The Presence of Anemia in Children with Abusive Head Trauma

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Objectives To evaluate the incidence of anemia in patients with abusive head trauma (AHT), noninflicted traumatic brain injury (TBI), and physical abuse without AHT and the effect of anemia on outcome.

Study design In a retrospective, single-center cohort study, we included children under the age of 3 years diagnosed with either AHT (n = 75), noninflicted TBI (n = 77), or physical abuse without AHT (n = 60) between January 1, 2014, and December 31, 2016. Neuroimaging was prospectively analyzed by pediatric neuroradiologists. Primary outcome was anemia at hospital presentation. Secondary outcomes included unfavorable outcome at hospital discharge, defined as a Glasgow Outcome Scale between 1 and 3, and intracranial hemorrhage (ICH) volume.

Results Patients with AHT had a higher rate of anemia on presentation (47.3%) vs noninflicted TBI (15.6%) and physical abuse without AHT (10%) ($P < .001$). Patients with AHT had larger ICH volumes (33.3 mL [10.1-76.4 mL] vs 1.5 mL [0.6-5.2 mL] ; $P < .001$) and greater ICH/total brain volume percentages than patients with noninflicted TBI (4.6% [1.4-8.2 %] vs 0.2% [0.1-0.7%]; $P < .001$). Anemia was associated with AHT (OR, 4.7; 95% CI, 2.2-10.2) and larger ICH/total brain volume percentage (OR, 1.1; 95% CI, 1.1-1.2) in univariate analysis. Unfavorable outcome at hospital discharge was associated with anemia (OR, 4.4; 95% CI, 1.6-12.6) in univariate analysis, but not after controlling for covariates.

Conclusions Patients with AHT were more likely to present to the hospital with anemia and increased traumatic ICH volume than patients with noninflicted TBI or physical abuse without AHT. Children with anemia and AHT may be at increased risk for an unfavorable outcome. *(J Pediatr 2020;223:148-55)*.

Abusive head trauma (AHT), defined by the Centers for Disease Control and Prevention as "an injury to the skull or intracranial contents of an infant or young child (<5 years of age) due to inflicted blunt impact and/or violent shaking," is the leading cause of death from traumatic brain injury (TBI) in infants.[1,2] In the US, approximately 1 in 3000 infants are victims of AHT each year, resulting in 1800 hospitalizations and 160 deaths.[3-5] Outcomes after AHT are generally poor, with reported mortality as high as 35% and significant morbidity, including seizures, blindness, and severe developmental delay.[6-9] The average annual cost in the US of AHT hospital visits alone has been estimated at $69.6 million.[10]

One of the mainstays of TBI management is ensuring adequate oxygen delivery to injured brain tissue.[11-13] Because hemoglobin is the main determinant of oxygen content in the blood and subsequent delivery to tissues, the ideal hemoglobin level in critically ill patients overall has been heavily researched.[14-18] Anemia has previously been associated with AHT and unfavorable outcome after noninflicted TBI.[19-25] The AHT population differs from the noninflicted TBI population in several key areas, including the mechanism of trauma and role of secondary hypoxic-ischemic injury, and the cause and implications of anemia in this specific population warrants additional investigation.[26-28]

Our objectives were to identify patient and injury characteristics that are associated with anemia on hospital presentation in 3 groups: patients with AHT, noninflicted TBI, and physical abuse without AHT. We hypothesized that anemia would be associated with AHT, intracranial hemorrhage (ICH) volume, presence of solid organ injury, and unfavorable outcome at hospital discharge.

CT	Computed tomography
AHT	Abusive head trauma
GCS	Glasgow Coma Scale
ICH	Intracranial hemorrhage
NIRS	Near-infrared spectroscopy
$PbrO_2$	Brain tissue oxygenation
PICU	Pediatric intensive care unit
PIM2	Pediatric Index of Mortality 2
SDH	Subdural hematoma
TBI	Traumatic brain injury
TBV	Total brain volume

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Methods

This retrospective single-center cohort study with prospective neuroimaging analysis was conducted at the University of Pittsburgh Medical Center Children's Hospital of Pittsburgh. This study was approved by the University of Pittsburgh Institutional Review Board.

Patients were included for study if they were less than 3 years of age; diagnosed with AHT, noninflicted TBI, or physical abuse without AHT; and evaluated at Children's Hospital of Pittsburgh between January 1, 2014 and December 31, 2016. All patients were identified using existing Child Advocacy Center and trauma databases. Exclusion criteria included known coagulation disorder, use of anticoagulant medication before presentation, a documented anatomically abnormal brain before trauma, lack of admission hemoglobin value, penetrating trauma, and ICH as a result of birth trauma. The diagnosis of AHT and/or physical abuse was made by a multidisciplinary child protection team.

Patients in the physical abuse without AHT group had extracranial injuries (fractures and/or bruising) that were the result of physical abuse. These patients had neuroimaging performed as clinically indicated; all studies performed were negative for ICH. Given the large number of these patients during the study period, a subset of 60 patients was selected for inclusion in a blinded fashion using a simple randomization scheme to approximately match the number of patients in the AHT (75 patients) and noninflicted TBI (77 patients) groups.

