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TRANSFUSION OF RED BLOOD CELLS IN PATIENTS WITH A PRE-HOSPITAL GCS ≤ 8 AND NO EVIDENCE OF SHOCK IS ASSOCIATED WITH WORSE OUTCOMES

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Abstract

Background—Red blood cell transfusion practices vary and the optimal hemoglobin for patients with traumatic brain injury (TBI) has not been established. We sought to identify the interaction between initial hemoglobin (Hgb) and red cell transfusion on 28-day survival, Acute Respiratory Distress Syndrome (ARDS) free survival, Multi-organ dysfunction (MODs) score, and 6-month Glasgow Outcome Score extended (GOSE) score in a cohort of patients with a pre-hospital Glasgow Coma Score (GCS) less than or equal to 8 and no evidence of shock.

Methods—A retrospective review of data collected prospectively as part of a randomized, controlled trial involving emergency medical services (EMS) agencies within the Resuscitation Outcomes Consortium was conducted. In patients with a GCS ≤ 8 without evidence of shock {defined by a systolic blood pressure (SBP) <70 or SBP of 70–90 with a heart rate (HR) ≥ 108 }, the association of red cell transfusion with 28-day survival, ARDS free survival, MODs score and 6 month GOSE was modeled using multivariable logistic regression with robust standard errors adjusting for age, sex, injury severity (ISS), initial GCS, initial SBP, highest field HR, penetrating injury, fluid use, study site and Hgb.

Results—1158 patients had a mean (m) age of 40, 76% were male and 98% suffered blunt trauma. The initial GCS (m) was 5 and initial SBP (m) was 134. The head abbreviated injury score (AIS) (m) was 3.5. A categorical interaction of red cell transfusion stratified by initial Hgb showed when the first hemoglobin was >10 grams/deciliter (g/dL), volume of pRBC was associated with a decreased 28 day survival (odd ratio [OR] 0.83 per unit with a 95% confidence interval [CI] of

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Authorship

J.E, S.B, MS designed the study. The Resuscitation Outcomes Consortium contributed to the data collection. S.B, J.E., M.S. contributed to statistical analysis and interpretation. All authors contributed to preparation of the manuscript and final revision.

[0.74, 0.93] $p < 0.01$) and decreased ARDS free survival (OR 0.82 per unit (95% CI [0.74, 0.92] $p < 0.01$). When the initial Hgb was > 10 , each unit of blood transfused increased the MODs score by 0.45 (Co-efficient 95% CI [0.19, 0.70] p -value < 0.01).

Conclusion—In patients with a suspected TBI and no evidence of shock, transfusion of red blood cells was associated with worse outcomes when the initial hemoglobin was > 10 . There was no relationship between blood transfusion and outcomes in the patients with initial hemoglobin 10.

Level of Evidence—II

Keywords

traumatic brain injury; transfusion; resuscitation

BACKGROUND

An estimated 1.4 million people suffer traumatic brain injuries (TBI) each year in the United States accounting for 50,000 deaths and leaving 80,000 – 90,000 patients with permanent disabilities. (1) It is well recognized that the primary injury to the brain occurs at the time of impact and that the focus of treatment for TBI is to prevent secondary injury. This is accomplished primarily by maintaining cerebral perfusion and reducing intracranial pressure. (2) The optimal resuscitation strategy to improve perfusion in patients with severe TBI has yet to be elucidated. Red blood cell transfusions are common in the management of severe TBI and have been estimated to occur in approximately 50% of TBI patients. (3) The principle goal of red cell transfusion in the management of TBI is to maximize brain tissue oxygenation and thereby minimize secondary injury. However, recent clinical studies continue to demonstrate the deleterious effects of blood transfusion in severely injured patients. (4–6)

Current guidelines from the Advanced Trauma Life Support (ATLS) manual advocate for early use of blood transfusion in patients with evidence of hemorrhagic shock. (7) The transfusion of blood products in patients with severe TBI without evidence of hemorrhagic shock however is outside of the scope of ATLS. Brain Trauma Foundation guidelines for the management of patients with severe TBI also do not address the use of red blood cells or other blood products for resuscitation in the absence of shock. (8)

Recently, a multicenter randomized controlled trial was completed to evaluate the early use of hypertonic fluids to restore cerebral perfusion and to reduce cerebral edema.

