



Research Article

Risk Factors For Post-Traumatic Seizures (Epilepsy) in The Patients With Brain Injury in Turkey

Ümit Naci GÜNDOĞMUŞ¹, Adalet Eda BUDAK¹, Abdulkadir KOÇER², H. Mehmet AKIN¹, Haluk İNCE¹

¹Council of Forensic Medicine, Ministry of Justice, Turkey ²Istanbul Medeniyet University Medical Faculty, Neurology Department, Turkey

Summary

Objective: Post-traumatic seizure following head trauma is a well-known and serious complication. Risk factors vary between study groups. Main objective of the present study is to determine the risk factors for post-traumatic epilepsy (PTE) among patients with head trauma.

Material and method: Medical cases with head trauma, which had been reported between 2006 and 2010 by the 2nd Forensic Medicine Expertise Council of Forensic Medicine Institute, were evaluated in terms of post-traumatic seizures and epilepsy and 1346 patients with serious head trauma were included in the study. Cranial CT and MRI of these patients were evaluated.

Results: PTE was diagnosed in 101 patients (7.5% of overall cases). Generalized tonic-clonic seizure was the most commonly encountered seizure with a prevalence rate of 33.66%. Twenty-four patients had early seizures (23.77%) and 77 patients had late seizures (76.23%). Fractures (68.31%), multiple lobe lesions (31.03%) and contusions (33.33%) were encountered in PTE population. Unconsciousness just after trauma and low Glasgow Coma score were determined in the patients with PTE.

Conclusions: Attention must be paid for the development PTE in the patients with compression fractures, multiple lesions and contusions. Unconsciousness and low Glasgow Coma Score are the other risk factors.

Key words: Trauma, Post-traumatic seizure / epilepsy, risk factor

Türkiye'de Beyin Hasarı Bulunan Hastalarda Post Travmatik Nöbet (Epilepsi) Açısından Risk Faktörleri

Özet

Amaç: Kafa travması sonrası ortaya çıkan post travmatik nöbet iyi tanımlanmış ve ciddi bir komplikasyondur. Risk faktörleri değişik çalışma grupları tarafından farklı bildirilmiştir. Bu çalışmada, ana amaç kafa travmalı hastalarda post travmatik epilepsi (PTE) gelişiminde etkili risk faktörlerini belirlemektir.

Materyal ve metod: 2006-2013 yıllarında Adli Tıp Kurumu 2. Adli Tıp İhtisas Kurulu tarafından hakkında rapor düzenlen kafa travmalı vakalar post travmatik nöbet ve epilepsi açısından değerlendirildi ve ciddi kafa travmalı 1346 hasta çalışmaya dahil edildi. Bu hastaların beyin CT ve MR görüntüleri değerlendirildi.

Bulgular: PTE 101 hastada (incelenen tüm vakaların %7,5'i) teşhis edildi. En sık görülen nöbet %33.66 ile jeneralize tonik klonik nöbet idi. 24 hastada (%23,77) erken nöbet ve 77 vakada (%76,23) geç nöbet vardı. PTE olan grupta kırıklar (%68,31), multipl lob lezyonu (%31,03) ve kontüzyon (%33,33) görüldüğü saptandı. PTE bulunan hastalarda travmadan hemen sonra ortaya çıkan bilinç kaybı ve düşük Glasgow koma skoru saptandı.

Sonuç: Çökme kırığı, multipl lezyon ve kontüzyon bulunan hastalarda PTE gelişimi bakımından dikkatli olunmalıdır. Bilinç kaybı ve düşük Glasgow koma skoru diğer risk faktörleridir.

Anahtar Kelimeler: Travma, Post-travmatik nöbet / epilepsi, Risk faktörü

INTRODUCTION

The nature of permanent neurological damage following traumas plays an important role in determining the penalties that would be imposed in the event of criminal liability. A regulation under the name of “crimes against body immunity” has been made for such cases by means of Turkish Penal Code; prison sentence from three months to three years has been stipulated in case of intentional or involuntary bodily harm; and judgment has been rendered to enhance the penalty in the presence of an incurable disease picture in the subject. Therefore, the relevance of post-traumatic clinical picture to the event the subject has been exposed to, i.e. whether there is causality between trauma and neurological picture (causal link), and improbability of improvement of neurological symptoms, i.e. whether they are permanent or not, should be evaluated precisely. In Turkey, initial evaluation of such cases is made by either relevant health institutions or forensic medicine institutions in the places where injury occurs and the opinion of Forensic Medicine Institute, the 2nd Expertise Council is asked in case there are objections by relevant parties or in case the results are not considered satisfactory by judicial authority.

