# Evaluating abusive head trauma in children < 5 years old: Risk factors and the importance of the social history☆,☆☆

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## ARTICLE INFO

## ABSTRACT

*Background:* Abusive head trauma (AHT) is the leading cause traumatic death in children ≤5 years of age. AHT remains seriously under-surveilled, increasing the risk of subsequent injury and death. This study assesses the clinical and social risks associated with fatal and non-fatal AHT.

*Methods:* A single-institution, retrospective review of suspected AHT patients ≤5 years of age between 2010 and 2016 using a prospective hospital forensic registry data yielded demographic, clinical, family, psycho-social and other follow-up information. Descriptive statistics were used to look for differences between patients with AHT and accidental head trauma. Logistic regression estimated the adjusted odds ratios (AOR) for AHT. A receiver operating characteristic (ROC) curve was created to calculate model sensitivity and specificity.

*Results:* Forensic evaluations of 783 children age ≤5 years with head trauma met the inclusion criteria; 25 were fatal with median[IQR] age 23[4.5–39.0] months. Of 758 non-fatal patients, age was 7[3.0–11.0] months; 59.5% male; 435 patients (57.4%) presented with a skull fracture, 403 (53.2%) with intracranial hemorrhage. Ultimately 242 (31.9%) were adjudicated AHT, 335(44.2%) were accidental, 181 (23.9%) were undetermined. Clinical factors increasing the risk of AHT included multiple fractures ($Exp(\beta) = 9.9[p = 0.001]$), bruising ($Exp\beta = 5.7$ [p < 0.001]), subdural blood ($Exp(\beta) = 5.3[p = 0.001]$), seizures ($Exp(\beta) = 4.9[p = 0.02]$), lethargy/unresponsiveness ($Exp(\beta) = 2.24[p = 0.02]$), loss of consciousness ($Exp(\beta) = 4.69[p = 0.001]$), and unknown mechanism of injury ($Exp(\beta) = 3.9[p = 0.001]$); skull fracture reduced the risk of AHT by half ($Exp(\beta) = 0.5[p = 0.011]$). Social risks factors included prior police involvement ($Exp(\beta) = 5.9[p = 0.001]$), substance abuse ($Exp(\beta) = 5.7[p = .001]$), unknown number of adults in the home ($Exp(\beta) = 4.1[p = 0.001]$) and intimate partner violence ($Exp(\beta) = 2.3[p = 0.02]$). ROC area under the curve (AUC) = 0.90([95% CI = 0.86–0.93] p = .001) provides 73% sensitivity; 91% specificity.

*Conclusions:* To improve surveillance of AHT, interviews should include and consider social factors including caregiver/household substance abuse, intimate partner violence, prior police involvement and household size. An unknown number of adults in home is associated with an increased risk of AHT.

*Study Type/Level of Evidence:* Prognostic, Level III.

## 1. Background

Abusive Head Trauma (AHT) is a leading cause of death in infants [1] and children under 5 years of age [2]. Estimates show that in children age 5 and younger, AHT occurs in 20–30 per 100,000 [3–9]. An estimated 18–25% of children diagnosed with AHT die, and up to 80% of survivors will live with significant lifelong physical, developmental, and emotional sequelae [1,10–12]. A recent study estimated that the lifetime cost of a single patient with AHT can exceed $3 million in

medical costs, lost wages, disability, and government and educational services [13].

Less severe forms of AHT are seriously under-surveilled [14]. As many as 31% of children with AHT may be misdiagnosed on initial visit [15] and milder forms of AHT may be missed entirely [16,17]. AHT screening is subject to considerable provider variation as well as provider biases [15]. There is provider hesitancy in diagnosing AHT when many head injuries in non-verbal children may be well-appearing or patients present with symptoms similar to other conditions such as lethargy, vomiting or fussiness [15,16,18,19]. There are very few validated screening tools for AHT and they are not in wide use [18,20]. Yet, failure to detect AHT on the initial presentation substantially increases the risk of repeated injury and death [21–23]. This study assesses the associated clinical and social risks of AHT for fatal and non-fatal patients who present to the Emergency Department (ED) in a Level I Pediatric Trauma Center. Screening for AHT has important implications for early detection, prevention and timely coordination of medical and therapeutic interventions to protect infants and young children [24].

