

# Clinical Indicators of Traumatic Brain Injury and Skull Fracture in Pediatric Head Trauma Patients

*Pediyatrik kafa travmalı hastalarda, travmatik beyin yaralanması ve kafa kemiği kırıkları için klinik belirleyiciler*

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## SUMMARY

**Background:** To determine whether clinical signs and symptoms of brain injury are sensitive indicators of traumatic brain injury (TBI) and skull fracture (SF) in pediatric patients admitted with head trauma.

**Methods:** A retrospective cross-sectional analysis of all patients younger than 17 years of age admitted to the university-based pediatric Emergency Department (ED) with the complaint of head trauma during a 7-year period was conducted. All children who were admitted into pediatric emergency department had a TBI and/or SF were included in the study.

**Results:** 1167 patients with the chief complaint of head trauma were admitted to pediatric emergency department during the study period. Of these, 984 subjects were excluded because they had no acute radiographic abnormalities noted or had inadequate records of symptoms. The remaining 183 subjects constituted the study sample. Isolated TBI without SF was found in 40 (21.9%) patients, whereas isolated SF was found in 82 (44.8%) patients, and both TBI and SF were found in 61 (33.3%) patients. Forty five (44.6%) of the patients who had TBI were conscious, with GCS of 15 and had no focal neurological abnormalities. The most common mechanism of head injury was >5-foot fall. Findings that were not significantly associated with TBI were vomiting, post-traumatic seizure, lethargy, irritability, and scalp hematoma without loss of consciousness (LOC) ( $p=0.044$ ). There was a significantly higher incidence of scalp hematoma ( $p=0.015$ ) and LOC ( $p=0.052$ ) in children with SF.

**Conclusion:** Our results support that seizure, vomiting, lethargy, scalp hematoma, irritability and LOC are insensitive clinical predictors of TBI. LOC appear to be a risk predictor of TBI for patients of only those younger than 2 years old. Scalp hematoma is a potential risk factor for SF.

**Key words:** Computed tomography; head injuries; physical examination; skull fracture; traumatic brain injury.

## ÖZET

**Amaç:** Bu çalışmada, travmatik beyin yaralanması (TBY) ve kafa kemiği kırığı (KKK) tespit edilen pediyatrik hastalarda klinik semptom ve bulguların hassas belirleyiciler olup olmadığı değerlendirildi.

**Gereç ve Yöntem:** Retrospektif kesitsel çalışmamızın evrenini, pediyatrik acil servise 7 yıllık dönem içinde, kafa travması ile başvuran 17 yaş altındaki çocuklar oluşturdu. Bu hastalar içerisinde TBY veya KKK tespit edilenler çalışmamıza dahil edildi.

**Bulgular:** Çalışma süresince 1167 hasta acil servise kafa travması nedeniyle başvurdu. Bu hastaların 984'ü tomografilerinde radyolojik anormallik bulunmaması veya yeterli tıbbi kayda ulaşamaması nedeniyle çalışmadan dışlandı. Geri kalan 183 hasta çalışma grubunu oluşturdu. Kırk (%21.9) hastada KKK olmaksızın TBY, 61 (%33.3) hastada hem TBY hemde KKK tespit edilirken, 82 (%44.8) hastada izole KKK olduğu belirlendi. TBY olan hastalar içerisinde, 45 (%44.6) hastanın bilinç düzeyinin normal, GKS'nin 15 olduğu ve herhangi bir fokal nörolojik defisitinin olmadığı tespit edildi. En sık yaralanma mekanizmasının, 1.5 metre üzerindeki yükseklikten düşmeler olduğu izlendi. TBY'nin bilinç kaybı ( $p=0.044$ ) dışında hiçbir klinik parametre ile (kusma, travma sonrası nöbet, uykuya meyil, irritabilite skalp hematomu) arasında istatistiksel anlamlı ilişki olmadığı görüldü. KKK olan hastalarda ise skalp hematomu ( $p=0.015$ ) ve bilinç kaybı ( $p=0.052$ ) bulgularının görülmesi istatistiksel olarak anlamlıydı.

**Sonuç:** Elde edilen bulgular, TBY'de nöbet, kusma, letarji, skalp hematomu, irritabilite ve bilinç kaybının hassas belirleyiciler olmadığını destekler niteliktedir. Bilinç kaybı, sadece <2 yaş altı grupta, TBY için risk belirleyici olarak görülmektedir. Skalp hematomu, KKK için potansiyel risk faktörüdür.