The primary outcome was anemia at hospital presentation. Anemia was defined as a serum hemoglobin concentration that was less than the lower limit of normal based on our institution's laboratory cutoff; this cutoff is based on a hemoglobin or hematocrit at or below the 2.5th percentile for age, sex, and race.[29] Secondary outcomes were unfavorable outcome at hospital discharge, ICH volume compared with total brain volume (TBV), abnormal prothrombin time or partial thromboplastin time, requirement of a packed red blood cell transfusion, and presence of solid organ injury, fractures, or bruising. Unfavorable outcome was defined as a Glasgow Outcome Scale score of 1 (death), 2 (severe disability), or 3 (moderate disability).[30] A favorable outcome was a Glasgow Outcome Scale score of 4 (mild disability) or 5 (no disability).

Data Collection and Definitions

Data were abstracted from the electronic health record and included patient demographics (age, sex, race, year of presentation), growth measures on hospital presentation (weight, length, head circumference, adjusted for age and sex but not for prematurity), initial Glasgow Coma Scale (GCS) score, length of stay, severity of illness score at hospital admission, need for mechanical ventilation or neurosurgical intervention, disposition, hospital discharge Glasgow Outcome Scale score, cranial and extracranial injury characteristics, laboratory values, and blood transfusion and fluid resuscitation requirements within 48 hours of hospital presentation. Prospective measurement of ICH volume and TBV on first cranial computed tomography (CT) scan for children in the AHT or noninflicted TBI cohorts were completed by 2 pediatric neuroradiologists using the ABC/2 method. The ABC/2 equation provides a validated estimation of intracranial hemorrhage volume based on CT scan where A = maximum hemorrhage length in centimeters, B = width of hemorrhage at the point perpendicular to A on the same CT slice, and C = the number of CT slices where hemorrhage is present multiplied by the slice thickness.[31-33] Because brain magnetic resonance imaging was not obtained for all patients, only cranial CT data were analyzed. Subdural hematomas (SDH) were classified by radiologists as either hyperdense, hypodense, or mixed density (both hyperdense and hypodense). Initial GCS score was defined as the first documented GCS score after the patient's arrival to our institution; a GCS of 8 or below indicated severe TBI. Severity of illness was defined by the Pediatric Index of Mortality 2 (PIM2) score and was assigned based on available admission information; scores were only assigned to patients admitted to the pediatric intensive care unit (PICU) because they have only been validated in this population.[34] The clinical practice at our institution is to admit all young children less than 3 years of age with ICH and suspicion of AHT to the PICU. Neurosurgical intervention was defined as placement of any intracranial device, decompressive craniectomy, hematoma evacuation, or elevation of depressed skull fracture. Initial laboratory values were defined as those values first obtained after patients' arrival at our institution; the highest and lowest laboratory values within the first 48 hours after patients' arrival were also recorded. Fractures were identified based on documented radiographic interpretation by attending pediatric radiologists at the time of initial presentation. Solid organ injury was defined as evidence of organ injury based on documented radiographic interpretation by attending pediatric radiologists or laboratory abnormalities at the time of initial presentation. Bruising was identified based on documented physical examination at time of initial presentation.

Statistical Analyses

Descriptive statistics were presented as number (%) or median (IQR) because the continuous variables were nonparametric. The Fisher exact test was used for categorical variables. For continuous variables, the Wilcoxon rank-sum (Mann-Whitney) test was used for analysis of 2 groups, and the Kruskal-Wallis test was used for analysis of 3 groups. Univariate analysis was performed to determine the association between variables and anemia on presentation. Multivariable regression to determine variables associated with anemia on presentation included variables with univariate association with anemia ($P < .2$). We examined the outcome of unfavorable outcome at hospital discharge with similar univariate and multivariable approaches to ascertain whether anemia had an independent relationship with discharge outcome.

All statistical analysis was completed using Stata statistical software, version 14 (StataCorp, College Station, Texas).

Results

A total of 212 patients were included: 75 (35.4%) with AHT, 77 (36.3%) with noninflicted TBI, and 60 (28.3%) with physical abuse without AHT (Table I). Overall, the median patient age was 5.5 months (IQR, 2.0-15.5 months); groups were similar with regard to age, sex, and race. Children with any TBI (abusive or noninflicted) had larger head circumferences compared with children without TBI (AHT, 81st percentile [IQR, 61st-97th percentile] vs noninflicted TBI, 81st percentile [IQR, 47th-93rd percentile] vs physical abuse without AHT, 54th percentile [IQR, 22nd-84th percentile]; $P = .003$). There was no difference in weight and height growth percentiles between the 3 groups. More patients with AHT presented with severe TBI (n = 19 [25.3%]) compared with noninflicted TBI (n = 5 [6.5%]; $P < .001$). Patients with AHT had longer PICU lengths of stay ($P < .001$), and a greater number required mechanical ventilation ($P < .001$) and neurosurgical intervention ($P < .001$) than the other patient cohorts. Of the 19 patients with AHT with severe TBI, 10 (52.6%) had intracranial pressure monitors placed; 3 of the 5 (60%) of patients with noninflicted severe TBI had intracranial pressure monitors placed. Eight of the 9 children who died by hospital discharge had AHT. More patients with AHT had unfavorable outcome at hospital discharge (n = 12 [16%]) vs noninflicted TBI (n = 4 [5.2%]) or physical abuse without AHT (n = 0; $P < .001$).

