

The Results of Renal and Liver Function Tests in Patients with Head Trauma

Mustafa Abdulkareem Aljaberi^{1*} and Abdullah H Abdullah

Department of Chemistry and Biochemistry, Saad alwatry Hospitel, Iraq

*Corresponding author: Mustafa Abdulkareem Aljaberi, Department of Chemistry and Biochemistry, Saad alwatry Hospitel, Iraq; E-mail: dr.mustafa.kareem24@gmail.com

Received: February 18, 2022, Manuscript No. IPJSEM-22-11528; **Editor assigned:** February 21, 2022, PreQC No. IPJSEM-22-11528 (PQ); **Reviewed:** March 07, 2022, QC No. IPJSEM-22-11528; **Revised:** March 11, 2022, Manuscript No. IPJSEM-22-11528 (R); **Published:** March 18, 2022, Invoice No. IPJSEM-22-11528

Citation: Aljaberi MA, Abdullah AH (2022) The Results of Renal and Liver Function Tests in Patients with Head Trauma. J Surgery Emerg Med Vol:7 No:1

Abstract

Traumatic brain injury is one of the most commonly diagnosed illnesses, frequently resulting in lifelong impairment. Brain injuries cause damage to brain tissue, which contributes to further harm. It is treated as a complicated distinct disorder.

Keywords: Traumatic; Mitochondrial damage; Intracranial pressure; Bacteria diseases; Traumatic Brain Injury

Introduction

Traumatic brain injury is one of the most commonly diagnosed illnesses, frequently resulting in lifetime impairment, and brain injuries cause damage to brain tissue, contributing to recurrent harm. It is treated as a complicated distinct disorder [1].

One of the most prevalent causes of traumatic brain injuries is a fall; it has been recognized as one of the most serious risk factors for increasing mortality and morbidity in the population. The second most frequent reason for this situation is vehicle accidents and suicide, and these causes vary by area, as do the ages impacted in terms of degrees, with the middle ages being one of the most common ages for brain damage [2]. We must acknowledge that drinking is one of the leading causes of traffic accidents that result in serious injury or death [3].

Learning about the origins of brain damage disease helps us determine the reasons and how to prevent it, allowing it to be classified into two types based on the injury.

A direct head injury creates structural changes as a result of the force that produced it, and this defines the degree of the

injury, which causes brain tissue death and damage to the blood arteries that supply brain cells...

Secondary injury can develop as a result of brain illness, causing damage to brain tissue. One of the most significant causes of secondary injury due to primary injury is free radicals and ion transport defects, as well as an imbalance of blood carriers and an imbalance in the energy that enters the brain as a result of mitochondrial damage and inflammatory factors that occur as a result of brain damage caused by bacteria diseases, and a lack of oxygen to the brain in low blood pressure and high intracranial pressure.

Secondary injury can develop as a result of brain illness, causing damage to brain tissue. One of the most significant causes of secondary injury due to primary injury is free radicals and ion transport defects, as well as an imbalance of blood carriers and an imbalance in the energy that enters the brain as a result of mitochondrial damage and inflammatory factors that occur as a result of brain damage caused by bacteria diseases, and a lack of oxygen to the brain in low blood pressure and high intracranial pressure [4].

The assessment process severity of traumatic brain damage dependent on Glasgow Coma Scale (GCS) is usually measured by a clinician to determine the severity of TBI.

The main TBI severity measure don't generally reflect the quantity of the condition resulting from TBI.

TBI severity was divided into three categories. (GCS: 13–15) TBI (GCS: 9–12) and TBI (GCS: 3–8).

Brain damage GCS 13–15 most cranial trauma falls into this group. Patients are alert, bewildered, and able to communicate [4].

To establish a statistical link between early response and outcome, the coma values were summed to provide a score ranging from 3 to 14 [5].

Table 1: Scores on the Glasgow Coma Scale.