Importantly, this trial focused on patients with a pre-hospital Glasgow Coma Scale (GCS) score of less than or equal to 8 without hemodynamic compromise consistent with hemorrhagic shock. While this trial did not demonstrate superior 6 month neurologic outcomes or survival with the use of hypertonic fluids compared to normal saline, it represents the largest prospective randomized clinical trial involving pre-hospital and early resuscitation of patients with suspected severe TBI in the absence of hemorrhagic shock. (9) We sought to identify the association between red cell transfusion and outcomes utilizing this patient cohort.

METHODS

We performed a retrospective review of data collected prospectively as part of a multicenter, double blinded, randomized, controlled trial. The study was conducted by the Resuscitation Outcomes Consortium (ROC), a multicenter clinical trial network including 11 regional clinical centers in the United States and Canada. The trial involved 114 emergency medical

services agencies within the catchment area served by the ROC. This 3-group trial compared a 250-mL bolus of 7.5% saline (hypertonic saline) vs. 7.5% saline/6% dextran 70 (hypertonic saline/dextran) vs. 0.9% saline (normal saline) as the initial resuscitation fluid administered to injured patients with suspected severe TBI in the out-of-hospital setting. (9)

Patient Population

Patients were included in the TBI cohort of the trial based on the following: blunt mechanism of injury, age 15 years or older and a pre-hospital GCS score of 8 or less without evidence of hemorrhagic shock. Hemorrhagic shock was defined by a pre-hospital systolic blood pressure (SBP) of 70 mm Hg or less, or of 71 to 90 mmHg with a concomitant heart rate (HR) of 108 beats per minute or greater. Exclusion criteria included known or suspected pregnancy, age younger than 15 years, out-of-hospital cardiopulmonary resuscitation, administration of more than 2000 mL of crystalloid or any amount of colloid or blood products prior to enrollment, severe hypothermia ($< 28^{\circ}\text{C}$), drowning, asphyxia due to hanging, burns involving more than 20% of total body surface area, isolated penetrating head injury, inability to obtain intravenous access, more than 4 hours between receipt of dispatch call to study intervention, prisoner status, and inter-facility transfer. For this analysis, we excluded those subjects who died in the field or were dead on arrival to the emergency department, and those who were missing key covariates.

Clinical Data Collection

Detailed pre-hospital and hospital data were prospectively collected through day 28 on all patients enrolled in the trial. Injury severity was determined using the Injury Severity Score (ISS) based on the Abbreviated Injury Score-98 (AIS). (10) The primary exposure of interest was the number of units of packed red blood cells (pRBC) transfused in the 24 hours following initial 911 call.

Outcome Measures

The primary outcome for this analysis was 28 day survival. Secondary outcomes included 24-hour survival, Acute Respiratory Distress Syndrome (ARDS) free survival through 28 days, Multiple Organ Dysfunction score (MODs), and neurologic status 6 months after injury based on the Extended Glasgow Outcome score (GOSE). (11) The GOSE was dichotomized to good outcome (moderate disability or good recovery), which was defined as GOSE score > 4 versus poor outcome (severe disability, vegetative state, or dead) GOSE score ≤ 4 . The definition for ARDS was based on the report of the American-European consensus conference on ARDS. (12) The Marshall criteria for the diagnosis of MODs was subject to patients having the required physiologic measurements available during their intensive care unit (ICU) stay. (13)

Data Analysis

To evaluate the association of red cell transfusion with dichotomous outcomes, multivariable logistic regression with robust standard errors was used, adjusting for age, sex, ISS, missing ISS, initial GCS, initial SBP, highest field HR, penetrating injury, parent study intervention, fluid use, and study site. Additionally, sensitivity models that also included initial hemoglobin and looked at categorical classification of volume of pRBC were performed. Data are presented as odds ratio (OR) with 95% confidence intervals (CI). Utilizing the same covariates, the association of red cell transfusion with the multiple organ dysfunction score was modeled using linear regression with robust standard errors.

