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# Medicolegal Evaluation of Post-Traumatic Cognitive Impairments

## Travmatik Kognitif Yıkımın Medikolegal Değerlendirilmesi

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### ABSTRACT

**Objective:** Head injuries and traumatic brain injury are a serious public health problem impacting. It is well-known that each year, 55 million people are exposed to traumatic brain injury. The identification of head traumas, Traumatic Brain Injury, and Post-Traumatic Cognitive Impairment in respect of clinical forensic medicine applications are important in many judicial cases, primarily the claim that a disability is due to trauma.

**Methods:** The files of the patients who were referred to our center for disability assessment between January 01, 2019 and December 31, 2019, and who were found to have suffered head trauma at the time of the incident, were retrospectively reviewed.

**Results:** The ages of the patients ranged between 14 and 78 years. The mean age was  $39.8 \pm 14.43$ , and the median age was 39. One hundred nine (81.3%) of them were male and 25 (18.7%) were female. As a result of the examination, imaging tests and neurocognitive test results for the disability assessment of the patients, 87 (64.9%) of them had organic brain damage causing significant cognitive damage. Post-traumatic stress disorder in 5 (3.7%) patients and other trauma-related sequelae (craniectomy defect, epilepsy, hearing or vision loss, etc.) were detected in 8 (6%) patients. The Wechsler Memory Scale (WMS)-I and WMS-II scores of the patients were found to be significantly lower in the Post-Traumatic Cognitive Impairment group ( $p=0.001$ ). Moreover, the test results were determined to be significantly lower in WMS-IV (logical memory) tests ( $p=0.001$ ).

**Conclusion:** The diagnosis of Post-Traumatic Cognitive Impairment can be made through multidisciplinary clinical examination, radiological imaging, and neurocognitive tests.

**Keywords:** Cognitive impairment, head trauma, brain damage, neurocognitive test



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## ÖZ

**Amaç:** Kafa travmaları ve travmatik beyin hasarı ciddi bir halk sağlığı sorunudur. Her yıl 55 milyon kişinin travmatik beyin hasarına maruz kaldığı bilinmektedir. Klinik adli tıp uygulamaları açısından kafa travmaları, travmatik beyin hasarı ve kognitif yıkımın tanımlanması ve ispatlanması travma kaynaklı engellilik iddiaları başta olmak üzere birçok adli dosyada önem arz etmektedir.

**Yöntem:** Merkezimize 01 Ocak 2019-31 Aralık 2019 tarihleri arasında özür lülük/maluliyet değerlendirmesi için başvuran ve olay anında kafa travması olduğu tespit edilen hastaların dosyaları geriye dönük olarak incelendi.

**Bulgular:** Olguların yaşları 14 ile 78 arasında değişmekteydi. Ortalama yaş  $39,8 \pm 14,43$ , median yaş ise 39'du. 109'u (%81,3) erkek 25'i (%18,7) kadındı. Olguların yapılan muayene, görüntüleme tetkikleri ve nörokognitif test sonuçları sonucunda 87'sinde (%64,9) belirgin kognitif yıkıma sebep olan travmatik beyin hasarı tespit edildi. On üç (%9,7) olguda ise travmaya bağlı diğer (kraniektomi defekti, epilepsi, işitme veya görme kaybı vb.) sekeller tespit edildi. Hastaların Wechsler Bellek Ölçeği (WMS)-I ve WMS-II puanları Post-Travmatik Kognitif Yıkım grubunda anlamlı olarak düşük bulundu ( $p=0,001$ ). Ayrıca WMS-IV (mantıksal bellek) test sonuçlarının anlamlı olarak daha düşük olduğu belirlendi ( $p=0,001$ ).

**Sonuç:** Post-Travmatik Kognitif Yıkım tanısı multidisipliner klinik muayene, radyolojik görüntüleme ve nörobilişsel testler ile konulabilir.

**Anahtar Kelimeler:** Kognitif yıkım, kafa travması, beyin hasarı, nörokognitif test

## INTRODUCTION

Head trauma and traumatic brain injuries are a serious public health problem, affecting 1.6 million people per year in the USA (1). It is extremely common throughout the world in general and is even referred to as a "silent epidemic" (2). Each year 55 million people are known to suffer traumatic brain injury (TBI) (3). Despite current developments in treatment methods, it is estimated that in the European Union and the USA, 5.3-7.7 million people have one or more disabilities due to the subacute or chronic processes of TBI (4).

