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ORIGINAL ARTICLE

Factors Affecting Mortality in Pediatric Severe Traumatic Cerebral Injury

Pediatrik Ağır Travmatik Beyin Hasarında Mortaliteyi Etkileyen Faktörler

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ABSTRACT

Aim: It is aimed to determine the factors affecting mortality in pediatric patients followed up with severe traumatic brain injury in the pediatric intensive care unit.
Material and method: All patients followed up in the Pediatric Intensive Care Unit between April 2019 and April 2021 due to severe traumatic brain injury were included. Demographic characteristics, pre-intensive care interventions and imaging findings, treatments applied in intensive care and intervention information of all patients were collected. Results were evaluated as survival rate, presence of tracheostomy requirement, brain death, and Pediatric Cerebral Performance Scale at discharge. The patients divided into two groups as survivors and non-survivors. All obtained data were compared between the two groups.
Results: During the study period, 47 patients with a diagnosis of severe traumatic brain injury were followed up. It was observed that the requirement of cardiopulmonary resuscitation, the need for inotrope-vasopressor and the need for erythrocyte transfusion were statistically significantly higher in the non-survivor group. (p value, respectively: 0.001, 0.001, 0.001) The survival rate in all patients in the study group was 70.2%. In non-survivor group most common pupil response at admission was fixed-dilated (71.4%). In non-survivor group 60% of the patients were lost in the first 24 hours of intensive care.
Conclusion: Mortality increases in patients who need resuscitation, erythrocyte transfusion and inotrope before intensive care. Patients who died showed pathologic pupillary response and low GCS. Severe TBH patients died mostly in the first 24 hours of admission.

Keywords: children, intensive care, severe head trauma

ÖZ

Amaç: Çocuk yoğun bakımda ağır travmatik beyin hasarı (TBH) ile izlenen pediatrik hastalarda mortaliteye etki eden faktörleri belirlemek hedeflemektedir.
Gereç ve yöntem: Çalışma geriye dönük, gözlemsel olarak planlanmıştır. Hastanemiz Çocuk Yoğun Bakım ünitesinde Nisan 2019-Nisan 2021 tarihleri arasında ağır travmatik beyin hasarı nedeniyle takip edilen tüm hastalar dahil edilmiştir. Çalışmaya alınan hastaların demografik özellikleri, yoğun bakım öncesi girişimler ve görüntülemeler, yoğun bakımda uygulanan tedaviler ve girişim bilgileri toplanmıştır. Sonuçlar yaşama oranı, trakeostomi varlığı, beyin ölümü ve taburculukta Pediatrik Serebral performans Skalası sonuçları olarak değerlendirilmiştir. Çalışmaya alınan hastalar yaşayanlar ve hayatını kaybedenler olarak iki gruba ayrıldı. Elde edilen tüm veriler iki grup arasında karşılaştırıldı.
Bulgular: Çalışma döneminde ağır travmatik beyin hasarı tanısı ile 47 hasta izlenmiştir. Hayatını kaybeden grupta kardiyopulmoner canlandırma uygulanması, inotrop-vazopressor gereksinimi ve eritosit transfüzyonu ihtiyacının istatistiksel olarak anlamlı oranda daha fazla uygulandığı görülmüştür. (p değeri sırasıyla: 0,001, 0,001, 0,001) Çalışma grubundaki tüm hastalarda yaşama oranı %70,2 olarak sonuçlanmıştır. Hayatını kaybeden hastaların yoğun bakım kabulde bakılan GKS tamamında 3 idi. Yoğun bakıma kabulde pupil yanıtı kaybedilen grupta büyük çoğunlukla fiks-dilate (%71,4) idi. Kaybedilen hastaların 60%'ı ilk 24 saatte kaybedilmiştir.
Sonuç: Yoğun bakım öncesinde canlandırma, eritrosit transfüzyonu ve inotrop ihtiyacı olan hastalarda mortalite artmaktadır. Hayatını kaybeden hastalarda patolojik pupiller yanıt ve düşük GKS olduğu görüldü. Ağır TBH olan hastalarda ölüm çoğunlukla kabülden sonraki ilk 24 saat içinde gerçekleşmiştir.