The 6-month GOSE scores were missing in 13% of subjects who survived to hospital discharge. To minimize the risk of bias from these missing data, this outcome was analyzed

using multiple hot deck imputation, as was done in the primary results paper. (9,14) The imputation model was based on data from all TBI patients discharged alive from the hospital, using either one-month or discharge GOSE score, length of hospitalization, and treatment arm. Twenty imputations were used in the analysis.

RESULTS

Of the 1282 patients enrolled in the ROC interventional trial, 1186 patients were included in this analysis. Patients declared dead in the field (n=4) or on arrival to the emergency department (n=3) and those missing 28 day vital status (n=75) or other key covariates (n=14) were excluded from the analysis. The majority of the analysis cohort were young (average age 39.4 years), male (75.8%), and sustained a blunt mechanism of injury (98.5%). Patient and injury characteristics are outlined in table 1.

Admission physiology and initial laboratory values are included in Table 2. The mean systolic blood pressure on admission was approximately 140 mmHg with a mean heart rate of 98 bpm. Mean initial INR was 1.3, PTT 31.6, and platelet count of 234. The mean volume of fluid transfused in the pre-hospital setting was 0.9 liters (range of 0.03 to 5.65 liters). In the first 24 hours after arrival to the emergency department, the mean volume of fluid administered was 6.2 liters (range 0 to 42 liters), and 333 (28%) received pRBC transfusions. Of those who received pRBCs, the mean (sd) number of units was 5.2 (6.1).

The results of the multivariable logistic regression are given in Table 3. Death occurred within 24 hours of ED admittance in 157 subjects (13%) and 302 (26%) died within 28 days. The odds ratio for 28 day survival and 24 hour survival decreased significantly as patient age increased. Gender was not statistically significant in predicting 28 day survival. As the Injury Severity Score increased, there was a decrease in the odds of both 28 day and 24 hour survival. Further, increasing GCS was associated with increases in both 28 day and 24 hour survival.

Neither volume of pre-hospital crystalloid received nor the type of fluid used was statistically significantly associated with mortality. An increase in volume of blood transfused was associated with significant decreases in both 28 day and 24 hour survival. Sensitivity models using categorical classification of RBC gave consistent results. Likewise, there was a decreased 28 day survival with increasing volume of FFP administration. Volume of platelet transfusion had no significant effect on mortality.

Results of the multivariable logistic regression evaluating ARDS free survival are shown in Table 4. ARDS was noted in 79 (7%) patients before day 28, 14 of whom also died before 28 days. Increasing age, higher ISS and decreasing GCS were associated with decreased ARDS free survival. The volume of crystalloid administered was not associated with a change in ARDS free survival. However, the volume of FFP transfused was associated with a statistically significant decrease in ARDS free survival. Volume of pRBCs transfused did not affect ARDS free survival.

A categorical interaction of red cell transfusion stratified by the initial ED hemoglobin level is shown in Table 5. When the initial hemoglobin is greater than 10 g/dL, transfusion was associated with a decrease in 28 day survival, ARDS free survival and the 6-month GOSE. This association was not seen when the hemoglobin was 10 g/dl or less. A test for an interaction between initial hemoglobin and the effect of red cell transfusion was statistically significant for the 28 day survival and ARDS-free survival endpoints ($p<0.01$), but not 6-month GOSE ($p=0.41$).