## 2. Methods

We performed a single-institution, retrospective review of a cohort of pediatric patients drawn from a prospective forensic registry at Phoenix Children's Hospital (PCH) between January 1, 2010 and December 31, 2016. The forensic registry contains data on all patients referred to the Child Protection team (CPT) for forensic evaluation of suspected physical abuse. A determination of AHT by the CPT was reserved strictly for patient injuries where an adult perpetrator with agency (and intent) could be specifically linked to the head injury and physical injuries were supported by medical data. Where the evaluation yielded definitive evidence of an unintentional head injury or head injuries where intentional infliction of injury could be ruled out were classified as accidental. Injuries where inflicted injury could not be ruled out or could not be adequately supported by medical evidence were classified as undetermined under the institution's CPT determination scheme. The CPT met weekly during the time frame of the study.

### 2.1. Data collection

All patients in the forensic registry aged 5 or younger who presented in the ED between 2010 and 2016 with a diagnosed head or brain injury alone or in combination with other injuries or conditions, or were admitted to the hospital with any fatal (in-hospital deaths only) or non-fatal head injuries where physical abuse was suspected were included in the study.

There were 782 (758 injured; 25 fatally injured) patients in the forensic registry with fatal or non-fatal head/brain injuries who met the inclusion criteria. Data on patient demographics, hospitalization, injury, laboratory tests, imaging, family characteristics, (e.g., suspected substance abuse, treatment for mental illness, intimate partner violence (IPV), prior involvement with police or child protective services (CPS)), discharge, determination and follow-up information were extracted from the institution's forensic registry. All data were collected and maintained in the PCH forensic registry under PCH IRB 09–055. This study was completed under PCH IRB 17–015.

### 2.2. Data analysis

Descriptive statistics, Mann–Whitney *U* tests and chi-square test statistics (with Yates correction) as appropriate were used to compare fatal vs. non-fatal injury forensic registry patients and forensic registry patients by case determination. Simple logistic regression were used to identify covariates of interest for inclusion in a multiple logistic regression model for AHT. Multiple logistic regression was then used to estimate adjusted odds ratios (AOR) for main effects while controlling for any potential confounders (a priori, from univariate or from appropriate collinearity

tests). Final model fit was evaluated based on the Akaike Information Criterion (AIC) and the Hosmer-Lemeshow goodness-of-fit statistic. A receiver operating characteristic (ROC) curve was calculated to determine area under the curve (AUC), model sensitivity and specificity. All patients with incomplete data were excluded from analyses on a case-wise basis by respective variable. SPSS version 26 was used for all data analyses. Statistical significance was set at $p < 0.05$ for all statistical tests.

## 3. Results

Forensic evaluations of 783 children ≤5 years old with head trauma met the inclusion criteria; 25 were fatal with a median[IQR] for age of 23[4.5–39.0] months. Of 758 non-fatal patients, median[IQR] age was 7 [3–11] months; 59.5% male; 435 patients (57.4%) presented with a skull fracture; 403 (53.2%) with intracranial hemorrhage. There were 31.8% (242) adjudicated AHT, 44.2% (335) were accidental, 23.8% (181) were undetermined. For the 25 fatal head injuries, all 25 (100%) were adjudicated AHT.

### 3.1. Head CT findings associated with AHT

Table 1 shows the computed tomography (CT) findings for all eligible fatally and non-fatally injured patients. Of the 758 non-fatalities, the head CT for 27 patients yielded normal or unremarkable results. In total, there were 965 CT abnormal findings for 731 non-fatal patients. Findings were as follows: 45.1% skull fracture, 20.4% subdural hemorrhage (SDH), 6.5% extra-axial hemorrhage (EAH), 5.0% epidural hemorrhage (EDH), 3.8% subarachnoid hemorrhage (SAH), 6% other intracranial hemorrhage, 15.5% other brain injury, and <1% hypoxic ischemic encephalopathy (HIE). There were 50 abnormal CT findings among the 25 patients with fatal injuries. Table 1 shows that skull fractures occurred in 10% of fatal head injuries but 45.1% of non-fatal head injuries ($p = 0.001$). Other intracranial hemorrhage occurred in 18% of fatal head injuries but only 6% of non-fatal head injuries ($p = 0.01$). There were no statistically significant differences between fatal and non-fatal head injuries found for EDH/SHD ($p = 0.30$), extra-axial hemorrhage ($p = 0.52$); or other brain injury ($p = 0.17$). The remaining imaging findings occurred too infrequently to compare statistically.