**Anahtar sözcükler:** Bilgisayarlı tomografi; kafa travmaları; fizik muayene; kafa kemiği kırığı; travmatik beyin yaralanması.

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## Introduction

Despite substantial efforts to reduce its incidence, traumatic head injury continues to be a major problem in pediatrics.<sup>[1]</sup> Skull fractures (SF) and traumatic brain injury (TBI) are common in children admitted into the pediatric Emergency Department (ED) with head trauma.<sup>[2]</sup> Considerable controversy surrounds the appropriate evaluation of children with mild alterations in consciousness after closed head trauma and a Glasgow Coma Scale (GCS) score of 13-14.<sup>[3]</sup> Many studies have found conflicting evidence over the use of clinical indicators to predict intracranial injury in pediatric mild head injury.<sup>[4]</sup> Children with minor head injuries and those with SF / TBI did not differ significantly in incidence of behavioral change, loss of consciousness, emesis, or seizures.<sup>[4,5]</sup> Several authors recommend using clinical symptoms and signs as the screening tools for determining which patients needed radiographic imaging after following head trauma.<sup>[6]</sup> Conversely, many authors have suggested that abnormalities on neurological examination and clinical symptoms are not reliably present in children with TBI.<sup>[7]</sup> Head computerized tomography (CT) imaging is an essential tool in the management of TBI. It can provide valuable information regarding the type, location, and severity of TBI and SF. As a consequence of improved technology, which allows faster and more detailed imaging to be obtained without sedation, there has been an increase in CT use in recent years in the management of TBI. Unfortunately, increased CT use leads to increased radiation exposure as well as increased costs and therefore its utility needs to be carefully evaluated.<sup>[8]</sup> The objectives of this study were to determine whether clinical signs and symptoms are sensitive indicators of TBI and SF in pediatric patients admitted with head trauma, and to describe the clinical characteristics of symptoms and signs of brain injury.

## Methods

We reviewed medical records of previously healthy children aged 17 years and younger who had computer discharge diagnoses from the ED of head injury, SF, epidural hematoma, subdural hematoma, cerebral contusion, subarachnoid hemorrhage, intracerebral hemorrhage, pneumocephalus or cerebral edema and who were treated in the ED between January 1, 1995 and January 1, 2002. Patient history, physical findings, GCS score, specific injuries seen in the CT, discharge diagnoses, and outcome were obtained from the medical record. Clinical signs and

symptoms were selected as risk factors to be analyzed in pediatric head trauma patients at emergency department. These factors were: history of loss of consciousness (LOC), post-traumatic seizure, history of vomiting, focal neurological deficit (FND), scalp hematoma, lethargy, abnormal pupils and irritability. Children with a previous history of blood dyscrasias, prior intracranial lesion, ventricular shunt, penetrating trauma or birth trauma and no identified acute TBI or SF, were excluded. The medical records of each eligible patient were reviewed. A GCS score of 13 to 15 represented minor injury, 8 to 12 moderate injury, and less than 8 represented severe injury. Data analysis was performed with the SPSS 14.0 for Windows software package (SPSS, Chicago, Ill). The relationship between the occurrence of clinical findings and appearance of intracranial post-traumatic lesions on cranial CT was analyzed by stepwise logistic regression model and categorical data were analyzed by  $\chi^2$  and the Fisher exact test. Statistical significance was defined at p value of less than 0.05.

## Results

A total of 1167 subjects younger than 17 years of age were admitted to the university-based pediatric ED with the complaint of head trauma within a 7-year period. Of these, 890 subjects were excluded because they had no acute radiographic abnormalities noted. 277 (23%) of the patients with TBI and/or SF were initially included. Due to inadequate records of symptoms (i.e, GCS score, LOC, vomiting, abnormal pupils) another ninety four patients were excluded from the study. The remaining 183 subjects constituted the study sample. The mean age of the patients was  $74.53 \pm 51.41$  months, with a range of 4 to 204 months. Sixty-five subjects (35.5%) were girls and 32 children (17.5%) younger than 24 months. Isolated TBI without SF was found in 40 patients (21.9%), isolated SF was found in 82 patients (44.8%), and both TBI and SF were found in 61 patients (33.3%). Characteristics of patients with TBI were summarized in Table 1. Specific mechanisms of injury are outlined in Table 2. Hundred and twenty-five of the subjects (68.3%) had linear fractures, 17 (9.3%) had depressed fractures and 1 (0.5%) had basilar SF. Greater than 5 feet fall was associated with a higher incidence of SF and TBI, but this did not predict SF or TBI both  $<2$  years old ( $p=0.906$ ) and  $\geq 2$  years old group ( $p=0.340$ ). When the patients were stratified by GCS level into mild head trauma (MHT) (GCS score of

