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Traumatic epidural hematomas in the pediatric population: clinical characteristics and diagnostic pitfalls☆,☆☆

Camilla Cremonini, Meghan Lewis *, Monica Darlene Wong, Elizabeth R. Benjamin, Kenji Inaba, Demetrios Demetriades

Division of Trauma, Emergency Surgery and Surgical Critical Care, LAC+USC Medical Center, University of Southern California, 2051 Marengo Street, IPT, C5L100, Los Angeles, CA 90033, USA

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ABSTRACT

Background/Purpose: The purpose of this study was to review the initial clinical presentation of EDH, identify potential clinical markers and highlight diagnostic pitfalls.
Methods: Retrospective review of all pediatric patients admitted to a Level I Trauma Center diagnosed with blunt traumatic EDH from 2008 to 2018.
Results: A total of 699 pediatric patients were identified with blunt traumatic brain injury (TBI); 106 with EDH made up the study population. A skull fracture was present in 84%. Overall, the most common clinical finding was a scalp hematoma (86%), followed by loss of consciousness (66%), emesis (34%), headache (27%), amnesia (18%), and seizures (12%). Importantly, 40% of patients with EDH presented with GCS 15. Four children (4%) had GCS 15 and were completely asymptomatic on admission. In three children (3%) the only symptom was a scalp hematoma. 50% of all EDH required craniotomy, and this was not significantly different if GCS was 15 on presentation (45%, $p = 0.192$). Mortality was 2%. Fourteen patients (13%) were discharged with cognitive/motor deficits.
Conclusions: Pediatric EDH frequently present with subtle clinical signs, including a normal GCS half the time. Irrespective of asymptomatic presentation, threshold for CT scan or an observation period should be low after head injuries in children.
Type of study: Prognosis study.
Level of Evidence: Level II/III.

Traumatic brain injury (TBI) is one of the most common causes of childhood morbidity and mortality, and epidural hematomas (EDH) reportedly complicate approximately 3% of all head trauma in the pediatric population [1,2]. EDH in children are often the consequence of a minor head injury, which is the most common cause of cranial trauma in this age group [3].

The diagnosis and treatment of EDH in the pediatric population can be challenging, because the clinical presentation is frequently subtle and nonspecific [4,5]. Many pediatric patients with a developing EDH present neurologically intact and with normal vital signs [3,6]. Furthermore, the widespread availability of computed tomography (CT) scan at primary care hospitals has contributed to a greater proportion of EDH diagnoses in patients with a normal neurological examination or only minor symptoms [4,7,8]. The subtle presenting symptomatology of pediatric EDH makes the diagnosis a potential area for missed injury or delayed treatment.

While symptomatic patients have improved prognosis of this life-threatening condition when treated with prompt surgical evacuation, several recent studies have demonstrated that nonoperative management with serial neurological examination may be a safe alternative in asymptomatic patients [4,5,9–11].

Due to its rare prevalence, the majority of the studies that outline the clinical presentation of EDH in children are based on a small number of patients. The primary aim of this study was to review the initial presentation of pediatric EDH, identify potential clinical markers, and highlight diagnostic pitfalls. The secondary aim was to identify potential risk

Abbreviations: EDH, epidural hematomas; TBI, traumatic brain injury; AIS, Abbreviated Injury Scale; GCS, Glasgow Coma Scale; CT, computed tomography; SBP, Systolic blood pressure; HR, heart rate; MVC, motor-vehicle crash; ISS, Injury Severity Score; ICH, intracranial hemorrhage; SDH, subdural hemorrhage; SAH, subarachnoid hemorrhage; IPH, intraparenchymal hemorrhage; LOS, length of stay; ICU, intensive care unite; ICP, intracranial pressure; IQR, Interquartile range.

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* Corresponding author at: Division of Trauma & Critical Care University of Southern California, LAC+USC Medical Center, 2051 Marengo Street, IPT, C5L100, Los Angeles, CA 90033. Tel.: +1 323 409 7761; fax: +1 323 441 9909.
E-mail address: meghan.lewis@med.usc.edu (M. Lewis).