Anahtar Kelimeler: ağır kafa travması, çocuk, yoğun bakım

Introduction

Trauma is the most common cause of mortality and morbidity in children (1). In United States of America 9.2 million children admitted to pediatric emergencies because of trauma in a year; 475.000 children suffer from traumatic brain injury (TBI) (1,2). Mild head trauma is the commonest head trauma in children; moderate and severe head trauma spotted less than 10% of trauma patients (3). Mechanism of trauma varies in different ages. Fall from height is common in 0-4 age group while it was seen less in 5-9 age group. Sports and vehicle collisions start increasing from 10 years old. Head trauma due to motor vehicle collisions peaks at adolescent age group (2).

Most childhood traumatic brain injury is caused by preventable causes such as falls, vehicle accidents and sports injuries (4). Depending upon the preventive measures, trauma related mortality decreased by 29% years between 2000-2009, according to data obtained from United States National Center for Injury Prevention and Control (NCIPC) (2). Although preventive measures are important, treatment applied to trauma patients are also vital. Enhancing clinical information about this important cause of mortality and morbidity will increase the quality of interventions applied to patients.

It is hard to predict the consequences of traumatic head injury (2). A child with a mild TBI may have learning difficulties or behavioral problems while a child with a severe head trauma may be dependent on their families or a device for the rest of their life (2).

This study aims to determine the factors affecting mortality of pediatric severe head trauma patients.

Materials and Method

Our study was conducted as observatory and retrospective. All patients included into the study in time period between April 2019-April 2021 with severe head trauma diagnosis followed up in third-level Pediatric Intensive Care Unit. Severe head trauma was defined as Glasgow Coma Scale Score below 9 by emergency doctor/neurosurgeon or pediatric intensivist. All pediatric trauma patients with GCS below 9 were included in the study. Moderate head trauma patients (GCS 13-15), mild head trauma patients (GCS 9-12) and patients with missing data were excluded. Patients' data was obtained from patient's files and computer registries.

Patients' demographic data was defined as age (in month), comorbid disease presence and patients body weight as kg. Mechanism of trauma (motor vehicle collisions, fall from height and other mechanisms), Pediatric Risk of Mortality Score (PRİSM-III), Injury severity score (ISS), pediatric trauma score (PTS), interventions before PICU admission (red blood cell transfusion, intubation, inotrope requirement, cardiopulmonary resuscitation) and patients cranial imaging findings (Positive findings in computerized tomography and magnetic resonance imaging) were defined as pre-PICU findings.

Glasgow Coma Score (GCS) at admission, pupillary response, laboratory parameters (blood gas parameters, complete blood count, serum glucose level, liver function tests, renal functions tests), respiratory support therapies (high flow nasal cannula therapy, non-invasive ventilation, mechanic ventilation), inotrope requirement, antiedema treatments which applied to patients (hypertonic saline, mannitol, steroid), antiepileptic requirement, convulsion presence in PICU and neurosurgery requirement data was collected.

Outcomes were examined as tracheostomy requirement, days in PICU, days in hospital and Pediatric Cerebral Performance Category level at discharge. We also determined patients with brain death during PICU process.

Patients were divided into two groups as Survivors and Non-survivors. All data was compared between two groups.

Our study was approved by our hospital's Ethic Committee Number 1 (Date:24.11. 2021.Decision Number: E2-21-1060). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Descriptive analysis of the results was conducted by using the SPSS 17.0 software package for Windows (IBM Company, New York, NY). Categorical data expressed as proportions (%). Median and inter quartile range were used for quantitative data. Differences were evaluated by Chi-Squared test in cases of categorical variables; non parametric test (Mann-Whitney U) for continuous variables. Data was considered statistically significant at p-value <0.05.

Results

During the study period forty-seven patients included into the study. Patients' demographic data is shown in Table-1. Adolescent age group suffered more from TBI. Male gender more prone to TBI. tended toward to TBI. Demographic data did not differ between survivors and non-survivors.

Table-1, Demographic data of pediatric severe head trauma patients, (n=47)

Age (month), median (IQR)	84 (29.0-156.0)
Age groups, n (%)	
Infant	10 (21.3)
Pre-school	10 (21.3)
School age	10 (21.3)
Adolescent	17 (36.7)
Gender, n (%)	
Male	33 (70.2)
Female	14 (29.8)
Weight (kg), median (IQR)	25 (15.0-50.0)
Co-morbid disease presence, n (%)	3 (6.4)

IQR: Inter quartile range.