An important aspect of TBI is cognitive loss in addition to the neurological motor and sensory outcomes of central nervous system damage after trauma (5).

Impairments in a series of brain executive functions, such as cognitive abilities, processing speed, memory, planning, and concentration have been reported in the survivors of moderate and severe TBI (6).

The identification of head traumas, TBI, and Post-Traumatic Cognitive Impairment (PTCI) in respect of clinical forensic medicine applications are important in many judicial cases, primarily the claim that a disability is due to trauma.

To be able to apply effective and accurate forensic medicine evaluation, these evaluations must be conducted by a multidisciplinary team including neurologists, psychiatrists, radiologists and trained psychologists in addition to clinical forensic medicine specialists. For a diagnosis of TBI, radiological examinations are important together with a detailed anamnesis and physical examination. Moreover, neuropsychometric tests are widely used to determine the severity of cognitive impairments caused by TBI, and to support the diagnosis (7).

The Wechsler Memory Scale (WMS) tests are often used to determine short and long-term memory functions with personal and current information, orientation, simple concentration, and maintaining concentration. In addition, the Stroop, Benton, Bender Gestalt, and Mini Mental State tests are used

for the measurement of planning skills, visual memory, global memory, concentration, calculation, recall, speech, motor functions, perception, and visospatial ability. It is recommended that in addition to these, radiological examinations are used in the determination primarily of neuron loss and intracranial sequela lesions (8).

The aim of this study was to determine the risk factors for PTCI in the acute period after TBI in the light of clinical and radiological findings, to compare lesions on radiological images showing findings of sequelae with the neurological examination and neurocognitive test results, and to thereby contribute to sequelae evaluation studies in clinical forensic medicine.

## MATERIALS and METHODS

A retrospective examination was made of the files of cases referred to our centre between 01.01.2019 and 31.12.2019 for disability evaluation, and who were determined with head trauma on the date of the event. Data were retrieved from the patient files in respect of age, gender, type of trauma, localisation and nature of the lesion at the time of the trauma, examination findings, computed tomography (CT) and magnetic resonance imaging (MRI) findings, and neurocognitive test results.

The neurocognitive tests of WMS, Benton test, Bender-Gestalt test, Stroop test, and the Mini Mental State test were applied.

The WMS subtest-I was applied to test personal and current information, the WMS subtest-II to test time and place orientation, WMS subtest-III for mental co-ordination, WMS subtest-IV for numerical range, WMS subtest-V for logical memory, and WMS subtest-VI for visual memory.

The Stroop test was applied to measure interference resistance ability (inhibiting an inappropriate response) in particular of the executive functions.

The responses to the questions asked in the WMS, Benton, and Mini Mental tests were scored proportionally between 0 (no

correct response) and 1 (all correct). In the Stroop test, the score was based on the time taken to complete the test.

Patients whose symptoms could not be explained by any psychiatric or organic reason and who were thought to be trying to make themselves look bad for secondary gain during the neurological examination and neurocognitive tests, and those with known cognitive loss, such as dementia, before the trauma were excluded from the study.

## RESULTS

The cases evaluated in the study comprised 109 (81.3%) males and 25 (18.7%) females with a mean age of  $39.8 \pm 14.43$  years (median age: 39 years, range: 14-78 years). The causes of the head trauma of the cases referred for the determination of the degree of disability are shown in Figure 1.

In the evaluation of the radiographs (cranial CT etc.) taken at the time of presentation, frontal fracture was determined in 39 (29.1%) cases, temporal fracture in 43 (32.1%), parietal fracture in 26 (19.4%), occipital fracture in 10 (7.5%) and fracture in the bones at the base of the skull in 7 (5.2%).

The intracranial lesions on the date of the trauma were determined as subarachnoid bleeding in 57 (42.5%) cases, subdural bleeding in 34 (25.4%), epidural bleeding in 32 (23.9%), cerebral contusion in 52 (38.8%), intracerebral hematoma in 18 (13.4%), diffuse axonal injury in 7 (5.2%), and widespread brain oedema in 35 (25.4%). The intracranial localisations of these lesions are shown in Figure 2.