Table 6 represents a linear regression to evaluate the effects of red cell transfusion on the MODs score. This shows that for each unit of pRBC's transfused, the MODs score is increased by an estimated 0.45 in the highest initial hemoglobin group. Again, the test for interaction between initial hemoglobin and the effect of red cell transfusion was significant ($p<0.01$).

DISCUSSION

The goal of transfusion in patients with severe TBI is to maximize brain tissue oxygenation and thereby minimize secondary injury. Anemia has been recognized as a contributor to secondary injury and has been associated with increased mortality and poor neurologic outcomes. (15,16,17) While the definition of anemia in clinical studies varies, one of the first studies in 1978 by Miller and associates defined anemia as a hematocrit less than 30 and demonstrated an association of anemia with increased mortality in patients with TBI. (15) Recently, the potential benefits of red cell transfusion have been questioned as blood transfusion in trauma patients has been shown to be associated with increased infection (18,19), multi-organ failure (6) and death. (4,20) While multiple studies have demonstrated an increase in brain tissue oxygenation with transfusion of packed red blood cells, this response can be variable with 21–46% of patients actually experiencing a decrease in brain tissue oxygenation. (21,22) Further, this increase in tissue oxygenation is transient and returns to baseline by 24 hours. (22) Transfusion of older blood has also been shown to decrease brain tissue oxygenation. (23) A thorough review by Utter and associates outlines the arguments for and against a liberal transfusion strategy in the setting of TBI. (3) Currently, however, there is no consensus as to what the optimum hemoglobin level should be after TBI or what clinical indications should trigger the need for transfusion. (24) In a survey of physicians at US trauma centers, transfusion practices vary widely among neurosurgeons, trauma surgeons and non-surgeon intensivists. (25)

The “Out-of-hospital hypertonic resuscitation following severe traumatic brain injury: a randomized controlled trial” study represents the largest randomized clinical trial of pre-hospital hypertonic resuscitation following severe traumatic brain injury. Importantly, this trial focused on patients with severe TBI in the absence of hemorrhagic shock. In our retrospective study of those patients, volume of red blood cells transfused was independently associated with a statistically significant decrease in both 24 hour and 28 day survival. In a sensitivity analysis, we stratified the patients by initial ED hemoglobin, and found that in patients with an initial hemoglobin level of 10 or higher, transfusion of red blood cells in the first 24 hours of their hospital stay was associated with a decreased 28 day survival, a decreased ARDS free survival and worse 6-month neurologic outcome based on the GOSE. Further, for each unit of red cells transfused, the MODs score was increased by 0.45 in those with an initial hemoglobin of 10 g/dL or higher. Therefore, our data suggest that transfusion of red blood cells in this patient population leads to worse outcomes.

We identified several limitations to this study. The first is use of the pre-hospital GCS as a marker of severe TBI. While our intention is to evaluate the effect of red cell transfusion on patients with severe TBI, pre-hospital GCS does not accurately predict the presence of TBI. (26) Of the 1282 patients enrolled in the controlled trial, 375 (29%) were found to not have an anatomic finding of TBI based on a Marshall score of 1 on the initial head CT. (9) Secondly, we were unable to elucidate what triggered the decision for transfusion in these patients. We were limited to an initial hemoglobin level and cannot be certain of the hemoglobin level at the time of transfusion. Blood transfusion could be a marker of developing hypotension or delayed hemorrhage and therefore be selective for a group with worse outcomes regardless of fluid management. Further, we were unable to identify which patients may have been on anticoagulation which may have been a confounding factor for

patients receiving FFP. Lastly, as noted in the initial study, TBI management in the hospital was not controlled and varied based on the preferences of the providers.