Head trauma may lead to death and permanent physical, cognitive and behavioral deficits as well as deficits that impair quality of social life. Epilepsy after trauma is one of these sequels⁽¹⁶⁾. In forensic medicine practices, post-traumatic epilepsies are considered as incurable diseases and form a basis to enhance penalty. Therefore it is of great importance to clearly exhibit the relation between epilepsy and trauma. International League against Epilepsy (ILAE) defines epileptic

seizure as temporary symptoms that arise from abnormal hyper or synchronous activity of brain neurons; whereas, epilepsy is defined as permanent tendency of the brain to form an epileptic seizure and a disorder characterized by neurobiological, psychological, cognitive and social consequences of this situation⁽³¹⁾. The diagnosis of epilepsy requires presence of at least two epileptic seizures⁽²³⁾. Post-traumatic epilepsy (PTE) is defined as recurrent and non-provoked seizures that appear in a time ranging from the first 24 hours to months, even years, after head trauma^(25,30,45). It is known that PTE patterns are quite variable in time and may be seen just after trauma or 20-30 years later⁽⁵²⁾. It is stated that the risk is two times higher in mild head traumas and skull fractures and seven times higher in severe head traumas as compared to normal population⁽³⁷⁾. The severity and type of head trauma are the most important risk factors for the development of PTE⁽²²⁾. In head traumas, presence of parenchymal blood, intracranial hematoma (particularly subdural hematoma)^(12,15,24,34,44,54,60), serious brain injury that results in neurological deficit, cortical-subcortical lesions, contusion-laceration, presence of cerebral diffuse contusion^(12,15,24,34,44,50,54,60), and duramater penetration^(11,36) are the risk factors for PTE^(8,11,15,28). Although it shows worldwide variation depending on sociocultural factors, the most common reasons for head trauma-related seizures generally include traffic accident and sports injury in young people and falls in elderly^(8,19,28,46).

In Turkey, there is no comprehensive study conducted on this subject. The present study, which evaluated post-traumatic epileptic seizure cases, inquired the factors demonstrated to be associated with

seizures in many previous studies [age of the individual at the time of event, type and severity of head trauma, presence of intracranial hematoma, whether it is compression fracture or not, presence and duration of loss of consciousness after the event, presence of fire arm injury, and score of Glasgow Coma Scale (GCS)].

MATERIAL AND METHODS

The present study comprises the individuals that had been exposed to head trauma, for whom Forensic Medicine Institute, 2nd Forensic Medicine Expertise Council delivered an opinion between January 2006 and December 2011. Presence of two or more seizures including those developed in early period after the event was considered PTE. Head trauma-related factors (type and severity of head trauma, presence of intracranial hematoma, whether it is compression fracture), age of the individual, and initial neurological examination findings after the event (presence and duration of loss of consciousness after the event, score of Glasgow Coma Scale) were recorded via an inquiry form (Table 1). Data from these parameters were subjected to frequency analysis first for overall 1346 cases and then for 101 cases with PTE; thereafter, both groups were compared (Table 2 and 3).

With regard to the probability of simulation effort made by forensic cases by means of displaying epilepsy-like seizures to show that they are in worse situation than it was, observation in a university hospital for definite diagnosis was recommended for these cases and post-observation diagnoses were regarded as the definite diagnoses.

Time of seizure after head trauma was dichotomized according to the times the seizure appeared (early and late seizures). Whilst the seizures encountered in 7 days

after trauma were assigned to early seizure group, seizures after the first week of trauma were assigned to late seizure group. Seizures in the first 24 hours of injury were defined as the earliest (immediate) seizure^(3,5,10,14,29,44,53,59). In the present study, the earliest and early seizures were evaluated in a single group.

The severity of head trauma was assessed using the head trauma classification criteria, which were recommended by Annegers et al. (1980) and have been used by many researchers until today. The cases were divided into three groups as “mild head trauma (presence of bone fracture and presence of posttraumatic amnesia or loss of consciousness for less than 30 minutes), moderate head trauma (bone fracture present or absent, posttraumatic amnesia or loss of consciousness for 30 minutes-24 hours), severe head trauma (contusion, intracranial hematoma, posttraumatic amnesia or loss of consciousness for 24 hours or longer)”⁽²⁵⁾.

Statistical method

Graph Pad In Stat 3 (Trial) Multiple Regression analysis was used. Type of head trauma, GCS score, state of consciousness, fracture in cranial bones, anatomic localization of lesion, anatomic side, type of lesion, antiepileptic drug use, type of seizure, posttraumatic amnesia, surgical procedure, and presence of fire arm bullet, pellet or bone particle penetrated into the brain tissue were considered as independent variables, whereas epilepsy was considered as dependent variable. At first, overall distribution and means of all variables were calculated. Thereafter, cross distribution of the variables that are related to each other was calculated. Level of significance was considered to be $p \leq 0.05$.

Table 1: Questionnaire used for the evaluation of 1346 cases that were exposed to head trauma

Age,
Gender,
Type of head trauma; traffic accident – any kind of in-car and out-car motor vehicle accidents, injuries due to blunt-force trauma, gunshot injuries, penetrating injuries, crush injuries, falls from height,
Glasgow coma score (GCS)
State of consciousness
*data stated by the hospital, which has been admitted first after trauma, were taken into account while evaluating GCS and consciousness,
Whether there is Cerebrospinal Fluid (CSF) drainage,
Presence of pneumocephaly,
Whether there is shift,
Presence of cerebral edema,
Presence of axonal injury,
Presence of posttraumatic amnesia,
Antiepileptic drug use; drug use at any time after trauma was taken into account,
Whether there is EEG analysis; if yes, characteristic of EEG,
Syncope at the time of event,
Presence of firearm bullet or pellet, or metallic object and bone particles in the cerebral tissue,
Fracture in the cranial bones
Anatomic side; right, left, bilateral,
Anatomic localization; parietal-temporal-occipital-frontal bones-sinus fractures
Type of fracture; linear, compression, extending to the base,
Of the intracranial lesion,
Anatomic localization; parietal-temporal-occipital-frontal-hemispheric,
Anatomic side; right, left, bilateral,
Type of lesion; SDH, EDH, SAH, intraparenchymal hematoma, contusion,
Surgical intervention
Craniotomy-craniectomy-burr hole-duraplasty,
Time and type of seizure
Early seizure: seizures that occur within the first week following trauma
Late seizure: Seizures that occur after the first week following trauma,
Comment: PTE (+) or (-).