Opening the eyes	1.Unplanned. Shows arousal as opposed to awareness.. 2.to speak When spoken to—not always to open the eyes 3.To inflict pain on When used on the limbs instead of the face, grimacing may cause a closure...
------------------	--

	4.No exist...
Motor reactivity	<ol style="list-style-type: none"> 1.Follows orders. Reflex and postural alterations excluded. 2.It is localized. The other limb shifts to the location of the pressure on the nail bed. 3.Resigns. An elbow or knee bending reaction to local painful stimuli that is normal 4.Prolonged flexion. Withdraw slowly while pronating the wrists and adducting the shoulders 5.Extensor reaction. Elbow extension with pronation and adduction 6.There isn't even a shudder.
Responses verbal	<ol style="list-style-type: none"> 1.Orientated. Understands who, where, and when; the year, season, and month 2.Confusion in the discussion. Attends and replies, although responses are muddled/incorrect 3.Words that should not be used in this situation. Some intelligent words, but they're all expletives or just plain weird. 4.Speech that is incomprehensible. Only moans and groans 5.there are no words to describe it

ALT (Alanine Aminotransferase) is produced by the liver and is increased in the presence of cell liver damage, cell death, or inflammation. AST (Aspartate Aminotransferase) is produced by the liver and is also produced by muscle and is increased in the presence of liver inflammation and heart attack. Because the ALT and AST levels increase in many cases of liver inflammation, this test provides a more comprehensive picture of the liver disease [6]. One of the more common laboratory abnormalities following TBI is mildly elevated liver enzymes. while the liver enzyme testing is often useful for monitoring individuals with known liver diseases and the clinical significance and specificity of abnormal values are not always clear in the general population [7].

Inflammatory cytokines in CKD-related brain dysfunction and Oxidative stress has been associated with both brain and kidney dysfunctions a significant increase of nitro tyrosine a reactive and cytotoxic product generated by the interaction of Nitric Oxide (NO) and Reactive Oxygen Species (ROS) [8].

The urea cycle disorders are a group of rare congenital disorders caused by a lack of the enzymes or transport proteins needed to remove ammonia from the body through a series of biochemical steps in which nitrogen, a waste product of protein metabolism, is removed from the blood and converted into urea.

A consequence of these disorders is hyperammonaemia resulting in central nervous system dysfunction with mental status changes and brain edema and seizures, coma, and potentially death [9].

Creatinine was one of substance result from organ metabolism which through glomerulus filtration process while

tubules secretion was very minimal so therefore creatinine was very useful for glomerulus evaluation [10].

A Case Control Study was used to show the feasibility of utilizing this enzyme in patients with traumatic brain injury and healthy individuals to evaluate their recovery from traumatic brain injury.

Methods

The research was carried out between May 2021 and September 2021. Baghdad's Medical City Hospital and Neurosurgical Hospital provided the serum for this study. There were 45 traumatic brain injury patients (both sexes) and 45 healthy people in this research.

Criteria for exclusion

*The following were the criteria used to weed people out of the study:

- Psychiatric patients.
- Seizures patients
- Infection of the CNS.
- The process of gathering samples is described below.
- Each patient and control participant had seven milliliters of blood drawn.
- To analyze the serum, it will be split into tiny aliquots and centrifuged at 2000 rpm for 15 minutes after blood clotting.
- RFT and LFT at their most basic levels.

Table 2: The measurement marker was varied depending on the material.

Biochemical kits	Supplied Company
Liver Function Test (ALT and AST)	Analyticon
Renal Function Test (B. Urea and Creatinine)	Analyticon

Result

The Study Population's Demographic Characteristics

Table 3: Shows the variation between TBI and Controls of P-value.

Variables.	TBI (n=45)	Controls (n=45) are used.	p-value
Age, years			0.113
± SD	40.37 ± 16.52	35.0 ± 13.31	
Range	19-80	19-70	
Gender			
Male	26 (62.5%)	16 (40%)	0.025
Female	14 (37.5%)	24 (60%)	

Paraclinical Study Population Characteristics

The kidney marker function (urea and creatinine) were severely impacted. (7.1 ± 3.23 mmol/L and 104.06 ± 44.4 mmol/L, respectively) compared with controls (4.85 ± 2.33

mmol/L and 74.93 ± 19.25 mmol/L, respectively) with highly significant differences. Likewise, liver function tests (ALT and AST) were remarkably higher in patients (40.92 ± 18.68 U/L and 44.13 ± 24.83 U/L, respectively) than controls (23.0 ± 10.45 U/L and 28.7 ± 10.58 U/L, respectively).

Table 4: Highly significant difference in table 3-1.