Pre-pediatric intensive care interventions and cranial imaging findings are demonstrated on Table-2. There was no difference between groups in trauma mechanisms but fall from height was more common in infant and preschool children and motor vehicle collisions was more common in adolescent age group. When we evaluated trauma scores of patients, ISS was statistically high in non-survivors and PTS was statistically low in non-survivors (p= 0.001, 0.001 respectively). In non-survivor group cardiopulmonary resuscitation,

inotrope-vasopressor requirement and RBC transfusion requirement was applied much more to patients (p= 0.001, 0.001, 0.001 respectively). Patients whose computerized tomography findings with brain edema and subarachnoid hemorrhage had more mortality than other patients (p=0.005, 0.008). Diffuse axonal injury was seen in 34% of survivors.

Table-2, Parameters and interventions done before pediatric intensive care admission

	Total (n=47)	Survivors (n=33)	Non-Survivors (n=14)	P value
Trauma mechanism, n (%)				0.828
Vehicle accidents	21 (44.7)	14 (42.4)	5 (35.7)	
Fall from height	19 (40.4)	14 (42.4)	7 (50.0)	
Drowning	3 (6.4)	2 (6.1)	1 (7.1)	
Abuse	2 (4.3)	1 (3.0)	1 (7.1)	
Crash (Television)	2 (4.3)	2 (6.1)	0 (0)	
Injury severity score, median (IQR)	39 (28.0-48.0)	34 (28.0-43.0)	54.50 (41.75-59.25)	0.001
Pediatric Trauma Score, median (IQR)	5 (2.0-6.0)	5 (3.5-7.0)	2 (-0.25-3.50)	0.001
Interventions, n (%)				
Intubation	47 (100.0)	33 (100.0)	14 (100.0)	0.546
RBC transfusion	12 (25.5)	3 (9.1)	9 (64.2)	0.001
CPR	12 (25.5)	4 (12.1)	8 (57.1)	0.001
Vasopressor treatment	11 (23.4)	1 (3.0)	8 (57.1)	0.001
CT findings, n (%)				
Fracture of base of scalp	34 (72.3)	25 (75.7)	12 (85.7)	0.125
Concussion	26 (55.3)	16 (48.4)	10 (71.4)	0.128
Cerebral edema	19 (40.4)	9 (27.2)	10 (71.4)	0.005
Epidural bleeding	5 (10.6)	5 (15.1)	0 (0)	0.123
Subdural hemorrhage	21 (44.7)	15 (45.4)	6 (42.8)	0.870
Subarachnoid hemorrhage	23 (48.9)	12 (36.3)	11 (78.5)	0.008
Brain parenchymal hemorrhage	6 (12.8)	5 (15.1)	1 (7.1)	0.452

CPR: Cardio pulmonary resuscitation; CT: Computerized Tomography; IQR: Inter quartile range; RBC: Red Blood Cell

Pediatric intensive care interventions, laboratory parameters at admission and intensive care interventions are shown in Table-3. Pediatric Risk of Mortality Score was higher in non-survivor group (p=0.001). Inotrope requirement and maximum VIS was significantly high in non-survivor group (p=0.001). Pathologic pupillary response at admission was seen in high rate in non-survivor group (p=0.001). Post-traumatic convulsion (PTC) was present in 19.1% of patients. In cranial imaging findings, cerebral infarct and subdural hemorrhage were related with PTC (p=0014, 0.003). Days in mechanical ventilation was statistically longer in survivor group (p=0.024). If we evaluated laboratory parameters at admission into PICU, venous blood gas pH, base excess, thrombocyte count was statistically at lower level I non-survivor group (p= 0.001, 0.001, 0.001 respectively). Venous lactate level, partial carbon monoxide pressure in venous blood gas (pCO2), serum sodium, aspartate aminotransferase (AST), blood urea nitrogen, creatinine, international normalization ratio (INR), prothrombin time (PT), partial thromboplastin time (PTT) levels were significantly high in non-survivor group (p= 0.001, 0.011, 0.004, 0.044, 0.003, 0.001, 0.002, 0.002, 0.002 respectively).

Outcomes was shown in Table-4. Days in PICU and days in hospital were significantly long in survivors' group (p= 0.001, 0.001 respectively). Survival rate of patients was 70.2%. Tracheostomy was applied to eight of survivors. Brain death was defined to three patients. None of patients accepted organ donation. If we evaluated Pediatric Cerebral Performance Category results, most of the patients were in Categori-1 (45.1%). 8 patients were defined in Categori-5 (24.2%).

Most of the non-survivors on adolescent age group. (50%) Male gender was prominent in non-survivor group. (70%) Glasgow Coma Score of non-survivors was three in all patients. Pupil response was fix-dilated in 71.4% of non-survivors. 8 non-survivor patients were lost in first twenty-four hour of admission. Mechanical ventilatory days, days in PICU and days in hospital were shorter than survivors in non-survivor group.