### Distribution of the types of trauma

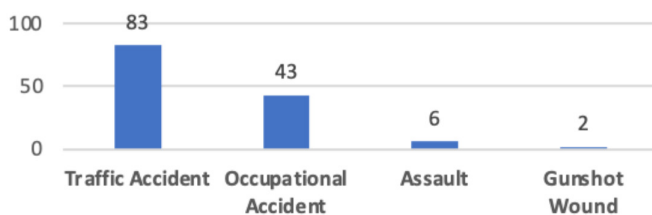


Figure 1. Distribution of the types of trauma

### The intracranial localisations of lesions

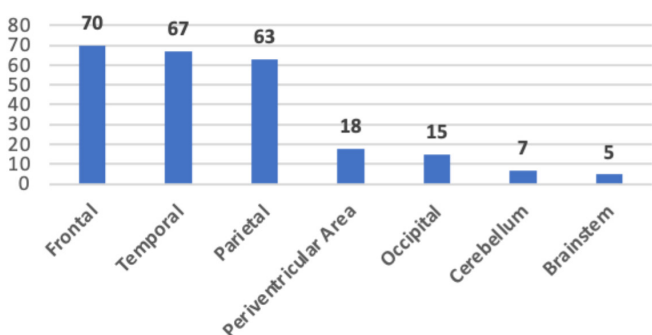


Figure 2. Localisation of the lesions

To evaluate sequelae lesions in the cases, cranial MRIs were taken after completion of treatment at least 18 months after the trauma. The examination of the MRIs showed encephalomalacic/gliotic area in 94 (70.1%) cases, widespread atrophy in 12 (9%), an appearance consistent with lobectomy in 1 (0.7%), and normal cranial appearance in 27 (20.1%).

The localisations of these determined sequelae lesions were seen to be primarily in the frontal and temporal regions; in the frontal area in 66 (49.3%) cases, in the temporal area in 58 (43.3%) cases, the parietal area in 29 (21.6%), the occipital area in 16 (11.9%), the periventricular area in 29 (21.6%), in the cerebellum in 6 (4.5%), and in the brain stem in 1 (0.7%).

In the images shown below of a 14-year-old male, on the cranial CT taken on the date of the trauma, left parietooccipital and right frontotemporal hemorrhagic contusion can be seen (Figure 3). On the cranial MRI of the same case taken after 3,5 years, there can be seen to be left parietooccipital and right frontotemporal gliotic encephalomalacic areas (Figure 4).

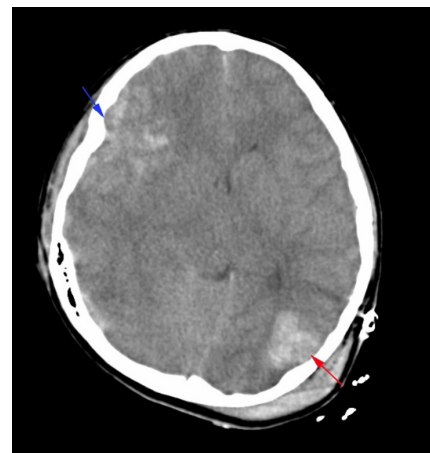


Figure 3. Cranial CT taken on the date of the trauma - Left parietooccipital and right frontotemporal hemorrhagic contusion  
CT: Computed tomography

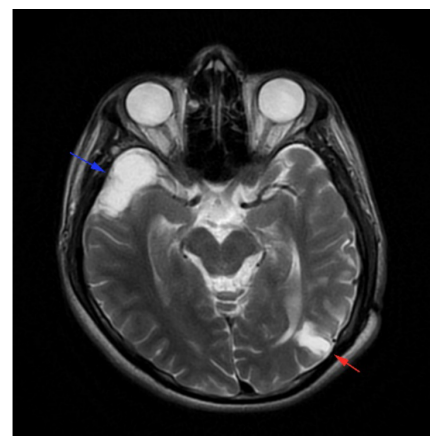


Figure 4. Cranial MRI of the same case taken after 3.5 years - Left parietooccipital and right frontotemporal gliotic encephalomalacic areas

MRI: Magnetic resonance imaging

As a result of the physical examination, imaging, and neurocognitive tests applied to evaluate the disability of the cases, TBI causing evident PTCI was determined in 87 (64.9%) cases. Other sequelae associated with the trauma (craniectomy defect, epilepsy, hearing or sight loss, etc.) were determined in 13 (9.7%) cases. No sequelae due to head trauma were determined in 34 (25.4%) cases.