Variables	Range in patients	TBI (n=40)	Controls (n=40)	Range in control	p-value
Urea, mmol/L	1.3-16	7.1 ± 3.23	4.85 ± 2.33	1.9-12.3	0.001
Creatinine, mmol/L	37.4-302	104.06 ± 44.4	74.93 ± 19.25	44-125	<0.001
ALT, U/L	10.1-76.1	40.92 ± 18.68	23.0 ± 10.45	9.0-51	<0.001
AST, U/L	8.3-95.4	44.13 ± 24.83	28.7 ± 10.58	15-55	0.001

Table 5: Association of different variables with gender in TBI patients in table 3.2.

Variables	Males(n=26)	Females (n=14)	P-value
GCS			
Mild (14-15)	5 (19.23%)	5 (35.71%)	0.463
Moderate(10-12)	9 (34.62%)	3 (21.43%)	
Severe(4-8)	12 (46.15%)	6 (42.86%)	
Age, years	39.27 ± 17.92	42.43 ± 13.94	0.571

Urea, mmol/L	7.59 ± 3.39	6.19 ± 2.79	0.192
Creatinine, mmol/L	116.6 ± 46.98	92.18 ± 35.25	0.097
ALT, U/L	40.6 ± 20.92	41.51 ± 14.3	0.884
AST, U/L	47.93 ± 27.93	37.06 ± 16.31	0.19

Table 6: Shows the relationship between several factors and GCS score in TBI patients table 3-3.

Variables	Mild	Moderate	Severe	P-value
Age, years	36.3 ± 18.95	41.17 ± 17.17	42.11 ± 15.23	0.67
Urea, mmol/L	7.69 ± 2.77	6.71 ± 2.76	7.03 ± 3.82	0.781
Creatinine, mmol/L	117.5 ± 23.0	101.97 ± 35.92	106.87 ± 57.62	0.718
ALT, U/L	40.11 ± 17.58	47.65 ± 19.31	36.88 ± 18.61	0.306
AST, U/L	37.36 ± 19.16	47.63 ± 22.74	45.56 ± 29.1	0.606

Different small letters indicate significant differences.

Discussion

Traumatic brain injury syndrome is a serious illness that may afflict people from all walks of life and cultures. The reason of this injury and the rise in the number of reported cases have been attributed to collisions and cases of falling from great heights, which have been reported in significant numbers in Iraq, most notably in motorcycle accidents and road accidents..

The High levels of traffic and a lack of understanding of traffic rules and, most importantly, safety precautions, as well as alcoholism, are all significant risk factors for severe TBI after it was discovered that the suggested mechanism for more serious injury is a combination of altered censorship and brain atrophy, as well as enzyme inhibition of TBI effects on neurons when ethanol is present. Falling from a great height is a common cause of Traumatic Brain Injury (TBI), especially in children and women. In addition to a lack of knowledge and insecurity, Traumatic Brain Injury (TBI) is prevalent in our nation as a result of the relatively simple availability of firearms. Our investigation has shown that the weapons may be blunt or sharp objects. That age appears to be another important factor that has a significant impact on morbidity and mortality rates, and that head injuries do not appear to be restricted to a specific age group, which is consistent with previous research that has found that age has a significant impact on mortality rates.

The kidney function test is performed: The results of the previous research showed that patients' kidney function tests (urea and creatinine) were significantly worse than controls' (7.13.23 mmol/L and 104.0644.4 mmol/L, respectively) as compared to controls (4.852.33 mmol/L and 74.9319.25 mmol/L, respectively). Behavioral and cognitive changes, as well

as neurotransmitter changes, are the most common neurological consequences of urea cycle defects. This is related to the consequences of hyperammonaemia, which can lead to behavioral and cognitive changes, neurotransmitter changes, and presumed energy failure [9]. To avoid AKI and, in particular, to reduce the risk of contrast-induced nephropathy, it is critical to maintain sufficient fluid intake and output (hydration). In addition, multiple contrast exposure should be reduced by using alternative imaging methods if feasible, and nephrotoxic medications should be avoided whenever possible to prevent causing kidney damage. The removal of azotemia retention products and the reduction of toxic drug levels are two ways in which renal replacement therapy can aid in the improvement of encephalopathy due to uremia. However, a rapid fall in serum urea may paradoxically cause cerebral edema, and this is something that many experts agree on [11].

In patients with traumatic brain injury, liver function is assessed.