Table 2: Descriptive findings in epileptic and non-epileptic cases

Variable	Non-epileptic n=1245 (%)	Epileptic n=101 (%)	P value
Age			<0.05
<15	86 (6.91)	29 (28.71)	
15-40	633 (50.84)	57 (56.43)	
>40	417 (33.49)	15 (14.85)	
Unknown	109 (8.76)	-	
Gender			NS
Male	1072 (86.10)	86 (85.14)	
Female	173 (13.90)	15 (14.85)	
Cause of Trauma			<0.01
Battery	520 (43.37)	36 (39.56)	
Traffic accident	283 (22.73)	34 (37.36)	
Gunshot injury	89 (7.14)	10 (9.90)	
Fall	60 (4.81)	7 (7.69)	
Sharp-force injury	17 (1.36)	4 (4.39)	
Unknown	276 (22.16)	10 (9.90)	
GCS Score			<0.05
<8	63 (5.06)	11 (11.19)	
9-15	142 (11.40)	12 (12.39)	
Unknown	1040 (83.53)	78 (77.22)	
Conscious			<0.01
Open	439 (35.26)	16 (15.84)	
Brain fog	205 (16.47)	23 (22.77)	
Close	227 (18.23)	40 (39.60)	
Unknown	374 (30.04)	22 (21.78)	
Post-traumatic amnesia			<0.05
Yes	33 (2.65)	6 (5.94)	
No	1212 (97.35)	95 (94.05)	
CSF drainage			NS
Rhinorrhea	7 (0.52)	-	
Otorrhea	17 (1.56)	4 (4.05)	
Rhinorrhea+ Otorrhea	1 (0.14)	1 (1.01)	
No	1220 (97.77)	96 (95.04)	
Pneumocephaly			NS
Yes	291 (23.77)	29 (28.71)	
No	954 (76.22)	72 (71.28)	

*n: number, NS: non-specific, GCS: Glasgow Coma Scale

Table 3: Clinical and radiological examination findings

Variable	Non-epileptic n=1245 (%)	Epileptic n=101 (%)	P value
Metallic object or bone			NS
Yes	53 (4.53)	8 (7.92)	
No	1192 (95.46)	93 (92.07)	
Bone fracture			NS
Yes	896 (71.69)	69 (68.31)	
No	349 (28.30)	32 (31.68)	

Side of trauma			NS
Right	325 (36.58)	28 (40.57)	
left	425 (47.15)	30 (43.47)	
Bilateral	69 (7.87)	7 (10.14)	
Unknown	74 (8.08)	4 (5.79)	
Type of fracture			<0.05
Compression	362 (40.93)	33 (47.82)	
Linear	234 (25.69)	14 (20.28)	
Extending to the base	43 (4.55)	1 (1.44)	
Multiple fractures	39 (4.04)	-	
Unknown	214 (24.35)	21 (30.43)	
Axonal injury			<0.05
Yes	19 (1.85)	6 (5.94)	
No	1226(98.14)	95 (94.05)	
Cerebral injury/lesion			<0.01
Yes	679 (56.90)	87 (86.13)	
No	566 (43.09)	14 (13.81)	
Side of injury			NS
Right	227 (33.28)	28 (32.18)	
Left	269 (39.03)	30 (34.48)	
Bilateral	183 (27.66)	29 (33.32)	
Type of lesion			<0.01
Subdural hematoma	169 (23.49)	11 (13.09)	
Subarachnoid hemorrhage	141 (19.32)	7 (8.33)	
Epidural hematoma	142 (19.06)	4 (4.76)	
Multiple (Mixed)	120 (19.45)	29 (34.52)	
Contusion/ parenchymal injury	16 (6.39)	33 (39.28)	
Lesion area			<0.01
Multiple lobes	414 (57.30)	25 (36.83)	
Frontal lobe	101 (14.36)	9 (13.04)	
Parietal lobe	90 (13.57)	14 (20.28)	
Temporal lobe	73 (11.74)	17 (24.63)	
Occipital lobe	11 (1.98)	4 (5.79)	
Cerebral edema			NS
Yes	126 (10.77)	19 (18.81)	
No	1119(89.22)	82 (81.18)	
Shift			<0.05
Yes	31 (2.80)	8 (7.93)	
No	1214 (97.10)	93 (92.07)	

*n: number, NS: non-specific

RESULTS

Socio-demographic and descriptive characteristics of the cases are demonstrated in Table 2. Clinical and radiological findings are demonstrated in Table 3. Whilst battery-related head trauma that appeared after fight/manhandling/quarrel was the most common reason both in PTE and non-PTE cases, traffic accident-related head trauma

was the second most common reason (Table 2). Low coma score, posttraumatic loss of consciousness, and presence of posttraumatic amnesia were more prevalent among PTE cases with p values found <0.05, <0.01 and <0.05 respectively (Table 2). Comparison of both groups in terms of clinical examination findings and radiological findings revealed that axonal injury (p<0.05), bone fracture type

($p<0.05$), brain injury and multi lesions ($p<0.01$), subdural hemorrhage ($p<0.01$), and lesion-related shift ($p<0.05$) were more prevalent in PTE cases (Table 3).