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factors associated with the need for operation in patients with Glasgow Coma Scale (GCS) score 15 on initial presentation.

1. Materials and methods

A retrospective observational study was performed of all pediatric patients (age < 18 years) admitted to a Level I Trauma Center between January 2008 and December 2018. Institutional Review Board approval was granted from the University of Southern California for this study. LAC + USC is a Level I Trauma Center for both adult and pediatric patients, staffed by fellowship trained trauma surgeons. Pediatric surgeons and pediatric intensivists are on call at all times and immediately available for consultation. Patients with blunt traumatic EDH were identified by ICD-9 and ICD-10 codes for TBI from the hospital trauma registry and hospital records. Patients were excluded if they were transferred to another facility within 48 h, as specific details relating to their outcomes were unavailable.

Patient demographics (age and gender), admission vital signs (systolic blood pressure [SBP], heart rate [HR] and GCS score on arrival), mechanism of injury (motor vehicle crash [MVC], automobile versus pedestrian [AVP], and fall), associated injuries, Abbreviated Injury Scale (AIS) for each body region, Injury Severity Score (ISS), radiographic investigations, and surgical procedures were reviewed. Patients were categorized into four groups according to GCS score on presentation: GCS = 15 (normal neurological examination, Group A), GCS 13–14 (minor TBI, Group B), GCS 9–12 (moderate TBI, Group C) and GCS less than 8 (severe TBI, Group D).

Admission and pre-hospital symptoms (loss of consciousness [LOC], seizures, amnesia, headache, and emesis), head CT scan report (EDH site and thickness, change in EDH size on serial scans, other associated intracranial hemorrhage (ICH), scalp fracture, scalp hematoma, midline shift), and neurological deficit at discharge (functional outcome) were abstracted from the charts. Associated intracranial hemorrhages included subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH) and intraparenchymal hemorrhage (IPH).

Study outcomes included mortality, hospital length of stay (LOS), intensive care unit (ICU) LOS, placement of an intracranial pressure (ICP) monitor, craniectomy, timing of craniectomy (divided into 3 groups: less than 6 h from presentation to the emergency room, between 6 h and 24 h from presentation, and after 24 h), and functional outcome at discharge.

Data collection was performed using a computerized spreadsheet (Microsoft Excel 2013; Microsoft Corporation; Redmond, WA) and analyzed using SPSS Statistics 23 (SPSS Inc., Chicago, IL). Descriptive statistics were calculated for all clinical variables described. Continuous variables are represented as median (interquartile range [IQR]). Categorical variables are presented as n (%). Univariate analysis was performed to identify differences between the four GCS groups, and then to compare patients observed with patients who underwent surgical evacuation, within Group A (GCS 15). The Kruskall-Wallis test was used to compare continuous variables. Pearson's chi-squared test or Fisher exact test were used to compare categorical variables. Multivariate analysis adjusting for factors with a p value <0.2 or with clinical relevance was used to identify possible risk factors for operation in the Group of patients with GCS 15.

2. Results

2.1. Patient demographics, injury pattern and mechanism of injury

Over the study period, 699 pediatric patients were identified with a diagnosis consistent with blunt TBI, 633 of whom underwent CT scan of the head. One hundred nine of these patients were diagnosed with EDH. Three patients were excluded because they were transferred within 48 h, resulting in a study population of 106 patients.

Overall, 42 (39.6%) of patients with EDH presented with a normal neurological examination (GCS 15). Twenty-seven patients (25.5%) had a GCS of 13–14 on presentation, 18 (17.0%) were admitted with GCS of 9–12, and 19 (18.0%) had a GCS ≤ 8.

The demographics and injury patterns for the four groups of patients are depicted in Table 1. There were no statistically significant differences between the groups. The overall mean age was 12 years (IQR 6–15). Seventy-five patients (71%) were male. The mean Injury Severity Score (ISS) was 14 (IQR 10–18). Forty-six patients (43.4%) had an ISS > 15. Head Abbreviated Injury Scale (AIS) was 3 in 71% (n = 75) of the total population. The majority of EDH patients (82%, n = 87) had "isolated" head injury (extracranial AIS <3).