### 3.2. Fatal versus non-fatal AHT

For fatally injured patients, the median [IQR] for age in months (23 [4.5–39.0]) was statistically different from non-fatal injuries (7 [3–11])

**Table 1**
Abnormal computerized tomography (CT) findings for non-fatally and fatally injured head trauma patients with suspected Accidental Head Trauma (AHT).

| Head imaging by CT<br>N = 783 | Non-fatally injured patients<br>N = 758(%) | Fatally injured patients<br>N = 25(%) |
|---|---|---|
| Patients with normal findings | 27 (3.6) | 0 (0) |
| Patients with abnormal findings | N = 731 (96.4) | N = 25 (100.0) |
| Total number of abnormal imaging findings[a] | 965 | 50 |
| **Specific findings** | **Number of patients with abnormal finding<br>(% total findings)[a]** | |
| Skull fracture | 435 (45.1) | 5 (10.0) |
| Subdural hemorrhage | 197 (20.4) | 13 (26.0) |
| Epidural hemorrhage | 48 (5.0) | 1 (2.0) |
| Extra-axial hemorrhage | 63 (6.5) | 4 (8.0) |
| Subarachnoid hemorrhage | 37 (3.8) | 3 (6.0) |
| Other intracranial hemorrhage | 58 (6.0) | 9 (18.0) |
| Other brain injury, hypodensity, swelling, and edema | 125 (13.0) | 11 (22.0) |
| Hypoxic ischemic encephalopathy | 2 (<1.0) | 4 (8.0) |

[a] Patients may have more than one abnormal finding.

**Table 2**
Patient, injury, and family characteristics of AHT, accidental and undetermined head trauma for fatally and non-fatally injured patients.