PTE was detected in 101 cases (7.5%). Early seizure was diagnosed in 24 cases (of which 5 had “earliest” seizure at the moment of trauma) and late seizure was diagnosed in 77 cases. Of the cases with seizures ($n=101$), 23.76% ($n=24$) had seizures that do not cause continuous weakening of sense or any organ functions and that can be controlled with medicines. Generalized tonic-clonic seizure was the most common type of seizure (33.6%). Other seizures included partial (10.89%), complex partial (9.90%), and secondary generalized (6.93%) seizures. Type of seizure could not be identified in 38.61% ($n=39$) of the cases. Of the early seizures, 54.16% were generalized tonic-clonic and 8.33% were partial; whereas, type of seizure could not be identified in 37.50% of these cases. Of the late seizures, 27.27% were generalized tonic-clonic, 11.68% were partial, 12.98% were complex partial and 9.09% were secondary generalized seizure.

Table 3 demonstrates that intracranial lesion was detected in 56.90% ($n=766$) of the cases and the most prevalent intracranial lesion was subdural hematoma with a prevalence rate of 23.49%. The most common localization of intracranial lesions was multiple (in more than one lobe) detected at a rate of 44.51%, whereas the second and third most common localizations were frontal lobe (14.36%) and parietal lobe (13.57%) respectively (Table 3). Distribution of the lesions was similar (34.48% left side, 32.18% right side) in 87 (86.13%) cases, which had intracranial lesion but not seizure. Although the lesions were multiple in 1/3 of the cases, they were in the frontal lobe in 25.28%.

Evaluation of 1346 cases in terms of brain edema demonstrated that 10.77% had brain edema; whereas, brain edema was present

in 18.81% ($n=19$) of the cases with seizure. It was determined that 18 of these 19 cases had intracranial lesion and skull fracture together with brain edema. It was determined that 2.8% of the cases had shift and that the shift was more prevalent in epileptic cases and more prevalently (62.5%) to the left side; i.e. right hemispheric lesions were more common. Brain edema, loss of consciousness and detecting lesion-related shift were found to be associated with presence of PTE ($p<0.05$).

Skull fracture was detected in 71.69% of PTE cases, of which 40.93% were depression fractures. Multiple bone fractures, particularly parietal bone fractures were the most commonly encountered traumatic fragmentations. Of the PTE cases, 86% had intracranial lesions located most commonly in the temporal lobe (16.5%). Surgical intervention had to be performed more prevalently in PTE cases as compared to non-PTE cases at a rate of 45% and 35% respectively.

DISCUSSION

PTE accounts for 20% of symptomatic epilepsies and 4-6% of overall epilepsies in general population^(10,17,42,44). Many studies reported that the risk of PTE is the highest in young adulthood between the ages of 15 and 24 years^(8,24,37). The risk is 3-4 times higher in males versus females^(8,14,42). Military studies reported the incidence of PTE after head trauma to be 32-53%^(1,4,11,48). Salazar et al. (1985) conducted a study in Vietnam veterans and reported that PTE was present in 53% of the individuals that had been exposed to penetrating head trauma 15 years ago and that seizures continued over the course of study. They reported that the risk of epilepsy was 580 times higher than normal population sample at the same age range in the first year of injury, whereas it decreased to 25 times after 10 years⁽⁵⁴⁾. The incidence of PTE after injuries in daily life has been reported between 4.4% and

13.6% depending on the severity of trauma, presence of comorbid condition, duration of hospital stay and presence of concomitant depression⁽²²⁾. In the present study, seizure was defined in 7.5% (n=101) of 1346 cases, which have been evaluated in terms of history of trauma. In the present study, the age group, in which seizure after head trauma has been most frequently defined, consisted of middle-aged adults between 36 and 40 years (Table 1). The present study found the incidence of seizure 5 times higher in males than females. This might be regarded as the reflection of six times higher number of males versus females among the cases that had been exposed to head trauma. Higher number of male cases with head trauma, which is consistent with the literature, was attributed to the fact that males most often work at hazardous occupations, involve in more tasks in the population, and are more prone to violence.

The most common causes of head trauma are traffic accidents, falls and battery^(35,47). In the present study, the most common cause of head trauma was battery (52.50%) followed by traffic accident (29.9%). It was observed that the most common cause was also battery (39.56%) followed by traffic accident in 91 cases with seizure, in which the cause of trauma has been identified (Table 2). Here, another conspicuous issue is determining that more complex injuries such as traffic accident or penetrating injuries were more prevalent in PTE cases (Table 1). Although, previous studies reported that the most common causes of head trauma are traffic accidents and falls from height⁽⁴⁷⁾, determining that the most common cause of trauma was battery in the present study can be explained by the cases' being forensic cases, traffic accident cases' being less commonly a litigious question than battery cases due to their nature, and the databases' in the other studies heavily comprising clinical cases.