Anemia Epidemiology

Patients with AHT had a higher rate of anemia on presentation (47.3%) vs noninflicted TBI (15.6%) and physical abuse

Table I. Patient characteristics

Variables	Overall sample n = 212	AHT (n = 75; 35.4%)	Noninflicted TBI (n = 77; 36.3%)	Abuse without AHT (n = 60; 28.3%)	*P* value
Age, mo*	5.5 (2.0-15.5)	4.0 (2.0-9.0)	9.0 (2.0-20.0)	6.0 (2.0-19.5)	.082
Female sex	93 (43.9)	30 (40.0)	36 (46.8)	27 (45.0)	.684
Race					.700
White	171 (80.7)	59 (78.7)	64 (83.1)	48 (80.0)	
Black	35 (16.5)	14 (18.7)	9 (11.7)	12 (20.0)	
Other	6 (2.8)	2 (2.7)	4 (5.2)	0 (0.0)	
Year					.929
2014	62 (29.3)	20 (26.7)	22 (28.6)	20 (33.3)	
2015	74 (34.9)	26 (34.7)	28 (36.4)	20 (33.3)	
2016	76 (35.9)	29 (38.7)	27 (35.1)	20 (33.3)	
Growth parameters					
Weight percentile	42.1 (15.4-65.4)	34.7 (12.0-61.6)	47.7 (18.0-69.5)	35.7 (10.8-71.1)	.217
Length percentile	26.9 (3.5-64.4)	21.0 (0.8-61.7)	32.3 (7.6-70.1)	27.3 (0.3-65.6)	.189
Head circumference percentile	76.9 (38.2-94.0)	81.2 (61.3-97.1)	81.1 (46.8-92.9)	53.9 (22.1-84.3)	.003
Initial GCS	15 (15-15)	15 (8-15)	15 (15-15)	15 (15-15)	<.001
Severe TBI (GCS ≤8)	24 (11.3)	19 (25.3)	5 (6.5)	N/A	<.001
Length of stay, d					
Overall	2 (1-5)	5 (3-12)	2 (1-3)	1 (1-2)	<.001
PICU	1 (0-2)	2 (1-6)	1 (0.5-1)	0 (0)	<.001
Acute care floor	1 (1-3)	3 (2-5)	1 (1-1)	1 (1-2)	<.001
Admitted to PICU	122 (57.5)	64 (85.3)	57 (74.0)	1 (1.7)	<.001
PIM2*	1.0 (0.9-1.6)	1.1 (0.9-5.1)	1.0 (0.9-1.1)	0.8 (0.8-0.8)	.030
Mechanically ventilated during admission	32 (15.1)	25 (33.3)	7 (9.1)	0 (0.0)	<.001
Required neurosurgical intervention	28 (13.2)	19 (25.3)	9 (11.7)	0 (0.0)	<.001
Intracranial pressure monitor†	17 (8.0)	13 (17.3)	4 (5.2)	0 (0.0)	<.001
External ventricular drain	16 (7.5)	13 (17.3)	3 (3.9)	0 (0.0)	<.001
Intraparenchymal intracranial pressure monitor	12 (5.7)	8 (10.7)	4 (5.2)	0 (0.0)	.022
Brain tissue oxygen monitor	6 (2.8)	4 (5.3)	2 (2.6)	0 (0.0)	.008
Decompressive craniectomy	5 (2.4)	5 (6.7)	0 (0.0)	0 (0.0)	.007
Hematoma evacuation and/or drain placement	9 (4.3)	6 (8.0)	3 (3.9)	0 (0.0)	.070
Elevation of depressed skull fracture	2 (0.9)	0 (0.0)	2 (2.6)	0 (0.0)	.334
Transferred from outside hospital	109 (51.4)	41 (54.7)	39 (50.7)	29 (48.3)	.744
Disposition					<.001
Died	9 (4.3)	8 (10.7)	1 (1.3)	0 (0.0)	
Home	124 (58.5)	21 (28.0)	73 (94.8)	30 (50.0)	
Foster care or kinship placement	65 (30.7)	35 (46.7)	0 (0.0)	30 (50.0)	
Rehabilitation facility	13 (6.1)	10 (13.3)	3 (3.9)	0 (0.0)	
Other (long-term care facility)	1 (0.5)	1 (1.3)	0 (0.0)	0 (0.0)	
Hospital discharge GOS					.001
GOS 1-3	16 (7.6)	12 (16.0)	4 (5.2)	0 (0.0)	
GOS 4-5	196 (92.5)	63 (84.0)	73 (94.8)	60 (100.0)	

GOS, Glasgow Outcome Scale.
Data are number (%) or median (IQR) unless otherwise indicated.
*Scores calculated only for patients admitted to the PICU.
†Includes patients that had either an external ventricular drain, intraparenchymal intracranial pressure monitor, or both.