Table 1 also displays the mechanisms of injury for the four groups of patients. The overall most common mechanism of injury was a fall (n = 48, 45%), and it occurred more frequently in the patients presenting with normal neurological examination (Group A), when compared to the other three groups (p = 0.038). In patients presenting with

Table 1
Demographics, clinical data, injury data and mechanism of injury.

	Total N = 106	GCS = 15 N = 42	GCS = 13–14 N = 27	GCS = 9–12 N = 18	GCS < 9 N = 19	p Value
Age	12 (6–15)	13 (7–14)	12 (6–14)	11 (4–17)	13 (6–14)	0.820
Gender						0.115
Male	75 (71%)	29 (69%)	22 (81%)	9 (50%)	15 (79%)	
Female	31 (29%)	13 (31%)	5 (19%)	9 (50%)	4 (21%)	
SBP on admission	127 (113–137)	124 (111–132)	127 (110–145)	127 (116–133)	133 (109–151)	0.601
HR on admission	96 (80–118)	93 (81–108)	100 (82–117)	104 (84–140)	93 (74–125)	0.560
Head AIS						0.341
3	75 (71%)	29 (69%)	21 (78%)	12 (67%)	13 (68%)	
4	28 (26%)	13 (31%)	5 (18%)	6 (33%)	4 (21%)	
5	3 (3%)	0	1 (4%)	0	2 (11%)	
ISS	14 (10–18)	10 (10–17)	14 (10–19)	17 (13–20)	14 (10–25)	0.595
Extracranial injuries						
Chest AIS ≥ 3	14 (13%)	4 (10%)	2 (7%)	2 (11%)	6 (32%)	0.112
Abdomen AIS ≥ 3	3 (3%)	1 (2%)	1 (4%)	0	1 (5%)	0.879
Extremity AIS ≥ 3	6 (6%)	1 (2%)	2 (7%)	1 (6%)	2 (11%)	0.498
Isolated TBI	87 (82%)	37 (88%)	23 (85%)	15 (83%)	12 (63%)	0.150
Mechanism						**0.038**
MVC	15 (14%)	3 (7%)	1 (4%)	5 (28%)	6 (32%)	
AVP	39 (37%)	13 (31%)	12 (44%)	6 (33%)	8 (42%)	
FALL	48 (45%)	23 (55%)	14 (52%)	6 (33%)	5 (26%)	
ASSAULT	4 (4%)	3 (7%)	0	1 (6%)	0	

SBP systolic blood pressure; HR heart rate; AIS abbreviated injury scale; ISS injury severity score; TBI traumatic brain injury: MVC motor-vehicle-crash; AVP auto-versus-pedestrian.

Table 2
Clinical presentation.*

Clinical presentation	Total N = 97	GCS = 15 N = 38	GCS = 13–14 N = 25	GCS = 9–12 N = 16	GCS < 9 N = 18	p Value
LOC	70 (66%)	20 (53%)	17 (68%)	14 (88%)	19 (100%)	<**0.001**
Seizures	13 (12%)	3 (8%)	3 (12%)	4 (25%)	3 (17%)	0.367
Emesis	36 (34%)	14 (37%)	9 (36%)	5 (31%)	8 (44%)	0.898
Absence of these symptoms	11 (10%)	6 (16%)	5 (20%)	0	0	0.066
Clinical presentation in patients older than 2 years**	Total N = 93	GCS = 15 N = 34	GCS = 13–14 N = 23	GCS = 9–12 N = 13	p Value	
Amnesia	19 (20%)	10 (29%)	8 (35%)	1 (8%)	0.210	
Headache	29 (31%)	21 (62%)	6 (26%)	2 (15%)	**0.003**	

LOC loss of consciousness.
Amnesia and headache were not recorded for patients with GCS <9.
* Missing information of 9 patients: 4 patients of groups A, 2 patients of group B, 2 patients of group C and 1 patient of group D.
** Missing information of 8 patients: 4 patients of groups A, 2 patients of group B, 2 patients of group C.

lower GCS (group C and D), motor vehicle accidents were a more common mechanism of injury.