No significant difference was determined between the PTCI group and the other group in respect of gender ( $p=0.721$ ).

The correlations between diagnosis and the intracranial lesions determined at the time of trauma are shown in Table 1.

A statistically significant correlation was determined between the diagnosis and injuries occurring in the frontal, temporal, and parietal lobes (Table 1).

The correlations between diagnosis and the localisation of the intracranial lesions determined at the time of trauma are shown in Table 2.

Correlations between the diagnosis and neurocognitive test results are shown in Table 3.

The scores in the subtests for personal and current information, and for time and place orientation were found to be significantly low in the PTCI group ( $p=0.001$ ). The test results for logical memory (short and long-term recall spontaneously and with clues) were found to be significantly low in the PTCI group.

## DISCUSSION

TBI is an injury in which impairments are seen in physical, cognitive, and mental-social functions. It is a cause of disability and death at an increasing rate throughout the world (10).

Although primarily traffic accidents and falls, every traumatic event causing head trauma can be in the etiology of TBI.

TBI is manifested with many clinical signs and symptoms such as dizziness, headache, vomiting, loss of memory, and motor deficits (9).

The determination of TBI in the acute period is of vital importance, especially for emergency interventions. The most preferred radiological method in the acute period is still non-contrast CT, which has high sensitivity for the determination of acute bleeding, hydrocephaly, herniation, and fractures (10). MRI is used more for the determination of ischemic conditions and for the evaluation of sequelae lesions in the chronic period.

Although previous studies in literature have reported that parietal fractures are seen most after head trauma in both paediatric and adult patient groups, in a study by Şimşek et al. (11) frontal fractures were seen to be determined at the highest rate. However, almost all studies have shown that the least fractured bone is the occipital bone, because of both the localisation and thickness of this bone. In this respect, the current study results are similar to the data in literature. In the current study cases, temporal fracture was determined in 43 (32.1%), frontal fracture in 39 (29.1%), parietal fracture in 26 (19.4%), occipital fracture in 10 (7.5%) and fracture in the bones at the base of the skull in 7 (5.2%). The distribution of these fractures is affected by the facts that the frontal bone localisation makes it more vulnerable to trauma, and the temporal and parietal bones are thinner than the other bones in the skull.

In addition to fractures, cranial vascular and central nervous system injuries are serious outcomes of head trauma. It has been reported in literature that epidural hematoma is seen in 1-4% of TBI cases, subarachnoid bleeding in approximately 11%, and subdural hematoma in 10-20% of all head trauma cases (12). In the current study, the pathologies seen on cranial CT taken in the acute period were mostly bone fractures, followed by subarachnoid bleeding, cerebral contusion, subdural bleeding, and epidural bleeding. It was thought that this order could change depending on the patient age and the severity of the trauma suffered. Thus, in the elderly, an expanded subdural distance, secondary to atrophy could be a reason for subdural bleeding with even simple trauma causing rupturing of bridging veins. In addition, the current study sample group included individuals referred for disability because of pre-existing neurocognitive complaints causing the complaints following head trauma, and who therefore underwent neurocognitive examination. Therefore, the distribution may show differences from reports in literature.

The correlations between diagnosis and the types of lesions determined at the time of trauma are shown in Table 1. A significant point in this table is that PTCI was determined in 6 of the 7 cases diagnosed with diffuse axonal injury at the time

**Table 1. Correlations between PTCI and the intracranial lesions determined at the time of trauma**

		PTCI (n=87)	Other (n=47)	p-value
Variables	Groups	n	n	
Subarachnoid bleeding	Absent	44	33	0.028
	Present	43	14	
Subdural bleeding	Absent	62	38	0.224
	Present	25	9	
Epidural bleeding	Absent	61	41	0.027
	Present	26	6	
Contusion-laceration	Absent	52	30	0.645
	Present	35	17	
Hematoma	Absent	74	42	0.486
	Present	13	5	
Diffuse axonal injury	Absent	81	46	0.421*
	Present	6	1	
Oedema	Absent	63	37	0.423*
	Present	24	10	

PTCI: Post-Traumatic Cognitive Impairment

of the trauma. Traumatic diffuse axonal injury (DAI) is known to lead to functional and cognitive impairments by cutting the links in the large-scale brain pathways (13). Therefore, DAI determined in the acute period can be considered a serious risk factor for PTCI.