| Characteristic | Non-fatally injured patients N = 758 | | | Fatally injured patients N = 25 |
|---|---|---|---|---|
| | AHT | Accidental | Undetermined | All determinations |
| | N = 242 (%) | N = 335 (%) | N = 181 (%) | N = 25(%) |
| **Patient Age** | | | | |
| Median [IQR] months | 7 [3–10] | 7 [3–16] | 8 [3–12] | 23 [4.5–39.0] |
| 0–12 m | 182 (75.2) | 275 (82.1) | 137 (75.7) | (32.0) |
| 13–24 m | 35 (14.5) | 35 (10.4) | 23 (12.7) | 5 (20.0) |
| 25–60 m | 25 (10.3) | 24 (7.2) | 20 (11.0) | 12 (48.0) |
| **Patient Gender** | | | | |
| Male | 136 (56.2) | 203 (60.6) | 112 (61.9) | 12 (48.0) |
| Female | 106 (43.8) | 132 (39.4) | 69 (38.1) | 13 (52.0) |
| **Patient race/ethnicity** | | | | |
| Caucasian | 92 (37.3) | 110 (32.8) | 58 (32.2) | 9 (36.0) |
| Hispanic | 80 (34.4) | 163 (48.7) | 78 (42.8) | 10 (40.0) |
| African-American | 21 (8.0) | 16 (4.8) | 17 (8.9) | 0 (0.0) |
| Native American | 37 (15.9) | 25 (7.5) | 20 (11.1) | 5 (20.0) |
| Asian | 1 (0.4) | 2 (<1.0) | 2 (1.1) | 0 (0.0) |
| Other | 11 (4.0) | 19 (5.7) | 6 (3.3) | 1 (4.0) |
| **Insurance** | | | | |
| Public | 187 (77.6) | 237 (71.0) | 149 (82.8) | 21(64.0) |
| Private | 49 (20.3) | 88 (26.3) | 29 (16.1) | 2 (8.0) |
| None/unknown | 5 (2.1) | 9 (2.7) | 2 (1.1) | 2 (8.0) |
| **Caregiver marital status** | | | | |
| Married | 58 (24.0) | 91 (27.2) | 45 (24.9) | 5 (20.0) |
| Divorced | 3 (1.2) | 0 (0.0) | 1 (<1) | 0 (0.0) |
| Single | 57 (23.6) | 37 (11.0) | 32 (17.7) | 8 (32.0) |
| Live-in partner | 49 (20.2) | 24 (7.2) | 24 (13.3) | 7 (28.0) |
| Other/Unreported | 4 (1.7) | 1(<1) | 4 (2.2) | 0 (0.0) |
| Unknown | 71 (29.3) | 182 (54.3) | 75 (41.4) | 5 (20.0) |
| **Transfer** | | | | |
| Yes | 115(60.8) | 88 (55.3) | 63 (53.8) | 16 (64.0) |
| **Method of injury** | | | | |
| Unknown | 101 (41.7) | 47 (14.0) | 58 (32.0) | 8 (32.0) |
| Fall | 49 (20.2) | 115 (34.3) | 54 (29.8) | 11 (44.0) |
| Other | 92 (38.0) | 173 (51.6) | 69 (38.1) | 6 (24.0) |
| **Glasgow Coma Score** | | | | |
| Severe (<8) | 18 (7.4) | 4 (1.2) | 10 (5.5) | 24 (96.0) |
| Moderate (8–12) | 18 (7.4) | 3 (<1.0) | 6 (3.3) | 0 (0.0) |
| Minor (≥13) | 147 (60.7) | 270 (80.6) | 122 (67.4) | 0 (0.0) |
| Unknown | 59 (24.4) | 58 (17.3) | 43 (23.8) | 0 (0.0) |
| **Altered/loss of consciousness** | | | | |
| Yes | 47 (19.4) | 19 (5.7) | 23 (12.7) | 15 (60.0) |
| No | 112 2 (46.3) | 275 (82.1) | 134 (74.0) | 0 (0) |
| Unknown | 83 (34.3) | 41 (12.2) | 24 (13.3) | 10 (40.0) |
| **Bruising** | | | | |
| Yes | 74 (30.6) | 46 (13.7) | 36 (19.8) | 10 (40.0) |
| **Presenting symptoms*** | | | | |
| | N = 495 | N = 537 | N = 325 | N = 66 |
| Fever | 12 (2.4) | 10 (1.9) | 12 (3.7) | 2 (3.0) |
| Vomiting | 58 (11.7) | 60 (11.2) | 35 (10.8) | 6 (9.1) |
| Unresponsive/lethargy | 79 (16.0) | 50 (9.3) | 34 (10.5) | 22 (33.3) |
| Swelling to head | 81 (16.4) | 203 (37.8) | 101 (31.1) | 3 (4.5) |
| Difficulty breathing | 26 (5.3) | 6 (1.1) | 10 (3.1) | 12 (18.2) |
| Poor eating | 13 (2.6) | 9 (1.7) | 11 (3.4) | 2 (3.0) |
| Fussiness/crying | 68 (13.7) | 109 (20.3) | 56 (17.2) | 3 (4.5) |
| Not using extremity | 7 (1.4) | 3 (<1) | 1 (<1) | 0 (0) |
| Swelling to extremity | 5 (1.0) | 2 (<1) | 5 (1.5) | 0 (0) |
| Seizures | 54 (10.9) | 15 (2.8) | 18 (5.5) | 7 (10.6) |
| Skin concern | 56 (11.3) | 23 (4.3) | 19 (5.8) | 9 (13.6) |
| Dizziness/nausea | 0 (0) | 0 (0) | 2 (<1) | 0 (0) |
| Other | 32 (6.5) | 36 (6.7) | 19 (5.8) | 4 (6.1) |
| Unknown | 4 (<1) | 11 (2.1) | 2 (<1) | 0 (0.0) |
| **Head CT findings[a]** | N = 313 | N = 435 | N = 217 | N = 50 |
| Skull fracture | 104 (33.2) | 228 (52.4) | 103 (47.5) | 5 (10.0) |
| SDH | 107 (34.2) | 52 (12.0) | 38 (17.5) | 13 (26.0) |
| EDH | 11 (3.5) | 22 (5.1) | 15 (7.0) | 1 (<1.0) |
| EAH | 21 (6.7) | 24 (5.5) | 18 (8.3) | 4 (8.0) |
| SAH | 15 (4.8) | 20 (4.6) | 2 (<1.0) | 3 (6.0) |