2.2. Clinical presentation and head CT scan findings

Tables 2 and 3 portray the clinical and radiographic features of the study population. The most common clinical finding was a scalp hematoma (n = 91, 86%; Table 3), followed by loss of consciousness (LOC) (n = 70, 66%), emesis (n = 36, 34%), headache (n = 29, 27%), amnesia (n = 19, 18%), and seizures (n = 13, 12%). The prevalence of headache or amnesia may be underestimated, as these symptoms were not necessarily discernable in patients with decreased level of consciousness or in children age under 2 years. When compared to the other three groups of patients, children with normal neurological

Table 3
Head CT scan findings, procedures and outcomes.

	Total N = 106	GCS = 15 N = 42	GCS = 13–14 N = 27	GCS = 9–12 N = 18	GCS < 9 N = 19	*P value*
RADIOGHRAPHICS FINDINGS:						
Scalp hematoma	91 (86%)	34 (83%)	24 (89%)	15 (88%)	18 (100%)	0.331
Skull fracture	89 (84%)	31 (74%)	25 (93%)	16 (94%)	17 (94%)	0.069
EDH site:						0.556
Frontal	27 (26%)	13 (31%)	4 (15%)	5 (28%)	5 (26%)	
Temporal	46 (43%)	14 (33%)	16 (59%)	8 (44%)	8 (42%)	
Parietal	22 (21%)	9 (22%)	4 (15%)	5 (28%)	4 (21%)	
Occipital	11 (10%)	6 (14%)	3 (11%)	0	2 (11%)	
EDH thickness (cm)	1 (0.6–1.5)	1 (0.5–2)	0.8 (0.6–1)	1 (0.7–1.9)	1 (0.8–1.7)	0.414
EDH thickness ≥ 10 mm	55 (52%)	20 (48%)	12 (44%)	10 (56%)	13 (77%)	0.166
Midline shift	49 (46%)	18 (44%)	11 (41%)	11 (65%)	9 (50%)	0.428
Midline shift (mm)	4 (3–7)	4 (3–5)	3 (3–6)	5 (4–9)	7 (4–8)	0.052
Other associated intracranial hemorrhage						
SDH	29 (27%)	7 (17%)	6 (22%)	8 (44%)	8 (44%)	**0.045**
SAH	28 (26%)	4 (10%)	7 (26%)	7 (39%)	10 (56%)	**0.001**
IPH/HC	31 (29%)	6 (14%)	9 (33%)	8 (44%)	8 (44%)	**0.033**
none	54 (51%)	29 (69%)	13 (48%)	7 (39%)	5 (28%)	**0.015**
OUTCOMES:						
Mortality	2 (2%)	0	0	0	2 (10%)	0.058
Functional deficit	14 (13%)	1 (2%)	1 (4%)	5 (28%)	7 (37%)	<**0.001**
ICU stay (mean ± SD, days)	4 (3–6)	3 (2–4)	4 (3–6)	6 (4–10)	6 (4–16)	<**0.001**
Hospital Stay (mean ± SD, days)	6 (4–9)	4 (3–6)	5 (4–8)	8 (7–17)	9 (5–21)	<**0.001**
INTERVENTIONS						
ICP monitoring	17 (16%)	1 (2%)	3 (11%)	4 (22%)	9 (47%)	<**0.001**
Craniectomy/Craniotomy	54 (50%)	19 (45%)	11 (41%)	11 (61%)	13 (68%)	0.192
Surgical timing:*						**0.010**
• Craniotomy < 6 h.	38 (70%)	8 (44%)	11 (100%)	9 (90%)	10 (91%)	
• Craniotomy 6–24 h.	9 (17%)	7 (39%)	0	1 (10%)	1 (9%)	
• Craniotomy >24 h.	3 (6%)	3 (17%)	0	0	0	

EDH epidural hematoma, SDH subdural hematoma, SAH subarachnoid hematoma, IPH intraparenchymal hematoma, HC hemorrhagic contusion, ICU intensive care unit, ICP intracranial pressure.
* Missing data of timing for 4 patients who underwent a craniectomy: 1 patient Group A, 1 patient group C, 2 patients Group D.

Table 4
Patients with GCS 15 on admission: observed vs surgical intervention.