The course of PTE may show differences and the risk continues for years^(52,54). Although development of PTE shows variations, it occurs by 50% in one year following trauma and the risk is the highest in the first two years^(21,37,50,60). Of the seizures described in the present patients, of whom the initial evaluation was made after a mean of 1.5 years and who then followed for a mean of at least 3 years, 23.76% were considered as early seizure and 76.23% were considered as late seizure. Early seizure is also a risk factor for the development of recurrent late seizures^(10,13,18,24,33,56). In the present study, this assessment could not be performed since we had difficulty in reaching to the follow-up records about early seizures. Various studies reported that seizures may appear during long-term follow-up. Eftekhar et al. (2009) stated that 189 veterans have been followed during Iran-Iraq war and the incidence of seizure was 86.4% after 16 years and 74.7% after 21 years and that early seizure, prophylactic drug use or surgical intervention does not influence long-term outcomes in terms of significantly permanent seizures⁽²⁰⁾. Englander et al. (2003) described PTE in 10% of 647 cases and reported the incidence of late seizure to be 7.8% in 16–30-year age group (21). In the present study, the incidence of late seizure in 48 cases in 16–30-year age group was found to be 7.1% as was in the study of Englander.

Temkin (2003) evaluated 783 patients for two years and reported the incidence of late seizure to be 4% in the patients with low GCS (3-8) but without cortical contusion, intracerebral hematoma, compression fracture, penetrating injury or early seizure, which were considered as criteria⁽⁵⁷⁾. In the present study, evaluation of 228 cases, the data of which were reached based on GCS values reported in the health care center they have been first brought to after head trauma (Table 2), revealed that cases with low GCS score ranked first as was in the study conducted

by Kırış et al. (1998)⁽³⁸⁾. In the present study, 28.10% of the cases were rendered unconscious. It was determined that 50.63% of the patients with definite seizure and available data on conscious were unconscious at the time of event. In many studies in the literature, the severity of loss of conscious is considered as one of the criteria that determine the severity of head trauma, which is one of the risk factors for the development of PTE⁽²⁵⁾. In the present study, duration of unconsciousness could not be determined in unconscious cases. Presence of unconsciousness at the time of event in 50.63% (n=40) of the cases with available data on state of consciousness despite undefined duration of unconsciousness can be considered as a significant data. In the literature, it is stated that conscious of patient is usually open in case of open penetrating injuries although they are severe and fatal injuries⁽⁴⁹⁾ but that unconscious cases are encountered more frequently in case of closed trauma⁽²⁶⁾. In the present study, of the cases with open conscious at the time of event, 92.96% had compression fracture, 73.40% had multiple fractures, and 74.94% had intracranial lesions in multiple lobes. Multiple bone fractures and intracranial lesions in multiple lobes were detected in 2.67% of 267 unconscious cases. These results are consistent with the literature.

In the present study, CSF drainage was detected in 9.90% of the cases with seizure. It is stated that rhinorrhea can be seen in anterior cranial fossa fractures and otorrhea can be seen in middle cranial fossa fractures^(26,40). In the present study, skull fracture was detected in all of 30 cases having CSF drainage. As is mentioned in the literature, these fractures can be called as fractures of base of skull.

Axonal injury is common among head traumas. In the literature, it is reported that early impacts may change into chronic vegetative state or death depending on the severity of injury^(43,49) and that traffic accident is the most common cause⁽⁵⁵⁾. In

the present study, detecting epileptic seizures and cognitive problems in 64% of 25 cases with axonal injury and traffic accident's being the cause of trauma in 80% are consistent with the literature.

In the present study, bone fracture was determined in 965 (71.69%) cases. It was observed that the left side was the most frequent distribution of fracture with the compression fracture determined to be the most prevalent type of fracture. Whilst fractures of multiple localizations ranked first, fractures of the parietal region ranked second. Similar findings were determined when 101 patients with seizure were taken into account; however, temporal bone injury was the most common lesion after multiple localizations. Compression fracture was the most prevalent (47.82%). In the literature as well, it is stated that the risk of PTE is high in compression fractures^(7,8,28,29,30). In a study conducted by Tuncer et al. (1989) with reference to Jennet and Lewin's study, PTE was determined in 12.5% of the cases with compression fracture, whereas they reported the prevalence of PTE to be 9% in their own study conducted in 122 cases with compression fracture⁽⁵⁸⁾. Likewise, in the present study, PTE was detected in 8% of 423 cases with compression fracture.

The prevalence of posttraumatic seizure in pediatric cases was reported to be 7–15%^(29,41). Ö.Ateş et al. (2006) conducted a study in 1785 head trauma cases aged less than 16 years and reported that the risk of seizure is high in the patients that were less than 3 years of age, had cerebral edema, intracranial hemorrhage and compression fracture⁽⁶⁾. In the present study, posttraumatic seizure was encountered in 13% of 131 cases under the age of 16 years, which is consistent with the literature. Ratan et al. (1999) reported posttraumatic seizure in 21% of 400 children aged less than 12 years, of which 20% had early seizure⁽⁵¹⁾. In the present study, posttraumatic seizure was observed in 11 (11%) of 90 cases aged less than 12

years. N.Temkin (2003) conducted a study in 783 cases and reported the incidence of early seizure as 25% in the cases with subdural hematoma, compression fracture and intracerebral hemorrhage⁽⁵⁷⁾. In the present study, the incidence of early seizure in the cases with subdural hematoma and compression fracture was close (17%) to the above mentioned study. The risk of PTE during penetrating head trauma to the duramater is reported to be 20–57%^(32,60). This rate was found quite low in the present study. PTE was identified in 11.25% of 80 cases that underwent duraplasty due to duramater laceration.