Contusional changes in head trauma are most common in the frontal and temporal lobes (14). In our study; in the cranial magnetic resonance imaging (MRI) taken at least 18 months after the traumatic event, areas with encephalomalacia and/or gliosis were detected most frequently in the frontal and temporal regions in accordance with the literature. Bone fractures, hemorrhages and often diffuse brain damage are reported in head traumas that cause death (15). Neuronal loss in non-fatal head traumas may present with various clinical consequences. Therefore; when investigating neurocognitive function losses, functions of the regions of the central nervous system must be taken into consideration, because dysfunction in the frontal and temporal lobes, which are the regions most vulnerable to injury, is known to have negative effects on learning, executive functions, and memory (16). In our study, a significantly higher incidence of PTCI was diagnosed in cases with sequelae lesions in the frontal, temporal, and parietal lobes (Table 2).

All the cases in the current study group were referred by judicial authorities to our institution for disability evaluation. Therefore, the neurocognitive test results could be different from those of the normal population. As seen in Table 3, the difference was examined between the PTCI group and the group of other head trauma patients. The results of the WMS subtests in respect of current information, time and place orientation, and logical

memory were determined to be significantly lower in the PTCI group than in the group of other head trauma patients. No significant difference was seen between the groups in the mean results of the other WMS subtests or in the visual memory-Benton test, or in the time difference in the Stroop test. Previous studies have reported that the most frequent function disorders following head trauma are problems related to memory and executive functions, and a decrease in information processing speed (17,18). Widespread TBI especially has been shown to be associated with memory, concentration and processing speed impairments (19). Of these deficits, forgetfulness is one of the most frequently reported complaints by the patient and their family (20). This forgetfulness, both as a deficit in learning and recall, has been associated more with long-term than short-term memory (21). It has also been shown that this condition is related to visual memory, whereas verbal and auditory memory are better preserved (22). In the current study, the PTCI group was seen to have significantly low success in the subtests assessing personal and current information, time and place orientation and logical memory. In the logical memory test, the capacity of the subject for both learning and recall are tested in the short and long term. Consistent with findings in literature, the PTCI group obtained statistically significantly low results. All these findings consistent with literature show that the WMS tests can be used in the diagnosis of PTCI. However, when it is considered that patients evaluated in a judicial process with the expectation of secondary gains may demonstrate simulative behaviours, the neurocognitive tests must be confirmed with imaging and examination findings.

**Table 2. Correlations between PTCI and the localisation of the intracranial lesions determined at the time of trauma**

Variables	Groups	PTCI (n=87)	Other (n=47)	p-value
		n	n	
Frontal injury	Absent	36	28	0.044
	Present	51	19	
Temporal injury	Absent	33	34	<0.001
	Present	54	13	
Parietal injury	Absent	40	31	0.027
	Present	47	16	
Occipital injury	Absent	76	43	0.469
	Present	11	4	
Periventricular injury	Absent	77	39	0.371
	Present	10	8	
Cerebellar injury	Absent	84	43	0.240
	Present	3	4	
Brain stem injury	Absent	84	45	1.000
	Present	3	2	