	Total N = 42	Observed N = 23 (55%)	Surgery N = 19 (45%)	*P* value
Age > 2 years	38 (90%)	19 (83%)	19 (100%)	0.114
LOC	20 (53%)	9 (41%)	11 (69%)	0.090
Amnesia	10 (29%)	5 (28%)	5 (31%)	1.000
Headache	21 (62%)	9 (50%)	12 (75%)	0.134
Seizures	3 (8%)	2 (9%)	1 (6%)	1.000
Emesis	14 (37%)	9 (41%)	5 (31%)	0.542
Absence of these symptoms	6 (16%)	5 (83%)	1 (6%)	0.370
Skull fracture	31 (74%)	18 (78%)	13 (68%)	0.504
Temporal location	14 (33%)	8 (35%)	6 (32%)	1.000
Thickness ≥ 10 mm	20 (48%)	3 (13%)	17 (90%)	< **0.001**
EDH thickness (mm)	10 (5–20)	5 (3–8)	20 (12–30)	< **0.001**
Midline shift yes/no	18 (44%)	3 (14%)	15 (79%)	< **0.001**
Midline shift (mm)	4 (3–5)	3 (2–3.5)	4 (3–5)	0.164
Scalp Hematoma	34 (83%)	19 (86%)	15 (79%)	0.685
Associated ICH	13 (31%)	7 (30%)	6 (32%)	0.936

LOC loss of consciousness; EDH epidural hematoma; ICH intracranial hemorrhage.

examination on admission (Group A) were more likely to present with headache ($p = 0.003$), whereas LOC was less common in this group of patients ($p < 0.001$). Amnesia, seizures and emesis were not significantly different between the GCS groups.

There were no statistically significant differences among the groups with regard to presence or absence of a skull fracture, EDH thickness, location of EDH, or presence or absence of midline shift (Table 3). A skull fracture was present in the majority of patients (N = 89, 84%) who had EDH. The overall mean EDH thickness was 10 mm (IQR 0.6–1.5), with 55 patients (52%) having a thickness ≥ 10 mm. Temporal was the most frequent site of EDH (n = 46, 43%), followed by frontal (n = 27, 26%), and parietal (n = 22, 21%). Occipital was the site of EDH in only 11 patients (10%). There were 49 patients (46%) with midline shift, of which the mean shift was 4 mm (IQR 3–7).

Approximately half of the study population (n = 54, 51%) had no other type of intracranial hemorrhage (ICH) present. Patients with normal neurological examination (Group A) were less likely to have other associated ICH (n = 29, 69%) when compared with the other GCS groups ($p = 0.015$). Conversely, associated intracranial hemorrhages such as subdural, subarachnoid, or intraparenchymal hematomas, were significantly more common in Group D (GCS < 9, $p = 0.045$, $p = 0.001$, $p = 0.033$ respectively).

2.3. Outcomes

There were several differences in outcomes among the four groups (Table 3). The overall mortality for pediatric patients with EDH was 2%. All mortalities were in Group D (admission GCS < 9), however, this increased mortality in comparison to the other groups did not reach statistical significance ($p = 0.058$). The mean ICU LOS was 4 days (IQR 3–6), while the mean HLOS was 6 days (4–9). Both of these outcomes were significantly lower in the group of patients who presented with normal neurological examination (Group A) ($p < 0.001$).

Table 5
Multivariate analysis for operation in patients with EDH and GCS 15, total N = 42.

	Adjusted *p*	OR	95% CI	
LOC	0.166	5.565	0.492	62.971
Headache	0.716	1.573	0.137	18.013
Skull fracture	0.624	0.465	0.022	9.939
Thickness ≥ 10 mm	**0.001**	**53.224**	4.739	597.752
Associated ICH	0.888	1.182	0.115	12.141

LOC loss of consciousness; ICH intracranial hemorrhage.
Binary logistic regression was performed with potentially causative variables in which p value was <0.2 in the univariate analysis or clinically relevant. Multicollinearity test was checked before doing multivariate analysis. AUROC: 0.913 (0.812–1.000).