**Table 1.** *Type of intracranial lesions.\**

	n (%)
Epidural hematoma + SF	29 (28.7%)
Epidural hematoma + Cerebral edema	16 (15.8%)
Cerebral contusion + SF	10 (9.9%)
Isolated cerebral contusion	7 (6.9%)
Isolated subdural hematoma	4 (4%)
Isolated cerebral edema	4 (4%)
Subdural hematoma + SF	4 (4%)
Epidural hematoma + Cerebral contusion + SF	4 (4%)
Cerebral edema + SF	4 (4%)
Cerebral contusion + Subdural hematoma + SF	3 (3%)
Pneumocephalus + SF	3 (3%)
Isolated intracerebral hemorrhage	3 (3%)
Cerebral contusion + Subdural hematoma + Cerebral edema	2 (2%)
Subarachnoid hemorrhage + Cerebral contusion	2 (2%)
Subarachnoid hemorrhage + Cerebral edema + SF	2 (2%)
Isolated pneumocephalus	1 (1%)
Isolated subarachnoid hemorrhage	1 (1%)
Intracerebral hemorrhage + SF	1 (1%)
Epidural hematoma + Cerebral contusion	1 (1%)

\* Isolated TBI without SF PLUS both TBI and SF groups.

13 to 15), moderate head trauma (GCS score of 8-12), and severe head trauma (GCS score  $\leq 8$ ); diagnoses included 134 (73.2%) minor, 33 (18.0%) moderate and 16 (8.7%) severe head injury. There was a significant association between FND, abnormal pupils and TBI ( $p < 0.05$ ); however, 45 (44.6%) of children with TBI with or without SF had normal levels of consciousness and non-focal neuro-

**Table 2.** *Mechanisms of injury.*

	<2 years old n (%)	$\geq 2$ years old n (%)
Pedestrian injuries	4 (12.5%)	40 (26.5%)
Motor vehicle crash	2 (6.3%)	8 (5.3%)
Stair falls	2 (6.3%)	9 (6.0%)
$\geq 5$ feet fall	13 (40.6%)	49 (32.5%)
<5 feet fall	5 (15.6%)	11 (7.3%)
Child abuse	2 (6.3%)	5 (3.3%)
Bicycles	1 (3.1%)	15 (9.9%)
Other trauma	3 (9.4%)	14 (9.3%)

logic examinations. There is no clinical risk factor found as a predictor for TBI (Table 3). Local abnormalities on scalp examination (contusion or hematoma) were noted in 78.1% patients with SF. A finding significantly associated with SF was scalp hematoma ( $p$  value of .008). Logistic regression was performed to ascertain predictors of clinical factors within the study population. There was a significant association between FND, abnormal pupils and TBI ( $p < 0.05$ ). LOC, were the independent predictors of traumatic brain injury for patients of  $< 2$  years old. Findings that were not significantly associated with TBI were vomiting, post-traumatic seizure, lethargy, irritability, and scalp hematoma. The only clinical predictors associated with an increase of TBI were posttraumatic LOC (Table 4). There were no significant association between seizure, vomiting, lethargy, irritability and SF except LOC, scalp hematoma, FND and abnormal pupils shown by logistic regression analysis (Table 5). There was a significantly

**Table 3.** *Results of  $\chi^2$  and Fischer's exact test analysis relating clinical risk factors and traumatic brain injury.*

	<2 years old, n (%)		$\geq 2$ years old, n (%)	
	$\chi^2$ and Fischer's exact Test (p value)		$\chi^2$ and Fischer's exact Test (p value)	
Seizure	5 (15.6%)	1.000	6 (4%)	1.000
Vomiting	19 (59.4%)	0.823	79 (52.3%)	0.326
Lethargy	12 (37.5%)	0.718	42 (27.8%)	0.718
Irritability	3 (9.4%)	0.238	8 (5.3%)	0.730
FND	3 (9.4%)	0.238	15 (10%)	<b>0.002</b>
Abnormal pupils	2 (6.3%)	0.492	12 (7.9%)	<b>0.001</b>
LOC	14 (43.8%)	0.720	70 (46.4%)	0.740
Scalp heematoma	22 (68.8%)	0.712	99 (65.6%)	1.000

**Table 4.** Results of likelihood ratio analysis relating clinical risk factors to the presence of traumatic brain injury.

