



Explosion Mechanism Associated with Higher INR in Combat Injured Patients



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Abstract

Background: Recent evidence suggests trauma involving total body tissue damage increases the Acute Coagulopathy of Trauma (ACOT) by various mechanisms, especially in massive transfusion (MT). Our hypothesis was that MT patients injured by explosion will have a higher international normalized ratio(INR) at admission than MT patients injured by gunshot (GSW).

Methods: A retrospective review was performed on all US military injured in OIF/OEF, from MAR03 to SEP08, who received MT (≥ 10 RBC in 24 hours). Two cohorts were created based on mechanism of injury. Admission vital signs, labs, and mortality data were compared. A one-tailed nonparametric test was used for INR.

Results: 777 MT patients were identified. 450 patients had admission INR and were injured by either gunshot or explosion. Though the patients demonstrated similar ISS, there was a significant difference in pulse, base deficit(BD), and INR between patients who were injured by gunshot and those injured by explosion

Conclusions: The primary finding of this study is that patients injured by explosion presented with a higher INR than those injured by GSW, even in the face of similar ISS. Additionally, patients injured by explosion presented more tachycardic, with a greater base deficit and received more blood products. These findings support the theory that ACOT is affected by the amount of tissue injured. Further research is needed into the pathophysiology of ACOT as this may impact care of patients with total body tissue damage/hypoxia and improve the treatment of their coagulopathy.



Introduction

Injuries sustained from improvised explosive devices (IEDs) have been a leading cause of mortality during Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF). Of potentially survivable battlefield injuries, hemorrhage is known to be a leading cause of mortality.¹ Anecdotal evidence from recent U.S. military surgeons has suggested that patients injured from IED blasts could be more coagulopathic and require more aggressive resuscitation with blood products when compared to patients injured from gun shot wounds.

An early coagulopathy prior to the onset of resuscitation has previously been identified in trauma patients and has been shown to be an independent predictor of mortality.^{2,3} The ACOT is a complex physiologic process with many interacting pathways. Tissue injury, shock, hemodilution, hypothermia, acidosis, and inflammation all appear to play a role in the development of the ACOT. Shock appears to be the main driver of early coagulopathy by inducing anticoagulation and hyperfibrinolysis.⁴ However, shock requires tissue injury with associated endothelial damage as an initiator. It has been shown that increasing injury severity, as measured by injury severity scores (ISS), is associated with the degree of coagulopathy.⁵ Explosion injuries have the potential to produce an extremely large burden of tissue injury, and when combined with shock can initiate an ACOT. Our hypothesis is that massive transfusion patients injured by explosion will have a higher INR at admission than massive transfusion patients injured by GSW.

Objectives

The purpose of this study was to determine if the mechanism of injury was associated with the severity of physiologic derangement in a cohort of patients with severe traumatic insult who required aggressive blood product resuscitation.

Methods

A retrospective review was performed on all US military injured in OIF/OEF, from MAR03 to SEP08, who received MT (≥ 10 RBC in 24 hours) as identified in the Joint Theater Trauma Registry. Two cohorts were created based on a primary mechanism of injury of explosion(i.e. IED, Mortar/Rocket/Artillery Shell, Blast) or gunshot wound(i.e. Bullet/GSW/Firearm). Admission vital signs and labs and mortality data were compared. A two-tailed t-test was used

Results

Seven hundred seventy seven patients from the Joint Theater Trauma Registry were identified who had undergone massive transfusion and were injured by either GSW or explosion. Four hundred fifty patients had INR upon admission data available for analysis.

The two groups demonstrated similar anatomic severity of injury between the explosive mechanism and GSW mechanism as measured by ISS (24 vs. 23, $p = 0.29$). The groups also demonstrated similar overall mortality(21% vs. 24%, $p=0.51$)

Patients with an explosive mechanism of injury demonstrated greater physiologic derangement on admission with increased pulse(114 vs. 101, $p=0.006$), worse base deficit (-8.2 vs. -5.8, $p=0.006$) and elevated INR (1.8 vs. 1.5, $p<0.001$). The explosion cohort was more likely to be coagulopathic on presentation (50% vs. 37%, $p=0.048$)

Table 1. Analysis by Cohort

	GSW N = 78	Explosion N = 372	p
Demographics			
Age	25 ± 6	26 ± 6	0.641
ISS	23 ± 10	24 ± 12	0.291
Vitals			
Pulse	101 ± 36	114 ± 34	0.006
SBP	100 ± 37	105 ± 36	0.291
Temp	97.5 ± 1.9	97.5 ± 6.5	0.999
GCS	11 ± 5	12 ± 5	0.686
Labs			
Hgb	11.7 ± 2.5	11.3 ± 2.8	0.285
BD	-5.8 ± 6.2	-8.2 ± 7.2	0.006
INR	1.5 ± 0.5	1.8 ± 1.0	<0.001
INR \geq 1.5	37%	50%	0.048
Mortality	24%	21%	0.508

Mean ± Standard Deviation

Conclusions

The primary finding of this study is an association between the mechanism of injury and the degree of physiologic derangement as measured by pulse, base deficit, and INR.

Although the patients presenting with both gunshot wounds and explosive injuries had similar anatomic injuries as assessed by the ISS, they were physiologically different. The explosion group demonstrated increased tachycardia, worse base deficit, and high INR with a larger percent coagulopathic by laboratory evaluation on presentation. This association suggests that the explosive mechanism causes a degree of diffuse tissue injury which is not assessed by a gross injury scale such as the ISS. This diffuse tissue injury may contribute to the ACOT seen in these patients which in manifested as the tachycardia, shock, and coagulopathy.

Thus explosive mechanism appears associated with :

- Increased tachycardia
- Greater base deficit
- Increased INR

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