There were also statistically significant differences among the four groups with regard to interventions performed in the hospital. Seventeen EDH patients (16%) underwent ICP monitoring; most of whom were in Group D (admission GCS < 9) (N = 9, 47%; $p < 0.001$). Half of all pediatric EDH patients required craniectomy (N = 54), however, this was not significantly different in the group with normal neurological examination on presentation (Group A) (N = 19, 45%; $p = 0.192$). There were statistically significant differences among the groups with regard to timing of craniectomy ($p = 0.010$). Most (78%) of the patients who underwent a delayed craniectomy (after 6 h) were in Group A (GCS 15 on presentation), whereas only 21% of the patients who underwent an immediate craniectomy (within the first 6 h) were in Group A (GCS 15 on presentation). Only three patients underwent a craniectomy after 24 h, and all had a normal neurological examination (Group A) on admission. The exact timing from first hospital admission to craniectomy was indeterminate in 4 patients who were transferred in from an outside facility, and these were therefore recorded as missing variables.

With regard to functional outcome at discharge, 12 patients were discharged with both a cognitive and motor deficit, and 2 patients were discharged with a minor cognitive deficit. The functional deficit was statistically associated with GCS on admission: seven of the children with deficit at discharge were in Group D (GCS < 9 on admission), five were in Group C (GCS 9–12), one was in Group B (GCS 13–14), and one was in Group A (GCS 15) ($p < 0.001$).

2.4. Patients with Normal neurological examination on presentation (group a)

Among the EDH patients with normal neurological examination on presentation to the emergency room (Group A) (N = 42, 40%), six of them (16%) also had none of the clinical symptoms classically associated with intracranial hemorrhage (LOC, seizures, amnesia, headache, or emesis). This subgroup of "asymptomatic" patients would not necessarily have required further radiographic study. However, two of these patients had a reported history of transiently altered mental status in the field. One was a 16-year-old patient who presented also with a scalp hematoma; the other was a 9-month-old infant who was described as lethargic by his mother. Both patients underwent CT scan that showed a less than 5 mm thickness epidural hematoma. They were both discharged after an uncomplicated observation period.

Three of the remaining "asymptomatic" patients in our population had only a scalp hematoma, prompting further evaluation with CT scan. Two of these "asymptomatic" patients were infants (age <2 years old), so lack of clinical symptoms was based solely on report of the parents. In both cases, the CT scan demonstrated an EDH of less than 5 mm in thickness. Another "asymptomatic" patient with a scalp hematoma was a 7-year-old child involved in a motor vehicle accident where there was a fatality, indicating a significantly high-risk mechanism. The CT scan of that child demonstrated a 2 mm thickness EDH. All three of these "asymptomatic" children with scalp hematomas were observed in the hospital with no changes in clinical status, and were subsequently discharged without complications.

The final "asymptomatic" patient had the most subtle presentation. He was a 5-year-old child involved in a motor vehicle accident who was ejected from the vehicle. He did not have a scalp hematoma; however, he did have several facial abrasions and a right maxillary canine avulsion. His first CT scan demonstrated a 5 mm thickness EDH. After 2 hours, the patient became increasingly lethargic, and an interval CT scan showed the EDH had increased in size (to a thickness of 20 mm). The patient was then taken to the operating room to evacuate the EDH, and he was ultimately discharged home with no deficits 5 days later.

Subgroup analysis was performed in the group of patients who presented with normal neurological examination (Group A) (Table 4).

Twenty-three patients (55%) were successfully observed without operative intervention. Of the 19 patients in Group A who required surgical evacuation, 10 of them (53%) had a thickness greater than 20 mm on the initial CT scan, whereas 8 of them (42%) demonstrated increased size of EDH on interval CT scan.

Comparing Group A patients who were observed with those who underwent an operation, EDH thickness and the presence of midline shift were positively associated with operative intervention ($p < 0.001$). Mean EDH thickness ranged from 3 to 8 mm (IQR) in the observation group, and from 12 to 30 mm (IQR) in the surgery group, and was an average of 15 mm greater in patients who underwent surgical evacuation ($p < 0.001$). Furthermore, multivariate logistic regression (Table 5) demonstrated that thickness ≥ 10 mm was independently associated with an increased risk of operative intervention (OR 53, adj $p = 0.001$). Other characteristics were not statistically different between the two groups.