PTCI: Post-Traumatic Cognitive Impairment

Table 3. Correlations between the PTCl and neurocognitive test results			
	PTCl (n=87)	Other (n=47)	p-value
Variables	Mean ± SD	Mean ± SD	
Age (years)	38.21±14.06	42.81±14.78	0.079
Personal -current information	0.72±0.28	0.89±0.18	<b>0.001*</b>
Time and place orientation	0.79±0.28	0.95±0.13	<b>0.001*</b>
Numerical range-forward	4.61±1.04	4.73±1.16	0.497
Numerical range-backwards	3.32±1.18	3.62±0.68	0.078
Mental Co-ordination	0.59±0.31	0.71±0.24	0.079
Logical memory-(short-term, spontaneous response)	0.51±0.24	0.66±0.26	<b>0.003*</b>
Logical memory-(short-term recall with clues)	0.67±0.23	0.82±0.21	<b>0.001*</b>
Logical memory-(long-term, spontaneous response)	0.56±0.3	0.79±0.26	<b>0.001*</b>
Logical memory-(long-term recall with clues)	0.77±0.25	0.9±0.23	<b>0.001*</b>
Benton	0.56±0.21	0.63±0.22	0.079
Stroop test time difference	48.09±20.18	48.91±23.87	0.957

PTCl: Post-Traumatic Cognitive Impairment, \*Significant at 0.05 level; Student's t-test or Mann-Whitney U test, SD: Standard deviation

## CONCLUSION

TBI and PTCl are a serious problem with economic and social dimensions in addition to health problems. The majority of patients who suddenly become physically and socially disabled have to be distanced from community life.

Within the clinical forensic medicine applications in Türkiye, the determination of trauma sequelae of a judicial nature is an important area of work. In this study, by evaluating and comparing the head trauma characteristics, radiographic findings, and neurocognitive test results of cases examined for disability evaluation, it was aimed to determine the most effective methods of determining PTCl and the factors causing this.

As in all trauma, head traumas are preventable causes of morbidity. There is a need throughout the world in general for more preventative measures to be taken, such as reducing workplace accidents, increasing protective measures, developing equipment to prevent head trauma in traffic accidents, etc.

There is a need for further studies of the early stage treatment and rehabilitation of head trauma and for more sensitive and detailed examinations of the long-term effects on the patient.

## Ethics

**Ethics Committee Approval:** This study was reviewed and approved by The Ministry of Justice Council of Forensic Medicine Research and Ethics Committee (10-09/2020-21589509/2020/799).

**Peer-review:** Internally and externally peer-reviewed.

## Authorship Contributions

Concept: H.İ.A., A.F., Design: H.İ.A., A.F., Data Collection or Processing: H.İ.A., E.B., S.Ç.A., Analysis or Interpretation: H.İ.A.,

E.B., S.Ç.A., A.F., Literature Search: H.İ.A., E.B., S.Ç.A., Writing: H.İ.A., E.B., S.Ç.A., A.F.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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## REFERENCES

- Rutland-Brown W, Langlois JA, Thomas KE, Xi Y. Incidence of traumatic brain injury in the United States, 2003. *J Head Trauma Rehabil.* 2006;21(6):544-548. <https://doi.org/10.1097/00001199-200611000-00009>
- Corrigan JD, Selassie AW, Orman JA. The epidemiology of traumatic brain injury. *J Head Trauma Rehabil.* 2010;25:72-80. <https://doi.org/10.1097/HTR.0b013e3181ccc8b4>
- Bruns J Jr, Hauser WA. The epidemiology of traumatic brain injury: a review. *Epilepsia.* 2003;44(Suppl 10):2-10. <https://doi.org/10.1046/j.1528-1157.44.s10.3.x>
- Tagliaferri F, Compagnone C, Korsic M, Servadei F, Kraus J. A systematic review of brain injury epidemiology in Europe. *Acta Neurochir (Wien).* 2006;148(3):255-268. [10.1007/s00701-005-0651-y](https://doi.org/10.1007/s00701-005-0651-y)
- Sinha1 S, Gunawat P, Nehra A, Sharma1 BS. Cognitive, functional, and psychosocial outcome after severe traumatic brain injury: A cross-sectional study at a tertiary care trauma center. *Neurology India.* 2013;61(5):501-506. <https://doi.org/10.4103/0028-3886.121920>
- Dikmen SS, Corrigan JD, Levin HS, Machamer J, Stiers W, Weisskopf MG. Cognitive outcome following traumatic brain injury. *J Head Trauma Rehabil.* 2009;24:430-438. <https://doi.org/10.1097/HTR.0b013e3181c133e9>
- Carlozzi NE, Grech J, Tulskey DS. Memory functioning in individuals with traumatic brain injury: An examination of the Wechsler Memory Scale-Fourth Edition (WMS-IV). *J Clin Exp Neuropsychol.* 2013;35(9):906-914. <https://doi.org/10.1080/13803395.2013.833178>
- Wang ML, Li WB. Cognitive impairment after traumatic brain injury: The role of MRI and possible pathological basis. *J Neurol Sci.* 2016;370:244-250. <https://doi.org/10.1016/j.jns.2016.09.049>
- Harmon KG, Drezner JA, Gammons M, Guskiewicz KM, Halstead M, Herring SA, et al. American Medical Society for Sports Medicine position statement: concussion in sport. *Br J Sports Med.* 2013;47(1):15-26. <https://doi.org/10.1136/bjsports-2012-091941>