Liver function tests (ALT and AST) were significantly higher in patients (40.9218.68 U/L and 44.1324.83 U/L, respectively) than in controls (23.010.45 U/L and 28.710.58 U/L, respectively), with a highly significant difference in TBI patients. TBI patients have a higher incidence of elevated serum liver enzyme levels, and these tests have been associated with liver dysfunction in previous studies [12-15]. TBI is also linked with a continuing neuro-inflammatory and systemic inflammatory response, with increased serum levels of inflammatory cytokines participating in 'organ crosstalk,' which may result in hepatocellular damage as a consequence of the inflammatory response. The abnormalities of ALT and ALP did not recover to baseline normal levels before patients were discharged from critical care [16-18].

Conclusion

Diabetes and liver enzyme (LFT) concentrations, as well as Renal Function (RFT) and electrolyte levels, are elevated in individuals who have had traumatic brain injury.

Following therapy for different types of brain damage, changes in enzyme levels are seen (fall, bullet).

References

1. Thelin E, Nimer FA, Frostell A, Zetterberg H, Blennow K, et al. (2019) A serum protein biomarker panel improves outcome prediction in human traumatic brain injury. *J Neurotrauma* 36:2850-2862
2. Haddad, Samir H, Yaseen M Arabi (2012) Critical care management of severe traumatic brain injury in adults. *Scand. J Trauma Resusc Emerg Med* 20:12
3. Sundstrom T, Grände PO, Luoto T, Rosenlund C, Undén J, et al. (2012) Management of severe traumatic brain injury: evidence tricks and pitfalls. *Springer Sci Bus Media* 4:150
4. Zollman, Felise S (2016) Manual of traumatic brain injury: Assessment and management. Springer Publishing Company
5. Jennett, Bryan (2005) Development of Glasgow coma and outcome scales. *Nepal J Neurosci* 2:24-28
6. Kunutsor SK, Apekey TA, Seddoh D, Walley J (2014) Liver enzymes and risk of all-cause mortality in general populations: a systematic review and meta-analysis. *Int J Epidemiol* 43:187-201
7. Fox A, Sanderlin BJ, McNamee S, Bajaj SJ, Carne W, et al. (2014) Elevated liver enzymes following poly traumatic injury. *J Rehabil Res Dev* 51:869
8. Simoes Silva, Ana Cristina, Miranda AS, Rocha NP, Teixeira AL, et al. (2019) Neuropsychiatric disorders in chronic kidney disease. *Front Pharmacol* 10:932
9. Gropman AL, M Summar, JV Leonard (2007) Neurological implications of urea cycle disorders. *J Inherit Metab Dis* 30:865-879
10. Sari, Erni A, Suharjono Suharjono, Joni Wahyu hadi (2020) Monitoring Serum Creatinine, Blood Urea Nitrogen in Patients Brain Injury with Mannitol Therapy. *Folia Medica Indonesiana* 56:254-260
11. Kulkarni, Dilip K (2016) "Brain injury and the kidney." *J neuroanaesth crit care* 3:16-19
12. Sanfilippo F, Veenith T, Santonocito C, Vrettou CS, Matta BF, et al. (2014) "Liver function test abnormalities after traumatic brain injury: is hepato-biliary ultrasound a sensitive diagnostic tool?." *Br J Anaesth* 112:298-303
13. Idowu, Olufemi Emmanuel, John O Obafunwa, Sunday O Soyemi (2017) "Pituitary gland trauma in fatal nonsurgical closed traumatic brain injury." *Brain Inj* 31:359-362
14. Villapol, Sonia (2016) "Consequences of hepatic damage after traumatic brain injury: current outlook and potential therapeutic targets." *Neural Regen Res* 11: 226
15. Salim A, Brown C, Inaba K, Martin MJ (2018) *Surgical Critical Care Therapy: A Clinically Oriented Practical Approach*. Springer.
16. Ookuma T, Miyasho K, Kashitani N, Beika N, Ishibashi N, et al. (2015) "The clinical relevance of plasma potassium abnormalities on admission in trauma patients: a retrospective observational study." *J Intensive Care* 3: 1-7
17. Freeman, William D, Hani M Wadei (2015) "A brain-kidney connection: the delicate interplay of brain and kidney physiology." *22:173-175*
18. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, et al. (2017) "Traumatic brain injury: current treatment strategies and future endeavors." *Cell transplantation* 26:1118-1130