In summary, transfusion of red blood cells in patients with a pre-hospital GCS ≤ 8 and no evidence of hemorrhagic shock was associated with a decreased 24 hour and 28 day survival. Further, if the initial hemoglobin was >10 , transfusion of red blood cells was associated with worse outcomes, including decreased ARDS free survival, decreased 6 month neurologic outcome and increased multi-organ dysfunction. Minimizing red cell transfusion in this patient population may improve survival. Further research is required to define the optimal hemoglobin and optimal fluid resuscitation strategy for patients with severe TBI in the absence of hemorrhagic shock.

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Table 1**Demographic, Injury Severity, and Out-of-Hospital Care Characteristics**

Patient Characteristics	N=1186
Age (years) - mean (sd)	39.4 (18.7)
Gender	
Male - n (%)	899 (75.8%)
Female – n (%)	287 (24.2%)
Mechanism of Injury	
Blunt injury - n (%)	1168 (98.5%)
Penetrating injury - n (%)	21 (1.8%)
Initial Vital Signs	
Initial SBP ¹ - mean (sd)	134.2 (31.9)
Initial SBP not detectable - n (%)	11 (0.9%)
Initial Respiratory Rate - mean (sd)	16.8 (7.9)
Initial GCS - mean (sd)	5.2 (2.6)
Highest field HR - mean (sd)	104.9 (25.5)
Injury Severity Score	
ISS - mean (sd)	27.0 (15.6)
Head AIS - mean (sd)	3.5 (1.8)
Face AIS - mean (sd)	0.7 (1.0)
Chest AIS - mean (sd)	1.6 (1.8)
Abdomen AIS - mean (sd)	0.7 (1.2)
Extremity AIS – mean (sd)	1.0 (1.3)
External AIS – mean (sd)	0.7 (0.6)

Table 2

Admission Physiology and Laboratory Values

Physiology and Laboratory Values	
Vital Signs	
SBP - mean (sd)	139.3 (32.8)
Admission HR - mean (sd)	98.0 (25.1)
First temperature - mean (sd)	35.9 (1.3)
Arterial Blood Gas	
pH - mean (sd)	7.3 (0.1)
pCO ₂ - mean (sd)	43.1 (11.2)
paO ₂ - mean (sd)	261.7 (154.2)
Lactate - mean (sd)	3.6 (2.6)
Coagulation Studies	
INR - mean (sd)	1.3 (0.8)
PTT - mean (sd)	31.6 (22.5)
Platelet count - mean (sd)	233.6 (72.9)
Fibrinogen - mean (sd)	215.4 (100.5)

Table 3

Multivariable logistic regression evaluating 28 Day and 24 Hour Survival

	28 Day Survival		24 Hour Survival	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (years)		<0.01		<0.01
<20	ref		ref	
20–39	0.48 (0.21, 1.11)		0.66 (0.24, 1.78)	
40–60	0.32 (0.14, 0.74)		0.38 (0.14, 1.02)	
61–75	0.18 (0.07, 0.45)		0.18 (0.06, 0.51)	
>75	0.04 (0.01, 0.11)		0.06 (0.02, 0.16)	
Male	0.94 (0.61, 1.43)	0.77	0.57 (0.33, 0.97)	0.04
ISS	0.96 (0.95, 0.97)	<0.01	0.97 (0.95, 0.98)	<0.01
ISS Missing	0.09 (0.03, 0.25)	<0.01	0.07 (0.02, 0.21)	<0.01
Initial GCS	1.42 (1.27, 1.58)	<0.01	1.42 (1.25, 1.61)	<0.01
Initial SBP		<0.01		0.29
110	ref		Ref	
111–150	0.86 (0.55, 1.36)		1.03 (0.60, 1.77)	
>150	0.30 (0.18, 0.50)		0.72 (0.40, 1.27)	
Highest field HR		<0.01		0.03
90	ref		ref	
91–110	2.85 (1.78, 4.57)		1.26 (0.72, 2.21)	
>110	1.53 (1.02, 2.32)		0.61 (0.37, 1.01)	
Penetrating injury	0.20 (0.08, 0.51)	<0.01	0.33 (0.10, 1.12)	0.07
Study arm		0.69		0.59
Normal saline	ref		ref	
Hypertonic saline + dextran	1.13 (0.74, 1.73)		1.06 (0.61, 1.84)	
Hypertonic saline	1.19 (0.79, 1.81)		1.29 (0.78, 2.14)	
First ED Hemoglobin (g/dL)	1.17 (1.07, 1.28)	<0.01	1.24 (1.11, 1.39)	<0.01
Pre-hospital crystalloid (L)	0.99 (0.74, 1.32)	0.93	1.22 (0.85, 1.77)	0.28
ED/Hospital crystalloid, 0–24 hours (L)	1.05 (0.98, 1.11)	0.17	1.12 (1.02, 1.23)	0.02
RBC, 0–24 hours (Units)	0.92 (0.85, 1.00)	0.04	0.89 (0.79, 0.99)	0.03
FFP, 0–24 hours (Units)	0.88 (0.79, 0.99)	0.04	0.95 (0.84, 1.08)	0.44
Platelets, 0–24 hours (Units)	1.00 (0.82, 1.23)	0.97	1.46 (0.85, 2.51)	0.17

