisms governing the interplay of procoagulant and anticoagulant enzymes, fibrinolysis, and inflammation. From fetal life and birth to adulthood through senescence, the system goes through quantitative and qualitative changes, some of which are understood. This concept of developmental hemostasis must be taken into account when studying ATC in younger and older populations to accurately characterize coagulation anomalies. We developed the current clinical study to evaluate the incidence of and mortality associated with ATC among children, adults, and older adults using age-adjusted and analyzer-adjusted references. This is in contrast to earlier studies that have used arbitrary conventional coagulation test cutoffs similar to those in adults.[18]

In a study of critically injured pediatric trauma victims, Leeper et al[17] identified INR > 1.3 as an ATC definition with adequate performance in association with mortality, which was present in about 30% of their critically injured pediatric patients with 30% mortality. Using age-adjusted references in the current study, we found a mortality of 40% among pediatric patients who had labs, using a definition of deranged INR relative to reference, versus 3% among those with normal INR. However, the incidence of INR prolongation in our study was only 13%, which may be related in part to a less critically ill sample.

In addition to INR, we also found that prolonged PTT and hypofibrinogenemia were diagnostic of ATC. Among pediatric patients with labs, mortality was more than 80% with PTT prolongation compared with <1% in those without (hypofibrinogenemia performed similarly to INR among children). Of note, the unadjusted association between coagulopathy and mortality among older adults was less clear. In fact, the mortality rates among the elderly with increased INR and hypofibrinogenemia were virtually equal to those without. This may be attributable to differences in hemostatic biology that continue to occur with age.[19]

One limitation of any retrospective study evaluating ATC is the availability of coagulation studies. Bias may be introduced by provider discretion in the use of conventional coagulation tests, or patients may die before obtaining labs. Of note, in the current study, more than 20% of patients who ended up deceased in the hospital never had conventional coagulation tests in the emergency department, perhaps owing to very early mortality or late complications.

We conclude that this single-center retrospective study supports the association between ATC and inhospital mortality across the spectrum of hemostatic development. In addition, we have demonstrated that there are important differences to consider when interpreting conventional coagulation test with respect to age in trauma victims. The association between PTT derangement independent of INR and fibrinogen deficiency may unexpectedly implicate the intrinsic pathway of coagulation in the pathogenesis of ATC. Further study may focus on understanding changes in the pediatric and older adult coagulum in response to trauma and resuscitation.

Disclosure

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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