**Table 2** *(continued)*

| Characteristic | Non-fatally injured patients N = 758 | | | Fatally injured patients N = 25 |
|---|---|---|---|---|
| | AHT | Accidental | Undetermined | All determinations |
| Other bleed | 10 (3.2) | 41 (9.4) | 7 (3.2) | 9 (18.0) |
| HIE | 2 (<1.0) | 0 (0) | 0 (0) | 4 (8.0) |
| Other brain injury | 43 (13.7) | 48 (11.0) | 34 (15.7) | 11 (20.0) |
| **Posterior rib fracture** | | | | |
| Yes | 19 (7.9) | 2 (<1.0) | 1 (<1.0) | 2 (8.0) |
| **Metaphyseal fracture** | | | | |
| Yes | 12 (5.0) | 6 (1.9) | 1 (1.3) | 2 (8.0) |
| **Multiple fracture** | | | | |
| Yes | 41 (16.9) | 12 (3.6) | 17 (9.4) | 5 (20.0) |
| **Length of stay** | | | | |
| Median [IQR] hours | 72 [48–206] | 24 [18–43] | 41 [23–66] | 25 [21.8–104.3] |
| 1–12 | 6 (2.5) | 35 (10.5) | 11 (6.1) | 1 (4.0) |
| 13–24 | 27 (11.2) | 166 (51.1) | 61 (33.7) | 11 (44.0) |
| 25–36 | 13 (5.4) | 34 (10.2) | 13 (7.2) | 2 (8.0) |
| 37–48 | 41 (16.9) | 49 (14.6) | 42 (23.2) | 2 (8.0) |
| 49–60 | 6 (2.5) | 1 (<1.0) | 7 (3.9) | 0 (0.0) |
| 61–72 | 24 (9.9) | 12 (3.6) | 20 (11.1) | 1 (4.0) |
| 73+ | 125 (51.7) | 28 (8.4) | 27 (14.9) | 8 (24.0) |
| **Number adults in home** | | | | |
| 1–2 | 102 (42.2) | 121 (36.1) | 78 (43.1) | 5 (8.0) |
| 3–7 | 19 (7.9) | 42 (12.5) | 22 (12.2) | 4 (4.0) |
| Unknown/unreported | 121 (50.0) | 172 (51.3) | 81 (44.8) | 16 (88.0) |
| **Alleged perpetrator[a]** | | | | |
| Mother | 34 (14.1) | 3 (<1.0) | 12 (6.6) | 3 (12.0) |
| Father | 61 (25.2) | 0 (0.0) | 7 (3.9) | 4 (16.0) |
| Significant Other | 29 (12.0) | 1 (<1.0) | 2 (1.1) | 7 (28.0) |
| Relative | 7 (2.9) | 1 (<1.0) | 2 (1.1) | 2 (8.0) |
| Caregiver (unrelated) | 12 (5.5) | 1 (<1.0) | 2 (1.1.) | 1 (4.0) |
| Other/unknown | 109 (45.0) | 4 (1.2) | 11 (6.1) | 8 (32.0) |
| Not applicable[b] | 0 (0.0) | 325 (97.0) | 145 (80.1) | 0 (0.0) |
| **Substance use** | | | | |
| Yes | 23 (9.5) | 7 (2.1) | 6 (3.3) | 2 (8.0) |
| **Mental illness** | | | | |
| Yes | 8 (3.3) | 15 (4.5) | 5 (2.8) | 2 (8.0) |
| **Intimate partner violence** | | | | |
| Yes | 47 (19.4) | 22 (6.6) | 24 (13.3) | 5 (10.0) |
| **Prior CPS report/contact** | | | | |
| Yes | 77 (31.8) | 45 (13.4) | 56 (30.9) | 10 (20.0) |
| **Prior police involvement** | | | | |
| Yes | 61 (25.2) | 28 (8.4) | 27 (14.9) | 8 (32.0) |

[a] Multiple findings/symptoms per patient; patients may have more than one perpetrator alleged.
[b] Not Applicable – "Alleged Perpetrator" is not applicable for non-inflicted injuries.

(p = 0.006). Fatally injured patients were not statistically different from non-fatally injured patients for transfer status (p = 0.49); posterior rib fracture (p = 0.43), metaphyseal fracture (p = 0.14), and multiple fractures in different body locations (p = 0.20). There were no differences in caregiver and household characteristics between fatal and non-fatal AHT patients: ethnicity (0.55); marital status (p = 0.49); prior CPS reports (0.12); prior police reports (p = 0.89); substance abuse (p = 0.88); intimate partner violence (IPV) (p = 0.91); or severe GCS = 4–7 (p = 0.33). Clinical differences noted included bruising rates (p = 0.005); severe GCS = 3 (p = 0.01); difficulty breathing as a presenting symptom (p = 0.001) and patient unresponsive/lethargy (p = 0.001).

### 3.3. AHT versus accidental head trauma

Table 2 compares patients by determination (AHT, accidental and undetermined). Of the 758 non-fatal injured patients, 242 (31.5%) were AHT; 335 (44.2%) accidental and 181 (23.8%) undetermined.