Table-4, Outcomes

	Total (n=47)	Survivors (n=33)	Non-survivors (n=14)	P value
Days in PICU, median (IQR)	7 (3-19)	9 (5.5-37.0)	1 (1.0-5.5)	0.001
Days in hospital, median (IQR)	15 (5.0-55.0)	26 (13.0-88.0)	1(1-5.5)	0.001
Survival, n (%)	33 (70.2)			
Tracheostomy applied, n (%) (n=33)	8 (24.2)			
Brain death reported	3 (6.8)			
PSPC in survivors, n (%) (n=33)				
1	15 (45.4)			
2	7 (21.2)			
3	1 (3.0)			
4	2 (6.0)			
5	8 (24.2)			
6	0 (0)			

IQR: Inter quartile range; PICU: pediatric intensive care unit; PSPC: Pediatric Cerebral Performance Category

Table-3, Laboratory parameters at pediatric intensive care admission, treatment and interventions applied at pediatric intensive care unit

	Total (n=47)	Survivors (n=33)	Non-survivors (n=14)	P value
PRİSM-III scores	6 (6-13)	6 (6.0-6.0)	15 (13.0-24.5)	0.001
GCS	3 (3-5)	5 (3.0-7.0)	3 (3.0-3.0)	0.001
Pupil response, n (%)				0.001
Isochoric	31 (65.9)	28 (84.8)	3 (21.4)	
Fix-dilated	13 (27.6)	3 (9.0)	10 (71.4)	
Anisocoric	2 (4.2)	1 (3.0)	1 (7.1)	
Mid-dilate	1 (2.1)	1 (3.0)	0 (0)	
Inotrope treatment required, n (%)	23 (48.9)	9 (27.2)	14 (100.0)	0.001
Maximum VİS, median (IQR) (n=22)	145 (20-220)	15 (10.0-22.5)	220 (156.25-220.0)	0.001
Antiedema treatments, n (%)				0.232
Hypertonic saline	41 (87.2)	27(81.8)	14 (100.0)	
Hypertonic saline+ Steroid	5 (10.6)	5 (15.1)	0 (0)	
Hypertonic saline+ Steroid+ Mannitol	1 (2.1)	1 (3.0)	0 (0)	
Clinic convulsion, n (%)	9 (19.1)	9 (27.2)	0 (0)	0.03
Antiepileptic drugs requirement, n (%)				0.131
Levetiracetam	27 (57.4)	17 (51.5)	10 (71.4)	
Phenytoin	14 (29.7)	11 (33.3)	3 (21.4)	
Levetiracetam+ Phenytoin	5 (10.6)	5 (15.1)	0 (0)	
Patients followed up without antiepileptic treatment	1 (2.1)	0 (0)	1 (7.1)	
Neurosurgery applied, n (%)	8 (17.0)	5 (15.1)	3 (21.4)	0.601
Days in mechanical ventilation, median (IQR)	3 (1-12)	5 (2-18)	1 (1-1)	0.024
Non-invasive respiratory support applied, n (%)	26 (55.3)	26 (78.8)	0 (0)	0.601
Days in non-invasive respiratory support, median (IQR) (n=26)		1 (1-4)	0 (0)	
pH, median (IQR)	7.25 (7.08-7.38)	7.33 (7.11-7.41)	7.07 (6.90-7.16)	0.001
pCO2, mmHg, median (IQR)	42.6 (33.8-52.6)	40.8 (29.1-49.8)	52.8 (36.8-60.9)	0.011
BE, median (IQR)	-8.6 (-13.0- -4.0)	-5.1 (-10.15- -2.10)	-15.9 (-22.10- -10.17)	0.001
Lactate, mmol/L, median (IQR)	5.04 (2.06-7.60)	2.69(1.89-5.89)	7.53 (6.30-12.03)	0.001
White blood cell, x10^9/L, median (IQR)	17000 (13.770-24040)	16970 (13185-22455)	21610 (13195-25832)	0.409
Hemoglobulin, g/dl, median (IQR)	11.0 (10.2-13.4)	11.50 (10.55-13.20)	10.25 (7.90-13.50)	0.146
Thrombocyte, x10^9/L, median (IQR)	264 (201-327)	295 (243-366)	202 (137-251)	0.001
Serum blood glucose, mg/dl, median (IQR)	173 (126.0-253.0)	160 (108.0-211.5)	212.5 (148.5-293.5)	0.061
Sodium, mEq/L, median (IQR)	141 (138-143)	140 (138.0-143.0)	144 (140.5-148.2)	0.004
AST, U/L, median (ÇAA)	121 (48-228)	74 (44.5-228.5)	180 (94.75-353.75)	0.044
ALT, U/L, median (ÇAA)	60 (31-159)	47 (28.0-139.0)	105 (43.50-262.25)	0.079
Urea, mg/dl, median (IQR)	32 (26.0-39.0)	29 (25.0-36.0)	43 (34.7-53.0)	0.003
Creatinine, mg/dl median (IQR)	0.53 (0.39-0.96)	0.43 (0.34-0.57)	1.10 (0.89-1.57)	0.001
INR, median (IQR)	1.40 (1.20-1.60)	1.30 (1.15-1.40)	1.90 (1.40-2.12)	0.002
PT, second, median (IQR)	16.2 (13.4-18.3)	14.80 (13.10-16.75)	21.25 (16.42-24.12)	0.002
PTT, second, median (IQR)	27.5 (25.0-38.0)	26.30 (24.35-29.35)	40.20 (31.17-51.17)	0.002