Although there is a great variety of PTE-related seizures, primary or secondary generalized tonic-clonic seizures are the leading^(12,26). Partial seizures may also be seen in symptomatic epilepsy due to cortical injury that results from infection, head trauma and birth traumas⁽³⁹⁾. Simple partial motor and sensorial seizures indicate structural brain injury, focal onset identifies the localization of lesion; contrarily, generalized seizures indicate cellular, biochemical or structural impairment⁽³⁶⁾. Early seizures may be generalized in closed traumas with diffuse brain injury⁽¹¹⁾. Focal seizures are usually encountered in children and in the adults with gunshot injuries⁽²⁶⁾. Although PTE appears as rapidly as secondary generalized seizures, they are usually partial at the beginning. Of PTE patients, one-fourth has local seizures, one-half has seizures that begin as focal and then progress as generalized, and remaining one fourth has generalized seizures alone⁽¹¹⁾. CA1 and CA3 pyramidal cells in the hippocampus are extremely sensitive to ischemia and PTE is seen in the form of temporal lobe epilepsy in hyperexcitability situations that emerge from trauma at cellular level⁽²⁷⁾. Temporal lobe epilepsy is also among the most common epilepsies in adults⁽⁹⁾. In the present study, as it would be seen in the conclusion section, the most prevalent type of seizures was reported to

be generalized tonic-clonic seizure. Focal and complex partial seizures were more common in the cases with gunshot injury, which is consistent with the literature. It is thought that secondary generalized seizures' being reported less frequently can be explained by the fact that diagnosis of seizure has been made based on observation of the family rather than clinical follow-up.

Detailed and precise recording from the time of initial health service to the time of last treatment is of great importance for clinical and radiological evaluation of the cases that have been exposed to head trauma. Difficulty in studying with standardized methods during acute phase of injury and lack of clinical documentations unfavorably influence the evaluation of retrospective studies⁽²⁾. This may be considered as the limitation of the present study. Nevertheless, in the present study that collected valuable data, it is observed that age distribution peaks between the ages of 16 and 40 years in all cases and in the cases with seizure; battery is the leading cause of trauma; and the lesions are most frequently encountered in the left side. Substantial proportion of the cases has been evaluated at least 18 months later. In the cases with fracture, multiple bone fractures ranked first, temporal bone fractures ranked second and parietal bone fractures ranked third. The most common type of bone fracture was compression fracture. Statistical evaluation of the study revealed correlation between the localization of intracranial lesions that occurred during head trauma and the development of PTE. In the majority of the cases that had seizure, intracranial lesion was in more than a single lobe and mostly in the frontal lobe. Contusion accounted for the great majority of intracranial lesions. In the present study, no evaluation was made in terms of the relation between early seizure and late seizure because of lack of detailed information on this subject.

In conclusion, the present study, which was conducted based on official report, is notable in terms of both sample size and long-term follow-up. We think that anamnesis on battery and traffic accident should be obtained precisely for the etiology of PTE after the present study, which exposed the extent of relation between trauma and epileptic seizure.

Correspondence to:

Abdulkadir Koçer

E-mail: abdulkadirkocer@yahoo.com

Received by: 04 December 2013

Revised by: 23 October 2014

Accepted: 10 December 2014

The Online Journal of Neurological Sciences (Turkish) 1984-2015

This e-journal is run by Ege University

Faculty of Medicine,

Dept. of Neurological Surgery, Bornova,
Izmir-35100TR

as part of the Ege Neurological Surgery

World Wide Web service.

Comments and feedback:

E-mail: editor@jns.dergisi.org

URL: <http://www.jns.dergisi.org>

Journal of Neurological Sciences (Turkish)

Abbr: J. Neurol. Sci.[Turk]