Table II. Laboratory characteristics

Variables	Total (n = 212)	AHT (n = 75)	Noninflicted TBI (n = 77)	Abuse without AHT (n = 60)	*P* value
Patients with anemia, n (%)	53 (25.0)	35 (47.3)	12 (15.6)	6 (10.0)	<.001
Time to initial hemoglobin, h*	4 (3-6)	4 (2-6)	4 (3-5)	4 (3-7)	.204
Initial hemoglobin, g/dL	11.3 (10.2-12.1)	10.3 (9.0-11.1)	11.8 (11.2-12.7)	11.8 (10.9-12.2)	<.001
Lowest hemoglobin within 48 h of admission, g/dL	10.7 (8.8-11.9)	8.6 (7.5-10.1)	11.4 (10.2-12.5)	11.8 (10.8-12.2)	<.001
Initial platelet count, $\times 10^9$	382 (301-477)	396 (309-467)	370 (303-449)	381 (300-486)	.714
Initial PT, s†	13.7 (13.1-14.5)	14.3 (13.6-15.3)	13.6 (13.0-14.5)	13.4 (12.7-14.0)	<.001
Initial PTT, s‡	31 (28-34)	31 (27-34)	32 (29-36)	31 (29-34)	.266
Initial D-dimer, μg/mL§	1.4 (0.8-3.8)	1.6 (0.8-5.1)	1.3 (0.9-2.9)	1.0 (0.8-3.6)	.817
Initial fibrinogen, mg/dL¶	267 (190-315)	263 (190-309)	298 (202-377)	305 (305-305)	.462
Initial MCV, fL	82.6 (79.1-87.5)	83.5 (79.3-87.1)	81.9 (79.0-86.0)	81.6 (78.9-87.9)	.582
Initial MCH, pg	27.8 (26.4-29.8)	27.9 (26.4-29.6)	27.6 (26.5-29.4)	27.9 (26.3-30.0)	.925
Initial MCHC, g/dL	34.3 (33.0-34.3)	33.3 (32.7-34.2)	33.7 (33.0-34.3)	33.9 (33.3-34.3)	.076
Initial RDW, %	13.9 (13.0-15.0)	14.1 (13.1-15.1)	13.8 (13.0-14.9)	13.9 (13.0-15.0)	.874

MCH, mean corpuscular hemoglobin; *MCHC*, mean corpuscular hemoglobin concentration; *MCV*, mean corpuscular volume; *PT*, prothrombin time; *PTT*, partial thromboplastin time; *RDW*, red blood cell distribution width.
Data are median (IQR) unless otherwise indicated.
*Time in hours from initial utilization of healthcare system (activation of emergency medical services, emergency department visit, pediatrician's office visit) to initial hemoglobin measurement at our institution.
†There were 204 patients who had a documented PT (74 patients with AHT, 75 patients with noninflicted TBI, and 55 abuse without patients with AHT).
‡There were 203 patients who had a documented PTT (74 patients with AHT, 74 patients with noninflicted TBI, and 55 abuse without patients with AHT).
§There were 48 patients who had a documented D-dimer (34 patients with AHT, 8 patients with noninflicted TBI, and 6 abuse without patients with AHT).
¶There were 49 patients who had a documented fibrinogen (40 patients with AHT, 8 patients with noninflicted TBI, and 1 abuse without AHT patient).

without AHT (10%; $P < .001$) (Table II). Patients with AHT had a lower presenting hemoglobin (10.3 g/dL; IQR, 9.0-11.1 g/dL) vs noninflicted TBI (11.8 g/dL; IQR, 11.2-12.7 g/dL) vs physical abuse without AHT (11.8 g/dL; IQR, 10.9-12.2 g/dL) ($P < .001$) and a lower hemoglobin nadir within 48 hours of admission (8.6 g/dL; IQR, 7.5-10.1 g/dL) vs noninflicted TBI (11.4 g/dL; IQR, 10.2-12.5 g/dL) vs physical abuse without AHT (11.8 g/dL; IQR, 10.8-12.2 g/dL; $P < .001$). Anemia was normocytic with normal red blood cell distribution width for all groups. Patients with AHT had a longer prothrombin time on admission (14.3 seconds; IQR, 13.6-15.3 seconds) compared with noninflicted TBI (13.6 seconds; IQR, 13.0-14.5 seconds) and physical abuse without AHT (13.4; IQR, 12.7-14.0 seconds; $P < .001$). More patients with AHT received packed red blood cell transfusions within 48 hours of admission (33.3%) vs noninflicted TBI (9.1%) and physical abuse without AHT (0%) ($P < .001$) (Table III; available at www.jpeds.com).