3. Discussion

EDH is reported to occur in approximately 3% of pediatric head trauma [1,2]. Several previous studies have examined the predictive value of different clinical variables for positive head CT scan findings after TBI [12–14]. The challenge of deriving CT scan indications for patients with head trauma is to identify every clinically important intracranial injury, while minimizing radiation exposure and its long-term risks to pediatric populations, such as malignancies [15,16]. The Pediatric Emergency Care Applied Research Network (PECARN) group [13] validated prediction rules able to identify pediatric patients at very low risk of clinically important traumatic brain injury, for whom CT scan may be unnecessary. The authors derived two different models for prediction rules; for children younger than or older than 2 years of age, considering that younger patients have a different brain injury risk profile and are more sensitive to the effect of radiation from CT scan [13]. These models incorporate several variables related to the clinical presentation of TBI in children. At our institution, we use the PECARN models to guide imaging decisions in pediatric head trauma. As of yet, no single symptom or sign has been recognized as a reliable predictor of the severity of intracranial injuries [17,18].

Several studies have described the clinical presentation and the management of EDH in the pediatric population, however, due to its rare prevalence, all of these studies were small series [3–5,8–11]. Children sustaining traumatic EDH represent a heterogeneous group of patients with a variety of symptoms, and they have clinical presentations that are often subtle [3–5]. In addition, the widespread availability and use of CT scans has resulted in greater numbers of children with minor symptoms undergoing head CT scan, and consequently, more EDH being diagnosed in children who are neurologically intact [4,8]. These factors make the diagnosis and treatment of pediatric EDH uniquely challenging. In infants especially, likely owing to their inability to communicate, EDH constitutes a distinct and evasive clinical entity [5].

One of the first apparent clinical markers that may be associated with EDH in children is the mechanism of injury. Several studies have demonstrated falls from low height with focal impact to be the most common mechanism of injury causing EDH [3–5,8–10,19]. Maggi et al. [3] explained the mechanism by which relatively mild injuries can cause this type of intracranial hemorrhage in children, based on the anatomical constitution of the brain in these patients. Mechanisms of injury such as temporary deflection and rebound of the skull or skull fracture can cause a stripping of the dura mater from the inner table of the skull itself, which results in tearing of the dura-to-bone fibrovascular attachments [20]. The dura mater is strongly adherent to the bone in infants and in the elderly, causing the incidence of EDH to be rarer in these age groups [4,21]. Older children, instead, are more susceptible to developing EDH, because the fibrovascular attachments decrease as the skull grows during childhood [4]. The most common sources of bleeding in EDH are the middle meningeal artery or from fractured bone, whereas bleeding from the venous sinuses is relatively rare [3]. In our population, falls were not only the most common mechanism of injury ($n = 48$, 45%), but were also statistically associated with a normal neurological examination on admission (55% of patients in Group A, $p = 0.038$).

Regarding the clinical presentation of EDH on admission, the two most frequent symptoms identified in this study were loss of consciousness (LOC) and emesis (66% and 34% of the population respectively). This is in accordance with other literature series [3,9,22]. Maggi et al. [3], in a series of 61 cases of EDH managed surgically, described vomiting in about 70% of their population across age groups, while LOC was more common in older children. Though they are common, these symptoms are not specific for EDH, as they are also a known consequence of nonspecific head trauma. Guzel et al. [17] showed that LOC and scalp hematoma are risk factors predicting any brain injury. In their study, LOC was associated with an increased risk of abnormal CT scan in children above 2 years.

Several studies have demonstrated that local pathological findings upon physical examination of the head (e.g. scalp hematoma or a visible fracture) represent significant predictors of intracranial injuries, especially in younger children [17,23–25]. For example, Ciurea et al. [5] presented a 30-case series of infants with a diagnosis of EDH: they reported that cephalohematoma was a common clinical sign which occurred in 66.6% of their infant population. In our study population, 86% of the pediatric EDH patients had a scalp hematoma, and in three patients this was the only clinical finding warranting radiographic study. Another important clinical finding was the association with skull fractures, present in 84% of our EDH patients. Similarly, other studies in the literature have demonstrated high incidences of concomitant skull fracture, supporting this finding as a major risk factor for diagnosis of EDH [3,4,26–28].