ISSNe 1302-1664

REFERENCES

1. Aarabi B, Taghipour M, Haghnegahdar A, Farokhi M, Mobley L. Prognostic factors in the occurrence of posttraumatic epilepsy after penetrating head injury suffered during military service. *Neurosurgical focus* 2000; 8(1), 1-6.
2. Angeleri F, Majkowski J, Cacchio G, Sobieszek A, d'acunto S, Gesuita R, et al. Posttraumatic Epilepsy Risk Factors: One Year Prospective Study After Head Injury. *Epilepsia* 1999; 40(9), 1222-1230.
3. Annegers JF, Grabow JD, Groover RV, Laws ER, Elveback LR, Kurland LT. Seizures after head trauma: a population study. *Neurology* 1980; 30(7), 683-689.
4. Annegers JF, Hauser WA, Coan SP, Rocca WA. A population-based study of seizures after traumatic brain injuries. *N Engl J Med* 1998; 338(1), 20-24.
5. Asikainen I, Kaste M, Sarna S. Early and Late Posttraumatic Seizures in Traumatic Brain Injury Rehabilitation Patients: Brain Injury Factors Causing Late Seizures and Influence of Seizures on Long Term Outcome. *Epilepsia* 1999; 40(5), 584-589.
6. Ateş Ö, Öndül S, Önal Ç, Büyükkiraz M, Somay H, Çaylı SR, et al. Post-traumatic early epilepsy in pediatric age group with emphasis on influential factors. *Child's Nervous System* 2006; 22(3), 279-284.
7. Barlow KM, Spowart JJ, Minns RA. Early posttraumatic seizures in non accidental head injury: relation to outcome. *Developmental Medicine & Child Neurology* 2000; 42(9), 591-594.
8. Benardo LS. Prevention of epilepsy after head trauma: do we need new drugs or a new approach? *Epilepsia* 2003; 44, 27-33.
9. Blume WT. Hippocampal Cell Loss in Posttraumatic Human Epilepsy. *Epilepsy Currents* 2007; 7(6), 156-158.
10. Bushnik T, Englander J, Duong T. Medical and social issues related to posttraumatic seizures in persons with traumatic brain injury. *The Journal of Head Trauma Rehabilitation* 2004; 19(4), 296-304.
11. Caveness WF, Meirrowsky AM, Rish BL, Mohr JP, Kistler JP, Dillon JD, et al. The nature of posttraumatic epilepsy. *Journal of neurosurgery* 1979; 50(5), 545-553.
12. Caveness WF, Walker AE, Ascroft PB. Incidence of posttraumatic epilepsy in Korean veterans as compared with those from World War I and World War II. *Journal of neurosurgery* 1962; 19, 122.
13. Chadwick D. Seizures and epilepsy after traumatic brain injury. *Lancet* 2000; 355(9201), 334-336.
14. D'Ambrosio R, Perucca E. Epilepsy after head injury. *Current opinion in neurology* 2004; 17(6), 731-735.
15. De Santis A, Sganzerla E, Spagnoli D, Bello L, Tiberio F. Risk factors for late posttraumatic epilepsy. *Acta neurochirurgica Supplementum* 1992; 55, 64.
16. Diaz Arrastia R, Agostini MA, Madden CJ, Van Ness PC. Posttraumatic epilepsy: the endophenotypes of a human model of epileptogenesis. *Epilepsia* 2009; 50, 14-20.
17. Diaz-Arrastia R, Agostini MA, Frol AB, Mickey B, Fleckenstein J, Bigio E, et al. Neurophysiologic and neuroradiologic features of intractable epilepsy after traumatic brain injury in adults. *Archives of Neurology* 2000; 57(11), 1611-1616.