Cranial CT Analysis

Comparing AHT and noninflicted TBI groups, more patients with noninflicted TBI had skull fractures than patients with AHT (77.9% vs 30.7%) ($P < .001$) on cranial CT. Patients with AHT had markedly larger ICH volumes (33.3 mL [IQR, 10.1-76.4 mL] vs 1.5 mL; IQR, 0.6-5.2 mL; $P < .001$) and larger ICH/TBV percentages than patients with TBI (4.64% [IQR, 1.4-8.2%] vs 0.2% [IQR, 0.1-0.7%]; $P < .001$). patients with AHT were more likely to have cerebral edema (20% vs 3.9%; $P = .002$) and SDH (89.3% vs 54.5%; $P < .001$) compared with patients with noninflicted TBI (Figure 1; available at www.jpeds.com) (Table IV). Only 1 of the 8 patients with AHT (12.5%) with isolated hypodense SDH was anemic on admission.

Extracranial Injuries

Compared with patients with noninflicted TBI, patients with AHT had more extremity and rib fractures (30 [40%] vs 4 (5.2%]), external bruising (47 [62.7%] vs 12 [15.6%]),

Table IV. Cranial imaging

Variables	Total (n = 152)	AHT (n = 75)	Noninflicted TBI (n = 77)	*P* value
Cerebral edema	18 (11.8)	15 (20.0)	3 (3.9)	.002
Skull fracture	83 (39.2)	23 (30.7)	60 (77.9)	<.001
SDH	109 (71.7)	67 (89.3)	42 (54.5)	<.001
Hyperdense only	75 (49.3)	35 (46.7)	40 (52.0)	
Hypodense only	8 (5.3)	8 (10.7)	0 (0.0)	
Mixed density (both hyperdense and hypodense)	26 (17.1)	24 (32.0)	2 (2.6)	
Extradural hemorrhage	31 (20.4)	4 (5.3)	27 (35.0)	<.001
SAH	26 (17.1)	16 (21.3)	10 (13)	.200
Intraparenchymal hemorrhage	13 (8.6)	5 (6.7)	8 (10.4)	.564
Multiple hemorrhage types	21 (13.8)	13 (17.3)	8 (10.4)	.246
ICH volume, mL	6.8 (1.1-34.5)	33.3 (10.1-76.4)	1.5 (0.6-5.2)	<.001
TBV, mL	825 (603-984)	812 (604-962)	854 (602-1024)	.396
ICH/TBV percentages, median (IQR)	0.9 (0.1-5.2)	4.6 (1.4-8.2)	0.2 (0.1-0.7)	<.001

SAH, subarachnoid hemorrhage.
Data are number (%) unless otherwise indicated.

and solid organ injury (12 [16%] vs 2 [2.6%]), particularly liver injury (9 [12%] vs 1 [1.3%]]) (Figure 2; available at www.jpeds.com).

Factors Associated with Anemia on Hospital Presentation

In univariate analysis, anemia on presentation was associated with mortality (OR, 6.6; 95% CI, 1.6-27.6; $P = .009$), solid organ injury (OR, 5.2; 95% CI, 1.8-15.5; $P = .003$), AHT (OR, 4.7; 95% CI, 2.2-10.; $P < .001$), unfavorable outcome at hospital discharge (OR, 4.7; 95% CI, 1.4-15.5; $P = .011$), larger ICH/TBV percentage (OR, 1.1; 95% CI, 1.1-1.2; $P < .001$), older age (OR, 1.0; 95% CI, 1.00-1.1; $P = .025$), and worse initial GCS score (OR, 0.8; 95% CI, 0.7-0.9; $P < .001$) (Table V). In multivariable analysis, AHT (OR, 3.9; 95% CI, 1.5-10.7; $P = .007$), older age (OR, 1.1; 95% CI, 1.0-1.1; $P = .001$), and lower initial GCS score (OR, 0.9; 95% CI, 0.8-1.0; $P = .003$) were associated with anemia on hospital presentation.

Factors Associated with Unfavorable Outcome at Hospital Discharge

In univariate analysis, unfavorable outcome at hospital discharge was associated with severe TBI with an initial GCS of 8 or below (OR, 130.2; 95% CI, 26.0-652.9; $P < .001$), anemia at hospital presentation (OR, 4.4; 95% CI, 1.6-12.6; $P = .005$), AHT (OR, 3.5; 95% CI, 1.1-11.3; $P = .039$), and PIM2 score (OR, 1.9; 95% CI, 1.4-2.6; $P < .001$) (Table V). In multivariable analysis with severe TBI, anemia, and AHT as covariates, severe TBI (OR, 112.1; 95% CI, 16.2-777.8; $P < .001$) was associated with an unfavorable outcome at hospital discharge.

Discussion

We investigated variables associated with anemia in young children with AHT, noninflicted TBI, and physical abuse without AHT to inform modifiable elements of care that may improve outcomes. One important finding is that patients with AHT were more severely ill on presentation; they had lower initial GCS scores, longer PICU lengths of stay, higher severity of illness scores, required more organ support including mechanical ventilation and neurosurgical intervention, and had higher mortality and morbidity than the other patient groups. Next, nearly one-half the patients with AHT presented with anemia, consistent with the findings of Acker et al.[19] These findings have important clinical implications.