10. Greene KA, Marciano FF, Johnson BA, Jacobowitz R, Spetzler RF, Harrington TR. Impact of traumatic subarachnoid hemorrhage on outcome in nonpenetrating head injury. Part I: a proposed computerized tomography grading scale. *J Neurosurg.* 1995;83:445-452. <https://doi.org/10.3171/jns.1995.83.3.0445>
11. Şimşek M, Kaya M, Hiçdönmez T, Süslü HT, Gergin YE. Research of Epidemological and Prognostic Factors on Cranial Fractures Depends Trauma. *Türk Nöroşirürji Dergisi.* 2013;(23)1:12-17. [http://norosirurji.dergisi.org/pdf/pdf\\_TND\\_811.pdf](http://norosirurji.dergisi.org/pdf/pdf_TND_811.pdf)
12. Provenzale J. CT and MR imaging of acute cranial trauma. *Emerg Radiol.* 2007;14:1-12. <https://doi.org/10.1007/s10140-007-0587-z>
13. Sharp DJ, Scott G, Leech, R. Network dysfunction after traumatic brain injury. *Nat Rev Neurol.* 2014;10(3):156-166. <https://doi:10.1038/nrneurol.2014.15>
14. McGinn MJ, Povlishock JT. Pathophysiology of Traumatic Brain Injury. *Neurosurg Clin N Am.* 2016;27(4):397-407. <https://doi: 10.1016/j.nec.2016.06.002>
15. Boto GR, Gómez PA, De La Cruz J, Lobato RD. Severe head injury and the risk of early death. *J Neurol Neurosurg Psychiatry.* 2006;77(9):1054-1059. doi: <https://10.1136/jnnp.2005.087056>
16. Wilde EA, Hunter JV, Newsome MR, Scheibel RS, Bigler ED, Johnson JL, et al. Frontal and temporal morphometric findings on MRI in children after moderate to severe traumatic brain injury. *J Neurotrauma.* 2005;22(3):333-344. <https://doi:10.1089/neu.2005.22.333>
17. Millis SR, Rosenthal M, Novack TA, Sherer M, Nick TG, Kreutzer JS, et al. Long-term neuropsychological outcome after traumatic brain injury. *J Head Trauma Rehabil.* 2001;16(4):343-355. doi:10.1097/00001199-200108000-00005
18. Novack TA, Alderson AL, Bush BA, Meythaler JM, Canupp K. Cognitive and functional recovery at 6 and 12 months post-TBI. *Brain Inj.* 2000;14(11):987-996. <https://doi.org/10.1080/02699050050191922>
19. Lezak MD, Howieson DB, Bigler ED, Tranel D. *Neuropsychological assessment.* New York, NY: Oxford University Press; 2012.
20. Arcia E, Gualtieri CT. Association between patient report of symptoms after mild head injury and neurobehavioural performance. *Brain Inj.* 1993;7(6):481-489. <https://doi.org/10.3109/02699059309008175>
21. Vakil E. The effect of moderate to severe traumatic brain injury (TBI) on different aspects of memory: a selective review. *J Clin Exp Neuropsychol.* 2005;27(8):977-1021. <https://doi.org/10.1080/13803390490919245>
22. West LK, Curtis KL, Greve KW, Bianchini KJ. Memory in traumatic brain injury: the effects of injury severity and effort on the Wechsler Memory Scale-III. *J Neuropsychol.* 2011;5(Pt 1):114-125. <https://doi.org/10.1348/174866410X521434>