18. Dichter MA. Posttraumatic epilepsy: the challenge of translating discoveries in the laboratory to pathways to a cure. *Epilepsia* 2009; 50, 41-45.
19. Dolinak D, Matshes E, Lew EO. Forensic pathology Principles and Practice Elsevier Academic Press; 2005.
20. Eftekhari B, Sahraian MA, Nouralishahi B, Khaji A, Vahabi Z, Ghodsi M, et al. Prognostic factors in the persistence of posttraumatic epilepsy after penetrating head injuries sustained in war. *Journal of neurosurgery* 2009; 110(2), 319-326.
21. Englander J, Bushnik T, Duong TT, Cifu DX, Zafonte R, Wright J, et al. Analyzing risk factors for late posttraumatic seizures: A prospective, multicenter investigation. *Archives of physical medicine and rehabilitation* 2003; 84(3), 365-73.
22. Ferguson PL, Smith GM, Wannamaker BB, Thurman DJ, Pickelsimer EE, Selassie AW. A population based study of risk of epilepsy after hospitalization for traumatic brain injury. *Epilepsia* 2010; 51(5), 891-898.
23. Fisher RS, Boas WE, Blume W, Elger C, Genton P, Lee P, et al. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). *Epilepsia* 2005; 46(4), 470-472.
24. Frey LC. Epidemiology of posttraumatic epilepsy: a critical review. *Epilepsia* 2003; 44, 11-7.
25. Garga N, Lowenstein DH. Posttraumatic epilepsy: a major problem in desperate need of major advances. *Epilepsy Currents* 2006; 6(1), 1-5.
26. Granacher PR. Traumatic brain Injury Methods for clinical and forensic neuropsychiatric assessment 2nd ed. Boca Raton: CRC Press Taylor & Francis Group; 2008.
27. Griesemer D, Mautes AM. Closed head injury causes hyperexcitability in rat hippocampal CA1 but not in CA3 pyramidal cells. *Journal of Neurotrauma* 2007; 24(12), 1823-1832.
28. Gupta Y, Gupta M. Post traumatic epilepsy: a review of scientific evidence. *Indian J Physiol Pharmacol* 2006; 50(1), 7-16.
29. Hahn YS, Fuchs S, Flannery AM, Barthel MJ, McLone DG. Factors influencing posttraumatic seizures in children. *Neurosurgery* 1988; 22(5), 864-867.
30. Haltiner AM, Temkin NR, Dikmen SS. Risk of seizure recurrence after the first late posttraumatic seizure. *Archives of physical medicine and rehabilitation* 1997; 78(8), 835-840.
31. Hauser WA, Annegers JF, Rocca WA, editors. Descriptive epidemiology of epilepsy: contributions of population-based studies from Rochester, Minnesota 1996: Mayo Clinic.
32. Hauser WA, Rich SS, Lee JRJ, Annegers JF, Anderson VE. Risk of recurrent seizures after two unprovoked seizures. *New England Journal of Medicine-Unbound Volume* 1998; 338(7), 429-434.
33. Hup CK, Tharakan J, Pal HK, Khan N, Chin TY, USM MMN. Risk Factors and Phenytoin Prophylaxis for Early Post-Traumatic Seizures among Patients with Traumatic Brain Injury. *Malaysian Journal of Medical Sciences* 2010; 17(3), 36-43.
34. Jantz PB, Coulter GA. Child and adolescent traumatic brain injury: Academic, behavioural, and social consequences in the classroom. *Support for learning* 2007; 22(2), 84-89.
35. Jennett B. Epidemiology of head injury. *Journal of Neurology, Neurosurgery & Psychiatry* 1996; 60(4), 362-369.
36. Kasper DL Harrison's principles of internal medicine. Ed's Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, McGraw-Hill Medical, 2005, ed 16.
37. Kharatishvili I, Pitkanen A. Posttraumatic epilepsy. *Current Opinion in Neurology* 2010; 23(2), 183-188.
38. Kırış T, İş M, İmer M. ve ark. Nöroşirürjide travma pratiği. Prospektif epidemiyolojik çalışma. *Ulusal Travma Dergisi*. 1998; 4, 281-284
39. Köksal A, Öztürk M, Sözmen V, Altunkaynak Y. Epilepsi Polikliniğinde izlenen hastaların ILAE'nin önerdiği yöntemlerle nöbet ve epilepsi tiplerine göre sınıflandırılması. *Epilepsi* 2004; 10(3), 149-153
40. Leestma JE. Forensic neuropathology Boca Raton CRC press, 2009, ed 2.
41. Lewis RJ, Yee L, Inkelis SH, Gilmore D. Clinical predictors of post-traumatic seizures in children with head trauma. *Annals of emergency medicine*. 1993; 22(7), 1114-1118.
42. Li S, Wang X, Wang J, Wu J. Cerebrovascular and posttraumatic epilepsy. *Neurology Asia*. 2004; 9(1), 12-13
43. Lindsay KW, Bone I Çev. Mustafa Bozbuğa Resimli Açıklamaları ile Nöroloji ve Nöroşirürji Nobel Tıp Kitabevleri, 2000
44. Lowenstein DH. Epilepsy after head injury: an overview. *Epilepsia* 2009; 50, 4-9.
45. Mazzini L, Cossa FM, Angelino E, Campini R, Pastore I, Monaco F. Posttraumatic Epilepsy: Neuroradiologic and Neuropsychological Assessment of Long term Outcome. *Epilepsia* 2003; 44(4), 569-574.
46. Moreno A, Peel M. Posttraumatic seizures in survivors of torture: manifestations, diagnosis, and treatment. *Journal of Immigrant Health* 2004; 6(4), 179-186.
47. Ökten Aİ, Ergün R, Beşkonaklı E, Akdemir G, Bostancı U, Gezici AR ve ark. Kafa Travmasında Prognozu ve Ölüm Oranını Etkileyen Unsurlar. *Türk Nöroşirürji Dergisi* 1997; 7(2), 51-59
48. Pagni CA, Zenga F. Prevention and treatment of post-traumatic epilepsy. *Expert Review of Neurotherapeutics* 2006; 6(8), 1223-1233.
49. Payne-James J, Busuttill A, Smock WS. Forensic medicine: clinical and pathological aspects: Cambridge University Press; 2003.
50. Pohlmann Eden B, Bruckmeier J. Predictors and dynamics of posttraumatic epilepsy. *Acta Neurologica Scandinavica* 1997; 95(5), 257-262.

51. Ratan SK, Kulshreshtha R, Pandey RM. Predictors of posttraumatic convulsions in head-injured children. *Pediatric Neurosurgery* 2000; 30(3), 127-131.
52. Rinaldi A, Conti L. Posttraumatic epilepsy. *Neurological Sciences*. 2003; 24(4), 229-230.
53. Rish BL, Caveness WF. Relation of prophylactic medication to the occurrence of early seizures following craniocerebral trauma. *Journal of Neurosurgery* 1973; 38(2), 155-158.
54. Salazar AM, Jabbari B, Vance SC, Grafman J, Amin D, Dillon JD. Epilepsy after penetrating head injury. I. Clinical correlates: a report of the Vietnam Head Injury Study. *Neurology* 1985; 35(10), 1406-1414.
55. Shkrum MJ, Ramsay DA, Forensic pathology of trauma Totowa, New Jersey Humana Press 2007.
56. Spencer S. Seizures and epilepsy. Cecil Medicine 23rd ed Philadelphia, PA: Saunders Elsevier. 2007.
57. Temkin NR. Risk factors for posttraumatic seizures in adults. *Epilepsia* 2003; 44, 18-20.
58. Tuncer R, Saveren M. Post-traumatic epilepsy and EEG abnormality in depressed fractures. *Turkish Neurosurgery* 1989; 1, 23-25.
59. Verellen RM, Cavazos JE. Post-traumatic epilepsy: an overview. *Therapy* 2010; 7(5), 527-531.
60. Willmore L, Ueda Y. Posttraumatic epilepsy: hemorrhage, free radicals and the molecular regulation of glutamate. *Neurochemical Research* 2009; 34(4), 688-97.