We included the comparison group of patients with physical abuse without AHT to investigate the role that non-AHT-related abuse and neglect and/or extracranial injuries may play in the development of anemia. Interestingly, noninflicted TBI and physical abuse without patients with AHT had similar hemoglobin levels on admission with the physical abuse group having the lowest rate of anemia. We did not find an association between isolated hypodense SDH and anemia. Generally, anemia for all patients was normocytic with a normal red blood cell distribution width, which can be seen with both acute blood loss and/or anemia of chronic disease.[35,36] Despite reports that malnutrition can manifest in

Table V. Univariate and multivariable regression for associations with anemia on hospital presentation and unfavorable outcome at hospital discharge

Variables	Univariate OR (95% CI)	*P* value	Multivariable OR (95% CI)	*P* value
Anemia on hospital presentation				
Mortality	6.6 (1.6-27.6)	.009	*	
Solid organ injury	5.2 (1.8-15.5)	.003	*	
AHT	4.7 (2.2-10.2)	<.001	3.9 (1.5-10.7)	.007
Unfavorable hospital discharge outcome	4.7 (1.4-15.5)	.011	*	
Fracture, excluding skull	1.4 (0.8-2.7)	.280	NS	
Sex	1.3 (0.7-2.5)	.380	NS	
ICH/TBV percentage	1.1 (1.1-1.2)	<.001	1.1 (1.0-1.2)	.154
Race	1.1 (0.8-1.6)	.542	NS	
Age, mo	1.0 (1.0-1.1)	.025	1.1 (1.0-1.1)	.001
Initial MCV	1.0 (1.0-1.0)	.588	NS	
Initial GCS	0.8 (0.7-0.9)	<.001	0.9 (0.8-1.0)	.003
Unfavorable outcome at hospital discharge				
Severe TBI (GCS ≤8)	130.2 (26.0-652.9)	<.001	112.1 (16.2-777.8)	<.001
Anemia at hospital presentation	4.4 (1.6-12.6)	.005	0.6 (0.1-3.0)	.528
AHT	3.5 (1.1-11.3)	.039	1.0 (0.2-5.2)	.983
PIM2	1.9 (1.4-2.6)	<.001	†	
Sex	1.3 (0.5-3.6)	.608	†	
Race	1.2 (0.8-2.0)	.416	†	
ICH/TBV percentage	1.1 (1.0-1.2)	.204	†	
Age, mo	1.0 (1.0-1.1)	.854	†	

NS, data that was not significant in univariate analysis was not entered in the multivariable analysis.
*Although significant on univariate analysis, sample sizes were too small to use in the multivariable analysis.
†Given the small sample size of patients with unfavorable outcome (n = 16), only the 3 most significant covariates on univariate analysis were entered into the multivariable analysis.

poor growth and anemia in children, there were no differences in height or weight among patient cohorts.[37] Patients with TBI, regardless of mechanism, had a larger head circumference than patients with physical abuse without TBI.

We found that patients with AHT had median ICH volumes of nearly 5% of their TBV, more than 20 times higher than median ICH volumes in patients with noninflicted TBI. There is published pediatric data that an ICH volume of more than 2% of TBV is associated with moderate disability and an ICH volume of 4% or greater of TBV is associated with severe disability and death.[38-40] These studies, however, included only intraparenchymal hemorrhage and intraventricular hemorrhage in their calculations of ICH volume, and we included extradural hemorrhage and SDH in our calculations as well. Although it is difficult to directly compare the effects of extradural hemorrhage and SDH volumes with intraparenchymal hemorrhage and intraventricular hemorrhage volumes given their extracerebral vs intracerebral locations, we do believe that given the known association with 2%-4% ICH volume and unfavorable outcome, a 5% ICH volume is clinically significant and may contribute to anemia.

We found mixed results in relating extracranial trauma with anemia. Fractures and bruising alone did not seem to be associated with anemia. Solid organ injury was associated with anemia, and patients with AHT had significantly higher rates of solid organ injury, particularly liver injury. Because traumatic liver injuries may cause significant blood loss leading to anemia, we cannot exclude the possibility that the increased rate of liver injury in the AHT patient cohort may have contributed to the higher rate of anemia in these patients.[41,42] However, because nearly one-half of patients with AHT had anemia on presentation and only 12% had any degree of liver injury, our findings do not support liver injury or other extracranial injuries as the primary cause of anemia.

Coagulopathy after TBI can contribute to bleeding and anemia.[11,43-45] Patients with AHT had the highest prothrombin time on presentation consistent with prior studies.[45,46] Clinical significance of this finding is unclear, as median values remained within our laboratory's normal limits.[47] Patients with AHT also demonstrated higher rates of extracranial trauma, which can also cause coagulopathy.[48-50]

We found that patients with AHT had the lowest hemoglobin nadirs and more often received packed red blood cell transfusions within 48 hours of admission. Lower hemoglobin levels may not allow for sufficient oxygen delivery to the injured brain, which may exacerbate injury and worsen outcomes. Indeed, studies in adult trauma patients have found a significant association between anemia and unfavorable outcomes.[21-25,51] Robust data for this association in pediatric TBI are lacking. One previous study did not find an association between anemia and mortality in pediatric patients with severe TBI, but did find an association between AHT and mortality.[52] We found associations with both anemia and AHT with unfavorable outcome at hospital discharge on univariate analysis, but not on multivariable analysis with inclusion of an initial GCS of 8 or less. We hypothesize that the association of an initial GCS of 8 or less with unfavorable outcome is so strong that, given the small number of patients with unfavorable outcome (n = 16), we were unable to detect any independent relationship between anemia or AHT and outcome.