ALT: Alanine Amino Transferase; AST: Aspartate Amino Transferase; BE: Base Excess; IQR: Interquartile range; GCS: Glasgow Coma Score; INR: International Normalization Ratio; PRİSM: Pediatric Risk of Mortality; PT: Prothrombin Time; PTT: Partial Prothrombin Time; VİS: Vasoactive Inotrope Score

Discussion

Due to the decrease in the main causes of mortality such as infectious and congenital causes with the development of modern medicine, trauma causes such as motor vehicle collisions and falls have emerged as the main cause of death. Traumatic brain injury not only causes mortality, but also leads to morbidities such as long-term cognitive disorders and personality changes (5).

Traumatic head injury was seen more common in adolescent male gender group in multi-centered studies (1,5). Our study also shows common male adolescent population in traumatic brain injury patients and supported these results. According to data obtained from United States of America NIPACC in infant population abuse was the commonest cause of trauma while falls are common in pre-school children (2). National Injury Prevention and Control Center has also determined that motor vehicle collisions are the most common trauma mechanism in school-aged and adolescent age group (2). Fall from height was the most common trauma mechanism in infant and pre-school aged children; motor vehicle collisions are major trauma cause in school aged and adolescents in our study as in NIPACC data. Although our findings were similar to the literature, there should be more multicentered studies to show Turkish populations trauma data.

Injury severity score has been used for detecting trauma severity in trauma patients since 1974 (6). Injury severity score above fifteen is defined as major trauma in adult patients. In pediatric patients ISS above twenty-five was associated with increased mortality (7). In our study population median value of ISS score was higher in non-survivor group. This situation can be interpreted as more severe trauma was seen in non-survivors and accordingly, and vital interventions such as transfusion, cardiopulmonary resuscitation and inotrope requirement before PICU admission were needed in non-survivor group. Like our data, Aoki et al. (8) reported that severe injured pediatric trauma patients had higher mortality than adults and cardiopulmonary resuscitation before or at hospital admission was associated with high mortality.

Most common findings in pediatric TBI patients at cranial brain computerized tomography imaging's were: diffuse axonal injury, edema result of ventricle compression, midline shift, subdural hemorrhage and intraparenchymal hemorrhage (9). In our population study, most common radiological findings on brain CT were: fractures in scalp, contusion, subarachnoid hemorrhage, subdural hemorrhage and brain edema respectively. Diffuse axonal injury (DAH) was demonstrated on brain MRI in 34% of survivors but most of the non-survivors did not have a cranial MRI due to short period of PICU hospitalization and clinical instability. So, we could not emphasize about diffuse axonal injury in our non-survivor patients. In the study of Hochstadner (9) et al. about TBI patients with subarachnoid hemorrhage, mortality did not differ with presence of subarachnoid hemorrhage. In a study from Korea with 256 pediatric TBI cases, SAH, DAH and subdural hemorrhage were related with mortality (5). In our study group, we spotted more SAH than in the literature data. Even though subarachnoid hemorrhage and brain edema were associated with mortality in our small population, more prospective multicentered studies should be carried out with larger patient groups.