Probable Risk Factor	Likelihood Ratio for TBI (p)	OR (95% CI)
LOC	0.044*	0.214 (0.48-0.960)
Seizure	–	–
FND	0.007	16.59 (2.15-127.57)
Vomiting	–	–
Scalp hematoma	–	–
Lethargy	–	–
Abnormal pupils	0.025	–
Irritability	–	–

\* For only <2 years old group.

**Table 5.** Results of likelihood ratio analysis relating clinical risk factors to the presence of skull fracture.

Probable Risk Factor	Likelihood Ratio for SF (p)	OR (95% CI)
LOC	0.052	0.072 (0.005-1.020)
Seizure	–	–
FND	0.032	0.018 (0.000-0.711)
Vomiting	–	–
Scalp hematoma	0.015	25.80 (1.88-352.41)
Lethargy	–	–
Abnormal pupils	0.000	10.41 (2.96-36.53)
Irritability	–	–

higher incidence of scalp hematoma and LOC in children with SF ( $p < 0.05$ ).

## Discussion

Pediatric minor head trauma comprises a large portion of pediatric trauma cases seen in emergency departments. One to two percent of all pediatric patients seen in the ED present with minor head trauma, but only 3% to 5% of those patients have TBI, and less than 1% of these patients require any neurosurgical intervention.<sup>[6]</sup> In recent studies, the incidence of TBI found to be varying from 5%<sup>[9]</sup> to 25%<sup>[10]</sup> of neurologically normal children. A physician's goal in evaluating head trauma is to accurately identify and diagnose patients who are at risk for serious injury and complications. In patients with decreased level of consciousness or focal neurologic signs, the TBI is easier to diagnose than in fully conscious patients, in whom an

evolving TBI can be difficult to recognize.<sup>[7]</sup> Therefore, it is essential that the risk factors used to predict TBI are reliable. Many of the recommendations that physicians have followed are based on limited data and small study samples. Physicians must determine which patient is at risk for TBI and who would benefit from head CT. Unfortunately, no predetermined set of clinical criteria exists to accurately and reliably predict the presence of TBI. The assessment of the GCS, LOC, mechanism of injury, and neurologic disability can help guide the physician in the evaluation and management.<sup>[4]</sup> Consensus is lacking about the acute care of children with minor head trauma. Davis and colleagues suggest that a normal mental status (GCS 15) after an isolated head injury does not warrant a head CT and that the child is not at risk for TBI.<sup>[11]</sup> A similar suggestion is that only children with LOC, decreased GCS, or SF on plain radiography should undergo head CT.<sup>[12]</sup> On the other hand, other studies have shown that clinical factors are poor predictors of for pathologic CT findings in patients with minor head trauma.<sup>[13]</sup> Even children with apparently minor head trauma and normal mental status can suffer TBI. Pediatric patients can be difficult to assess clinically in terms of their mental status, behavior, and neurologic function.<sup>[10]</sup> Many authors have suggested that neurological examination abnormalities are not reliably presented in children with TBI.<sup>[7]</sup> In a prospective subgroup study, Teasdale et al. found that 16% of 99 children with TBI who required neurosurgery, had no SF or any history of loss of consciousness.<sup>[14]</sup> Quayle et al. reported that 16 (59.0%) of 27 pediatric case who had TBI had no focal neurological abnormalities or abnormal mental state; but one of these patients needed a surgical operation because of epidural hematoma.<sup>[6]</sup> It was noted in Lloyd's study the presence of neurological abnormalities had a sensitivity for identification of intracranial injury of 91% (21 of 23) and a negative predictive value of 97%.<sup>[7]</sup> There was also a trend toward an association between seizures and TBI, but it was not statistically significant. Nevertheless, over half of the children with TBI had a normal mental status and no focal abnormalities, two thirds of whom were infants.<sup>[11]</sup> In a study of head-injured children who required neurosurgical procedures, almost 70% had a GCS less than or equal to 8.<sup>[15]</sup> A normal Glasgow coma score does not preclude TBI because as many as 28% of patients with TBI are neurologically intact. Ultimately, significant injuries may still exist despite a normal neurologic examination, GCS 13 to 15, or no documented LOC.

<sup>[16]</sup> As seen in our study, in 45 (44.6%) of the patients who had TBI were conscious, GCS is 15 and no focal neurological abnormalities however LOC independent predictors of TBI for patients of younger than 2 years old.