Given the vital need to optimize oxygen delivery to injured brain tissue, experts agree in recently published transfusion recommendations that neurocritically ill children should receive red blood cell transfusions when the hemoglobin level is less than 7.0 g/dL. The ideal hemoglobin threshold for transfusion, however, has not been determined in prospective trials, and pediatric patients with severe TBI may benefit from a higher hemoglobin threshold.[11,53,54] Given the unique mechanism of injury and role of secondary hypoxic-ischemic insult in AHT compared with noninflicted TBI, the role of anemia and ideal hemoglobin threshold for these patients merits further investigation.[26-28]

Brain tissue oxygenation ($PbrO_2$) monitoring is becoming more common in pediatric patients with TBI, and optimizing hemoglobin is one component of optimizing $PbrO_2$.[11,55-57] Although invasive $PbrO_2$ monitors are available, only 6 patients in our study had them placed. Near-infrared spectroscopy (NIRS) has been used as a noninvasive way to calculate and monitor cerebral oxygen saturation with some success, particularly in neonatal and perioperative populations.[58-60] Results in adult patients with TBI have been mixed.[61] NIRS monitoring may be more successful in pediatric patients than adults because of their thinner skulls and scalps, but there are limited data in the pediatric TBI population.[61,62] There have been no published studies examining the relationship between $PbrO_2$, NIRS, and autoregulation in the AHT population.

Our findings along with previous work support screening hemoglobin levels for children presenting with symptoms concerning for AHT.[19,20] Although it is the practice at our institution to initially admit and observe all patients with suspected AHT in the PICU, this is not the case at all institutions. Given our findings that several markers of severity of injury, including unfavorable outcome at hospital discharge, are associated with anemia on admission, it may be reasonable for the clinician to use this information to help guide their decision about initial triage and PICU admission.

Potential areas of future studies in patients with AHT include evaluation of the independent association of anemia with outcome; relationship of NIRS to $PbrO_2$ and outcome; ideal transfusion threshold; coagulation abnormalities specific to this population; and relationship between ICH/TBV percentages and outcome.

Our study was limited by the nature of a retrospective single-center design. More than one-half of patients first presented to an outside facility and may have had laboratory tests and interventions performed before transfer to our center, potentially affecting initial laboratory values that were used in our analysis. Laboratory studies often used to support the diagnosis of anemia of chronic disease (eg, reticulocyte count, serum iron concentration, ferritin, transferrin level,

transferrin saturation, or inflammatory markers) or assess nutritional status (eg, albumin, prealbumin, vitamin and mineral levels) were infrequently obtained and not analyzable.[35-7,63,64] We were unable to assess additional markers of coagulation abnormalities previously associated with AHT (eg, D-dimer) in this analysis because only a minority of patients had these values recorded.[65] Although all neuroimaging done in the patients with physical abuse without AHT was negative, we do not know how many had neuroimaging performed. As a result, we do not know whether there were differences in the patients who did or did not undergo neuroimaging.

We can only assert an association, not a causality, between anemia and the various outcomes. We were limited by the small event size of several variables that may be associated with anemia and were unable to include them in our final regression model, including solid organ injury and mortality. Given the small sample size of patients with unfavorable outcomes at hospital discharge, we were only able to include 3 variables in our multivariable regression, and it may still be at risk for sparse data bias.[66] We were also unable to adjust for injury severity and perform more advanced statistical analysis such as propensity matching given our small sample size.

Children with anemia and AHT may be at increased risk for unfavorable outcome. Future work is needed to better characterize and clarify key neurocritical care pathways in AHT. ■

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Data Statement

Data sharing statement available at www.jpeds.com.