Eighty-one percent of non-fatal AHT patients were <12 months; 32% of fatal patients were <12 months (p = 0.001). Twenty-four percent of patients with AHT had married parents, a number comparable to patients where the injury was accidental (27.4%) (p = 0.59). Unreported parental marital status was highest among patients with accidental injuries (54.3%). Married parents represented only 20% of fatal injuries. There was a significant difference in transfer status for AHT patients compared to those with accidental injuries (p = 0.001).

Other clinical characteristics that differed between AHT and accidental injuries included: Glasgow Coma Score (p = 0.001); altered or loss of consciousness (p = 0.001); skull fracture (p = 0.001), SDH (p = 0.001); and other intracranial bleeding (p = 0.027). Symptoms that differed include: unresponsive/lethargy (p = 0.001); swelling to the head (p = 0.001); difficulty breathing (p = 0.001); fussiness/crying (p = 0.003); seizures (p = .001); and skin concern (p = 0.001). Among injury characteristics, there were differences for: posterior rib fracture (p = 0.001]), healing fractures (p = 0.001), metaphyseal fracture (p = 0.001), multiple

fractures in different body locations (p = 0.001), and bruising (p = 0.001). Almost 40% of patients with AHT did not report a method of injury (MOI), did not observe the injury or did not know the MOI, while only 14% of patients with accidental injuries had an unknown MOI (p = 0.001). Falls were the most frequently reported MOI for fatal AHT injuries (44.0%) while unknown/unobserved was the most frequently reported MOI for non-fatal AHT injuries (41.7); accidental injuries demonstrated more variety in the MOI as indicated by the proportion of 'other' (51.6%).

Family factors where differences between AHT and accidental injuries were noted included prior CPS contact (p = 0.001), prior police involvement (p = 0.001), substance use (p = 0.001), (IPV) (p = 0.001) and diagnosis/treatment for mental illness (p = 0.036).

### 3.4. Associated risks for AHT

Table 3 reports the adjusted odds ratios for clinical and family risks associated with AHT for children 5 years old or younger. The reference group is patients with accidental injuries; undetermined injuries were excluded. Of clinical risks, patients with multiple fractures were 10 times more likely to be adjudicated AHT (Exp($\beta$) = 10.34 [p = 0.001] ). Bruising of any kind increased the odds of AHT 5-fold (Exp$\beta$ = 4.77 [p = 0.001]); SDH increased the risk of AHT three-fold (Exp($\beta$) = 2.89 [p = 0.001]); loss of consiousness was associated with a four-fold increase in AHT risk (Exp($\beta$) = 4.69 [p = 0.001]). seizures were also associated with four–fold increase in risk(Exp($\beta$) = 3.95[p = 0.004]). Lethargy/unresponsiveness doubled the risk of AHT (Exp($\beta$) = 2.24 [p = 0.018]). Unknown MOI was also an associated risk (Exp($\beta$) = 3.944 [p = 0.001]). Other family risk factors associated with AHT were: substance abuse (Exp($\beta$) = 3.51 [p = 0.001]); unknown number of adults in the home (Exp($\beta$) = 4.35 [p = 0.001]), prior police involvement (Exp($\beta$) = 5.33 [p = 0.001]), and IPV (Exp($\beta$) = 2.89 [p = 0.02]). Patients with skull fractures were 50% less likely to be determined AHT (Exp($\beta$) = 0.55 [p = 0.052]).

We estimated the area under the ROC using probabilities from the model in Table 2. Area under curve (AUC) = 0.90 (95% CI = 0.86–0.93 [p = 0.001]) indicating the model was able to effectively discriminate between AHT and accidental injuries for 90% of the sample. Our model attained 73% sensitivity and 91% specificity. These results suggests the model will incorrectly screen in a patient as AHT 27% of the time while incorrectly screening a patient out as accidental only 9% of the time.