Literature shows that low GCS, no pupil response, hypotension and ischemia spotted at first cranial imaging are related with mortality (9). Low GCS and pathological pupillary response were related with mortality in our study group. These findings were a common situation in severe head trauma patients. Depending upon the trauma severity, high inotrope-vasopressor requirement was at higher doses in non-survivor group. Early loss of patients resulted to short PICU days and short days in mechanical ventilation.

Traumatic brain injury can cause cerebral edema, elevated intracranial pressure (ICP) and distorted cerebral perfusion (10). Hyperosmolar treatments are effective treatment modality to decrease ICP (10). Hypertonic saline (%3) is the main antiedema treatment method in TBI (11). Hypertonic saline was the commonest method in our severe TBI patients to decrease ICP. There is no correlation between hypertonic saline application and mortality in our study. Multicenter studies should be conducted with large patients' groups to determine which method is more effective and decrease mortality.

Post-traumatic convulsions (PTC) could cause secondary brain damage (12). Post-traumatic convulsion was related with bad prognosis in TBI patients (13). Clinic and sub clinic PTC were seen in moderate and severe TBI in 11-42%of patients (13). Bennett et al. (12) found that PTC rate was 25.2% in 2122 pediatric trauma patients. The presence of post-traumatic convulsion is seen in high rate at small age patients,patients with abuse as a trauma mechanism, and subdural hemorrhage. We did not determine PTC difference between age groups and trauma mechanism in our population. Subdural hemorrhage and infarct at cranial MRI were related with clinical convulsion.

Mortality from TBI differs between countries. Ten-year retrospective, single-centered study from Norway stated that mortality from TBI was 1.2/100000 (3). Mortality reported from USA was 5.7/100000 in 2017 (14). There is no mortality incidence data of pediatric TBI patients from Turkiye unfortunately. Mortality rate of pediatric TBI in Korea was 1.5% in all trauma patients and 26.7%in severe head trauma patients (5). A single-centered paper from Turkiye reported pediatric TBI mortality rate as 26.5% (15). Mortality rate of our study was 29.8%. However, there is no data about ISS of patients with mortality in studies. Ongun et al. (15) reported that median value of ISS score of severe

TBI patients was 15.5 while median value of ISS of our patients was 39. Our higher mortality rate could be explained with high median ISS of our patients.

Prevalence of brain death in pediatric intensive care patients was found as 15% of PICU patients (16). In a study from Turkiye, Yener et al. (17) reported that brain death prevalence was 10.8%. 3 patients had brain death in our study. There was no patient who was an organ donor. There are limited data about brain death patient after pediatric TBI. It will be beneficial to increase education about organ donation to draw attention to organ donation.

Hyperglycemia and abnormal coagulation parameters were independent risk factor for mortality in pediatric traumatic head injury patients (18-20). Coagulation parameters being also high in our on-survivor group were consistent with literature data. However blood glucose level at admission did not differ in our study groups. We determined differences in blood gas parameters (pH, pCO2, lactate, base excess) and renal function tests (blood urea nitrogen, creatinine) in non-survivor group. These results were may be associated with hypoxemia, hypotension and insufficient ventilation.

Our study was conducted as single-center and retrospective. We could not apply intracranial pressure monitorization due to lack of equipment. Patients' data about patient's clinic and interventions made before PICU admission were reliable. Convulsion other than clinic convulsion could not be determined because of lack of continuous electroencephalography (EEG). Pediatric trauma score and ISS was not calculated at emergency service admission. Pediatric trauma score and ISS were calculated from patients records retrospectively. This situation could affect PTS and ISS reliability.

Conclusion

In conclusion, pediatric severe head trauma is an important cause of mortality and morbidity. Adolescent male gender had high incidence of severe TBI. Mortality was high in patients with more severe scores of ISS and PTS, which indicates the severity of trauma. Mortality was also high in patients required RBC transfusion, inotrope and cardiopulmonary resuscitation. Abnormal pupillary response and low GCS at admission were correlated with mortality. Mortality in pediatric severe head trauma was on early period of trauma, especially at the first twenty-four hour. Inotrope requirement and high VIS in PICU score indicate the bad prognosis. Multicentered, prospective studies should be done to determine severe pediatric TBI patients' mortality factors and preventive measures.

Ethical Declarations

Ethics Committee Approval: Our study was approved by our hospital's Ethic Committee Number 1. (Date:24.11. 2021.Decision Number: E2-21-1060)

Informed Consent: Because of the study design was retrospectively, no written informed consent form was obtained from patients.

Conflict of Interest Statement: The authors have no conflict of interest to declare.

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Author contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version of paper.

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