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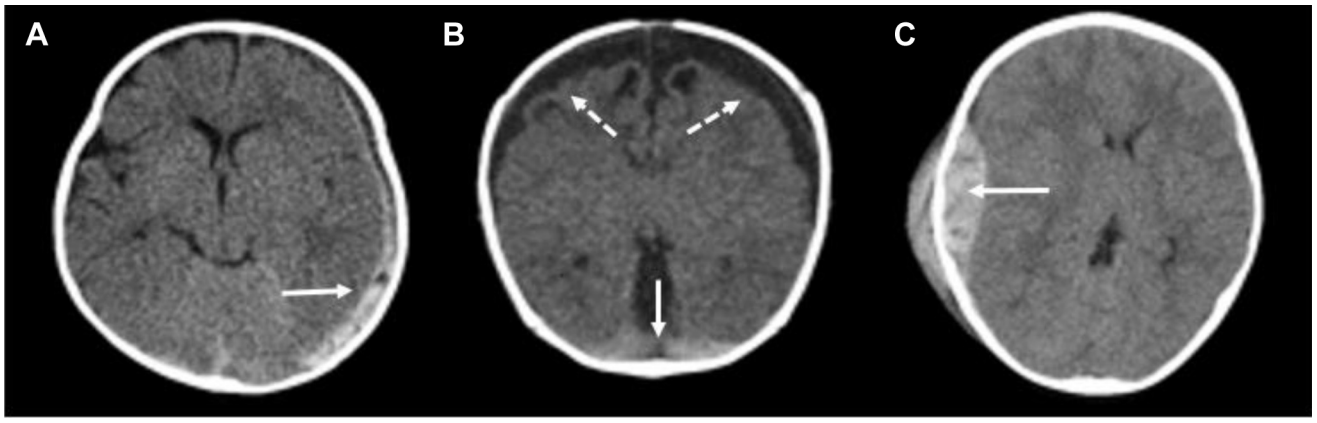

Figure 1. Cranial CT findings. **A,** A 5-month-old girl, with hyperdense SDH owing to AHT, with an ICH volume of 76.4 mL, TBV of 815.6 mL, ICH/TBV% of 9.37%. **B,** A 1-month-old boy with hyperdense (*solid arrow*) and hypodense (*dotted arrows*) SDH owing to AHT, with an ICH volume of 87.2 mL, TBV of 556.5 mL, and ICH/TBV% of 15.7%. **C,** A 13-month-old girl with a hyperdense extradural hemorrhage owing to noninflicted TBI, with an ICH volume of 71 mL, TBV of volume 917.2 mL, and ICH/TBV% of 7.7%.

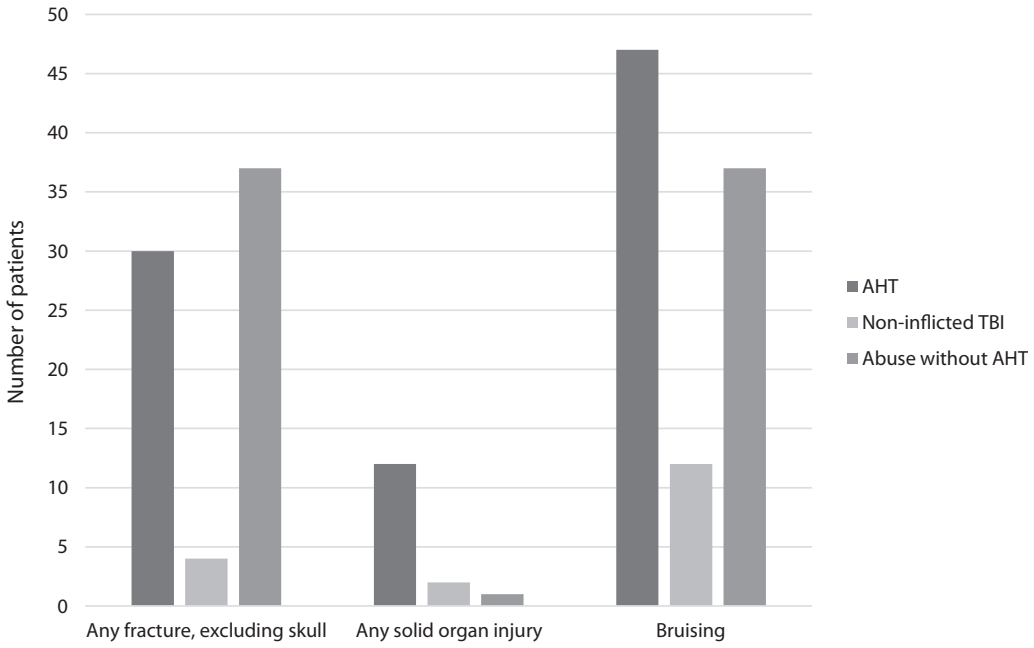

Figure 2. Extracranial injury characteristics. $P \leq .001$ for all variables.

Table III. Blood product transfusions and fluid resuscitation

Variables	Total (n = 212)	AHT (n = 75)	Noninflicted TBI (n = 77)	Abuse without AHT (n = 60)	*P* value
Received pRBC in first 48 h	32 (15.1)	25 (33.3)	7 (9.1)	0 (0.0)	<.001
Received platelets in first 48	4 (1.9)	4 (5.3)	0 (0.0)	0 (0.0)	.021
Received FFP in first 48	10 (4.7)	6 (8.0)	4 (5.2)	0 (0.0)	.060
Received crystalloid fluid resuscitation in first 48*	57 (26.9)	35 (46.7)	12 (15.6)	10 (16.7)	<.001

pRBC, packed red blood cell.
Data are number (%).
In addition, 3 patients with AHT received cryoprecipitate, 1 AHT patient received a whole blood transfusion, and 3 patients with AHT received supplemental Vitamin K.
*Calculated based on fluid boluses patient received and did not include maintenance fluid infusions.