Interestingly, about a half of our patients (51%) had no other associated intracranial hemorrhages (ICH). This was especially true in Group A (patients presenting with normal neurological examination). Associated ICH can have a significant impact on outcome [26], and can be associated with failure of nonoperative management in asymptomatic patients [29].

In our study, 45% of patients who presented with normal neurological examination (Group A) ultimately underwent a surgical evacuation, highlighting the difficulty in identifying those who may require operation at initial presentation. Overall, the frequency of surgical treatment for EDH was not significantly different between GCS groups ($p = 0.192$). The timing of the operation, however, was related to GCS group. Delayed craniectomy was more common in Group A ($p = 0.01$). This likely represents the tendency to manage neurologically normal patients with a nonoperative observation period and serial CT scan, as opposed to immediate evacuation of EDH seen on CT scan. Indeed, seven of the 10 patients who underwent delayed craniectomy (after 6 h) had demonstrated an increase in the EDH size on serial CT scan.

Some studies have demonstrated the temporal location of a traumatic EDH as a predictive factor for the need for surgical intervention [8,29,30]. Other studies, on the contrary, have not supported this finding, indicating that location alone should not be an absolute indication for surgery [9,20,31]. In our population, temporal site was the predominant location (46 patients, 43%), but it was not related to an increased risk for operation ($p = 1.000$).

Nonoperative management of acute traumatic EDH in neurologically intact patients is being increasingly recognized as a safe approach in the literature [4,5,8–11,22]. Flaherty et al. [9] suggested that normal mental status was most predictive of successful observation, but that EDH size parameters (such as EDH thickness and volume) were also significantly different between patients observed and operated on. Bejjani et al. [8], defining the radiographic characteristics of a series of 33 pediatric patients with EDH, demonstrated that thickness of more than 18 mm, midline shift greater than 4 mm, a moderate or severe mass effect, or temporal location were predictive of the need for surgical intervention. However, there are several documented cases of very large hematomas

in patients with normal mental status who were successfully observed non-operatively with good outcomes, indicating that the combination of neurological status and radiographic findings together should guide the need for surgical intervention [9,10]. For example, Champagne et al. [10], studying a series of 16 cases with voluminous EDH, demonstrated that non-operative management is a safe alternative in these patients [10]. In line with these authors, our study compared clinical features of the patients who were observed to the ones who underwent surgical evacuation, both of whom presented with normal neurological examination (Group A). EDH thickness ≥10 mm and the presence of midline shift were statistically significant differences between the two groups predictive of surgical intervention. On logistic regression, EDH thickness ≥10 mm represented an independent risk factor for operation in patients with blunt traumatic EDH and normal neurological examination on presentation. Nevertheless, three patients from Group A with EDH thickness ≥10 mm were successfully managed non-operatively, supporting the conclusion that size alone should not dictate the need for surgical intervention.

Our study has several limitations. It was a retrospective study, allowing for determination of associations, but not causality. Also, due to the retrospective nature, it is possible that patients could have had some clinical features on presentation that were not appropriately recorded in the medical chart, and were therefore missed on our analysis. In addition, since EDH occurs in a minority of pediatric TBI, our sample size is relatively small. We selected a 10-year period for evaluation to overcome this issue and achieve a sufficient sample size.

4. Conclusions

Pediatric acute traumatic EDH frequently presents with subtle clinical signs, including a normal GCS score almost half of the time. Physical examination findings of trauma to the scalp or skull may be even more important than classic symptoms or neurological examination. Irrespective of asymptomatic presentation, threshold for CT scan or an observation period should be low after head injuries in children. Many patients may be managed non-operatively, however, a high degree of suspicion is necessary to prevent missed injury and to allow for rapid surgical treatment, if it does become necessary. In asymptomatic patients, EDH thickness greater than 10 mm is a factor independently associated with the need for surgical evacuation.

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