Table 4

Multivariable logistic regression evaluating ARDS free survival

ARDS Free Survival		
	OR (95% CI)	p-value
Age (years)		<0.01
<20	ref	
20–39	0.38 (0.18, 0.78)	
40–60	0.21 (0.10, 0.45)	
61–75	0.15 (0.07, 0.36)	
>75	0.04 (0.01, 0.10)	
Male	0.83 (0.55, 1.23)	0.35
ISS	0.95 (0.94, 0.96)	<0.01
ISS Missing	0.05 (0.02, 0.14)	<0.01
Initial GCS	1.32 (1.21, 1.44)	<0.01
Initial SBP		<0.01
110 [†]	ref	
111–150	1.15 (0.76, 1.73)	
>150	0.47 (0.30, 0.75)	
Highest field HR		<0.01
90	ref	
91–110	2.66 (1.74, 4.08)	
>110	1.45 (0.99, 2.13)	
Penetrating injury	0.24 (0.10, 0.58)	<0.01
Study arm		0.61
Normal saline	ref	
Hypertonic saline + dextran	1.11 (0.75, 1.65)	
Hypertonic saline	1.22 (0.82, 1.81)	
First ED Hemoglobin (g/dL)	1.15 (1.05, 1.25)	<0.01
Pre-hospital crystalloid (L)	1.09 (0.83, 1.45)	0.53
ED/Hospital crystalloid, 0–24 hours (L)	1.02 (0.96, 1.07)	0.58
RBC, 0–24 hours (Units)	0.93 (0.86, 1.01)	0.10
FFP, 0–24 hours (Units)	0.87 (0.78, 0.96)	<0.01
Platelets, 0–24 hours (Units)	1.07 (0.89, 1.28)	0.49

Table 5

Adjusted association of red cell transfusion and outcomes by initial Hgb level

Initial Hgb	28 Day Survival		ARDS Free Survival		6-month GOSE >4	
	OR (95% CI)	p-value	OR (95% CI)	p-value	OR (95% CI)	p-value
	<0.01		<0.01		0.41	
<7	0.95 (0.78, 1.16)		0.98 (0.79, 1.22)		0.88 (0.57, 1.34)	
7-10	0.99 (0.91, 1.09)		1.00 (0.92, 1.09)		0.92 (0.84, 1.02)	
>10	0.83 (0.74, 0.93)		0.82 (0.74, 0.92)		0.82 (0.71, 0.94)	

Table 6

Adjusted association of red cell transfusion with MODs score

Initial Hgb	MODs Score	
	Coef (95% CI)	p-value
		<0.01
<7	−0.05 (−0.51, 0.42)	
7–10	−0.07 (−0.28, 0.14)	
>10	0.45 (0.19, 0.70)	