## 4. Discussion

Our research supports previous research on clinical indicators of AHT [20,25–31]. We also confirmed two family risk factors supported by pre-

**Table 3**
Adjusted odds ratios (AOR) for clinical and family risk factors associated with non-fatal AHT.

| Factor | AOR | 95% CI | p-Value |
|---|---|---|---|
| **Clinical Factors** | | | |
| Head fracture | 0.55 | 0.30–1.01 | 0.052 |
| Multiple fractures | 10.34 | 4.22–25.31 | 0.001 |
| LOC | 4.69 | 2.21–9.91 | 0.001 |
| Bruising | 4.77 | 2.56–8.86 | 0.001 |
| Seizures | 3.60 | 1.49–8.67 | 0.004 |
| SDH | 2.89 | 1.28–6.51 | 0.001 |
| Lethargy/unresponsiveness | 2.24 | 1.15–4.37 | 0.018 |
| MOI unknown | 3.94 | 2.07–7.46 | 0.001 |
| | | | |
| **Family Factors** | | | |
| Prior police reports | 5.33 | 2.61–10.90 | 0.001 |
| Unknown adult count | 4.35 | 2.38–7.97 | 0.001 |
| Substance abuse | 3.51 | 0.98–12.56 | 0.001 |
| IPV | 2.89 | 1.31–6.37 | 0.008 |

CI, confidence interval; MOI, method of injury; IPV, Intimate Partner Violence; SDH, subdural hematoma; CPS, Child Protective Services.

vious research on non-accidental trauma are also specific to AHT: substance abuse [32,33] and intimate partner violence [34–37]. In addition, we identified two family risks as significant in screening for AHT: prior police involvement and an unknown number of adults in the home.

The Glasgow Coma Score was not an associated risk for our study cohort. The GCS has been used as a clinical indicator of AHT but this indicator has been described as more reliable in severe head injury and/or older patients [38,39]. Mild AHT (GCS = 13–15) can be easily missed [18]. Seventy-one percent of our non-fatal patients had a GCS score ≥ 13 reflecting mild head trauma. Despite the absence of severity as measured by the GCS, our model attained a high degree of sensitivity and specificity suggesting its accuracy in early detection of AHT.

We found three presenting symptoms: lethargy/unresponsiveness, loss of consciousness, and seizures were associated with AHT. Seizures have been widely recognized as a clinical indication of AHT [27,40]. In this study, seizures elevated the risk of AHT three-fold. As expected, some non-specific presenting symptoms, e.g. fussiness, nausea and vomiting, were not associated risks for AHT but lethargy/unresponsiveness and LOC were moderate risks.

Similar to prior research, bruising was an associated risk of AHT in our study cohort [41]. We did not find sentinel injuries, e.g. posterior rib fractures, metaphyseal fractures, long bone fractures, were associated with AHT for our study cohort but these injuries may have not occurred with sufficient frequency to attain significance. Instead, we found the more general condition, i.e., multiple fractures, was associated with AHT. We also found isolated head fractures reduced the risk of AHT by 50%. Research suggests skull fractures are more likely associated with non-inflicted injury than AHT [14,20]. Our results provide further support for previous research that skull fractures are less likely to be associated risks for AHT [20,42].

Most SDH found in infants and toddlers is associated with a mechanism of child abuse [43]. In our study cohort, we also found SDH was an associated risk for AHT. However, Kemp et al. (2011) suggest SDH should be identified as a radiologic finding associated with AHT only when presenting with unexplained traumatic head injury, where no explanation has been provided or the explanation provided did not match the injury pattern [44]. In our study cohort, unknown MOI increased the risk of inflicted head trauma almost 4-fold. It is widely accepted that caregivers often provide no or inaccurate injury histories [18]. Misleading accounts of MOI can serve as a protective mechanism for caregivers against law enforcement and CPS involvement [14]. It must be noted that a prior CPS report was not an associated risk for AHT. Although research on general child maltreatment has supported the association between prior CPS reports and physical abuse [ 36], the relative young ages of patients in this study cohort may have limited the extent of prior reporting.

The absence of information has been associated with AHT [32]. Similarly, we also found that a lack of reporting may be a risk factor for AHT. Unknown number of adults in the home and an unknown MOI were associated risks for AHT. As many as 50% of families in our study cohort had missing data for the number of adults in the household or reported an unknown number of adults (Table 2). Substance use and overcrowding have been linked to disorganized households [45]. Our results provide additional support for research finding that children who live in homes with disorganized households and impermanent housing were more likely to be victims of fatal and non-fatal injury [45].

### 4.1. Fatal vs. non-fatal AHT

Overall, there were few significant differences in imaging, demographics, or clinical characteristics between non-fatally injured and fatally injured patients in our forensic registry, a result consistent with more recent research [46]. Fatally injured AHT patients demonstrated more bleeding on CT scans as previous research has found [25]. Bleeding has also been linked with low hematocrit levels and used in screens for AHT in previous research [47,48]. We also found low hematocrit was an

unadjusted associated risk factor for AHT ($Exp(\beta) = 3.1$ [$p = .001$]) but was not significant after adjusting for a CT finding of SDH. Evidence of bleeding in AHT patients also supports Ferguson's (2016) recommendation regarding more consistent intracranial pressure (ICP) monitoring as a standard of care for suspected AHT [46]. Fatally injured patients in our study cohort also presented with clinical conditions linked to AHT in previous clinical reviews, e.g., GCS < 8, bruising and altered or loss of consciousness [20,26,27,29].