Many studies have demonstrated that LOC is a significant indicator of TBI after blunt head injury.<sup>[9]</sup> Palchak and colleagues found the risk of TBI to be higher in pediatric patients with a history of LOC or amnesia.<sup>[17]</sup> Similar to the other published studies, the result of our study has showed that detection of a normal neurological examination, absence of both altered mental status and a history of loss of consciousness do not rule out a substantial percentage of cases with TBI. Clinical neurological abnormalities and GCS are not reliable predictors of TBI.

Vomiting after head injury is common in children and its significance is controversial. The majority of studies report no strong correlation between emesis and TBI, but some authors conclude that its presence does correlate with an increased risk of TBI.<sup>[6,18,19]</sup> In our study, of the 183 patients whose ED charts harbored notes on presence of vomiting, 51 (50.5%) of those with TBI with or without SF and 47 (44.8%) of those with isolated SF were noted to have vomited. Vomiting does not appear to be an independent risk factor for SF or intra cranial hematoma in the pediatric population. Overall, the literature suggests that although certain clinical signs such as LOC, abnormal mental status, or vomiting increase the likelihood of TBI, the predictive value of these symptoms is low because the majority of children with these symptoms have no TBI.<sup>[20]</sup> Despite their higher sensitivity for SF, skull radiographs have limited utility because they give little to no information about TBIs. CT is considered the standard diagnostic procedure of choice to search for an acute TBI, although sensitivity may be reduced for events in the posterior fossa.<sup>[2]</sup> In our study, isolated TBI was found in 40 patients (21.9%), in all TBI with out SF. Neither the absence of SF makes us predict absence of TBI nor the physical examination and skull radiography. In children, severe TBI can occur in the absence of SF. CT is therefore the imaging modality of choice and should be ordered with a low threshold if TBI is to be ruled out in children with mild head injury.

More recently, scalp hematomas have been shown to be markers of TBI in infants. The incidence of SF in children with minor head trauma ranges from 29% to almost 49%.<sup>[21]</sup> Many physicians use the presence of scalp ab-

normality to dictate ordering radiographic studies. Studies have identified abnormal scalp examinations and SF as important predictors of underlying TBI.<sup>[5]</sup> In our study 66 (%67.3) of the children with TBI and 54 (%65.9) with isolated SF had local findings of contusion or abrasion of the scalp. And there was a significantly higher incidence of scalp hematoma ( $p < 0.05$ ). In order to identify all pediatric patients with TBI and SF, any child with a marked scalp hematoma should be considered for CT.

Many studies of pediatric head injury cite falls as the most common mechanism of injury, ranging from 32% to 91%<sup>[7,16]</sup> and head trauma is the most frequent injury of fall-related injuries in childhood. Craniocerebral trauma is the most common injury in fatal falls.<sup>[22]</sup> Previous investigators have suggested that assessing the strength of the mechanism of injury is not a sensitive means of predicting which infants would turn out to have significant injuries after head trauma. Current data suggest that TBIs, like skull fractures, may also result from low-impact injuries.<sup>[23]</sup> In our series, five (15.6%) of the patients under the age of two were injured as a result of a fall from a height of less than 5 feet. This was a very common mechanism of falls in this group of age. This finding suggests that it should not be the power of the mechanism of injury that should prompt clinicians to search for TBI. The majority of fall-related head injuries in childhood mostly occurs in private houses. To decrease the frequency of the fall injuries, strategies should include parents' education about the mechanism of falls and increase prevention strategies.<sup>[24]</sup> Although falls may be the most commonly cited mechanism of injury in pediatric head trauma, motor vehicle accidents have been reported to have more significant intracranial pathologies.<sup>[23]</sup> The two most frequent mechanisms of injury were > 5-feet falls 49 (32.5%) and pedestrian injury 40 (26.5%) in  $\geq 2$  years old in our study. Therefore primary precautions for head injury must be taken according to each age group.<sup>[25]</sup>

Some limitations of our study must be noted. Because this was a retrospective study, all of data were not always available from the records. Since our study involved TBI/SF patient who were admitted, we may have missed some patients who were diagnosed with TBI and then discharged to home from the ED, since there is no uniform protocol in our ED to guide the management of children with head injuries. Another limitation of this study findings are generalization only previously healthy subjects.

## Conclusion

Our results support that seizure, vomiting, lethargy, scalp hematoma, irritability and LOC are insensitive clinical predictors of TBI. LOC appear to be a risk predictors of TBI for patients of only <2 years old group. Scalp hematoma is a potential risk factor for SF.

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