### 4.2. Screening to improve surveillance of AHT

The lack of guidelines for clinical decision making in AHT has limited detection and surveillance. Only recently, have guidelines for detection of non-accidental trauma (NAT) been formally developed [49] but there are still many research gaps to bridge in order to insure comprehensive detection and surveillance. Clinical decision-making has also been hampered by provider biases [15], provider concerns over unnecessary radiation and the controversial nature of child abuse reporting based on screening factors [49]. In addition, screening for the purpose of both diagnosis and the development of forensic evidence to support the child protection process and criminal prosecution [42] has resulted in a reliance on clinical indicators and biomarkers needed for a confirmed diagnosis [18,20]. In the quest for greater sensitivity needed for diagnostic and forensic purposes, milder forms of AHT may continue to go undetected. In addition, current tools offer little guidance on patients *excluded* by the screen or where a screening determination cannot be made. As a result, AHT as a public health concern may remain systematically under surveilled until milder forms of AHT can be detected.

This research helps to fill a critical research gap in non-fatal AHT. We developed a screening tool to detect mild AHT in support of epidemiological surveillance. We acknowledge the screen may not be as useful to the development of forensic evidence. However, current screens [18,20,48] may provide strong evidence for prosecution but are more likely to miss milder forms of AHT. Our research adds a tool to detect AHT with a focus on the responsibility for reporting, not prosecuting. We also acknowledge the provider must balance the risks of child injury against reporting and investigation. However, the risk of missing mild AHT could be repeat injury, lifelong disability or death [17,42]. Research on injuries associated with supervisory neglect found 20% of patients had a previous head injury [14]. Our results suggest that obtaining additional family information could prevent a provider from screening out a child with mild AHT as accidental 91% of the time. Adding a few key family risk factors to the patient history or ED checklist (substance abuse, unknown or large number of adults in the home, prior history of police; IPV, unknown/unobserved MOI), could improve clinical decision-making substantially and initiate early interventions to prevent subsequent injury or death. Counterintuitively, stepping back from forensic evaluation but towards epidemiologic surveillance could potentially save more lives by identifying and reporting suspected AHT as early as possible. The purpose of this research was to develop a screen for epidemiological surveillance and identify milder forms of AHT as early as possible. However, more research is needed to improve surveillance of all forms of AHT and develop therapeutic interventions to address these preventable injuries early.

### 4.3. Limitations

The current study has limitations that affect the interpretation of the results. This was a single-institution study from a large, urban, regional pediatric trauma center, and the results may not generalize to other settings. Our referral process for determination did not rely on a single set of clear guidelines or a well-defined screening algorithm. As a result, children may have been under-surveilled. There could be a significant risk of type I error, namely by excluding children who should have been identified. CTs are somewhat limited in detecting older accumulations, petechial hemorrhages, and HIE. Labs were available for only 25%

of patients; IPV, prior police involvement, prior CPS reports, substance use and mental illness may have been underreported given the sensitive nature of this information despite a thorough background assessment performed by the CPT. Only concurrent risks were captured in this study; prior injuries could not be reliably collected without expanding collection beyond the single institution. Additionally, a third of patients who suffered serious head trauma were classified as undetermined because they did not meet the strict criterion for classification as AHT or accidental under our single institution's CPT determination scheme. These patients had characteristics fitting both AHT and accidental injuries suggesting that our institution's clinical pathway for child maltreatment during the study time frame was not specific enough to rule patients out. In addition, the rate of fatal AHT may be higher than reported because of classification bias.

## 5. Conclusions

AHT constitutes the overwhelming majority of fatal head injuries for children under the age of 5. To improve surveillance of AHT and detect milder forms of AHT, in addition to clinical evaluation, interviews should include caregiver substance abuse, intimate partner violence, prior police involvement and household size. An unknown number of adults in home is associated with AHT. Continued research is needed on AHT to improve detection in all medical settings. Early identification of family and clinical risk factors associated with AHT may help prevent further injury, disability and death in very young children.

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