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Evaluation of the Possible Role of S100B, Troponin I and C- Reactive Protein Levels as Prognostic Markers in Cases of Traumatic Intra-Cerebral and Subarachnoid Hemorrhage

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Abstract Introduction; Traumatic head injury is a major cause of morbidity and mortality across the globe. Even in minor head injuries, long term neuropsychological deficits may occur. The assessment of certain biochemical markers in serum can help to predict outcomes. S100B is a protein biomarker that reflects CNS injury. Cardiac troponin I (cTnI) is a specific marker of myocardial injury. C-reactive protein (CRP) is an acute-phase protein that increases rapidly in response to infection, trauma, and other inflammatory conditions. The present study aimed to evaluate the possible role of S100B, troponin I and C- reactive protein levels as prognostic markers in cases of traumatic intra-cerebral and subarachnoid hemorrhage.

Subjects and method; This study was conducted on 40 patients of both sexes; 20 patients of traumatic intracerebral hemorrhage (ICH) and 20 patients of traumatic subarachnoid hemorrhages (SAH) within 24 hours after traumatic hemorrhage. Glasgow Coma Scale (GCS) was evaluated on admission, within 24 of trauma, for all patients. Brain CT scan was also performed for all patients and Fisher grade was assessed for SAH patients. A daily electrocardiogram was performed. Serum samples were taken from all patients at day one and day three. All samples were analyzed for S100B, cardiac troponin I and C-reactive protein. Glasgow Outcome Scale (GOS) obtained at time of death or after three months

Results; serum S100B, cTnI and CRP levels were negatively correlated with the severity of GCS and GOS among patients with ICH or SAH, except the level of cTnI level on third day among patients with ICH. Furthermore, patients with unfavorable outcome had significantly higher serum S100B, cTnI and CRP levels than favorable outcome. The highest correlation of all biomarkers, in both ICH and SAH, was noticed between S100B level on third day and GOS.

Conclusion; S100bB levels on third day showed a highest correlation with GOS in patients with ICH or SAH.

Keywords ICH, SAH, S100B, troponin I, CRP

Introduction

Traumatic head injury is a major cause of morbidity and mortality across the globe at all ages. Even in minor head injuries with a good outcome, long term neuropsychological deficits may occur (Teasdale et al., 1998).

Accurate prediction of long term outcome and neurological assessment can help clinically, allow early rehabilitation and assess medico legal responsibility of health care providers. An accurate evaluation of the severity of CNS injury can help to predict outcomes and rationally help to decide when the application of aggressive therapeutic interventions would be appropriate (Bloomfield et al., 2007).

Standard methods to prognosticate the severity of initial brain injury and anticipate the onset of secondary injury have included the neurological examination, neuroimaging studies, intracranial pressure monitors, electro diagnostic testing, and transcranial dopplers. The standard tests have limited reliability in patients who are frequently given sedatives, analgesics, and muscle relaxants, or are stable enough to leave the ICU for frequent neuroimaging studies (Signorini et al., 1999).

Therefore, over the past 50 years, intensivists have searched for biological markers that can reliably reflect the severity of injury to predict outcomes or are sensitive enough to detect the early onset of secondary injury. The difficulties associated with the use of CSF markers have led investigators to search for an ideal serum marker that might be highly specific for brain injury, sensitive to minor injuries, appears rapidly in the serum and is easy to be measured with lab tests whose results could be available in a very short time (Ikeda et al., 2001).

S100B is a renal excreted protein concentrated in glial cells of the nervous system, it is called so because of its solubility in 100% saturated ammonium sulfate at neutral pH. S100B is a marker of the primary injury that causes disruption of the blood-brain barrier through which brain-specific markers are released into the blood stream (Delgado et al., 2006; Bloomfield et al., 2007).

On the other hand, serum troponin (Tn) is a sensitive and specific marker of myocardial injury. Elevated Tn levels after injury is usually attributed to mechanical chest trauma, but this relationship remains unproven (Martin etal., 2005).

It has been suggested that elevated cardiac troponin I (cTnI) level is a marker of increased risk of mortality in acute ischemic stroke. However, serum cTnI level has been sparsely investigated as a marker in the prognosis of traumatic intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH) (Angelantonio et al., 2005).

Moreover, C-reactive protein (CRP) is an acute-phase protein, that's blood level increases rapidly in response to infection, trauma, and other inflammatory conditions. Most laboratories have a suitable equipment to measure the level of CRP. The cost for CRP measurement is relatively cheap. The relation of CRP levels to traumatic brain injury (TBI) is not well investigated (Bengzon et al., 2003).

Therefore, the present study aimed to evaluate the possible role of S100B, troponin I and C- reactive protein levels as prognostic markers in cases of traumatic intra-cerebral and subarachnoid hemorrhage.

Subjects and methods

This prospective study was conducted on 40 patients of both sexes; 20 patients of traumatic intracerebral hemorrhage (ICH) and 20 patients of traumatic subarachnoid hemorrhage (SAH) who were admitted to Critical Care Medicine department at the Alexandria Main University Hospital within 24 hours after traumatic hemorrhage.

Approval for the study was obtained from the local Research Ethics Committee of Alexandria Main University Hospital. An informed consent was obtained from the patient or a family member.

Glasgow Coma Scale (GCS) was evaluated on admission, within 24 hours of trauma, for all patients (Mcnett, 2007). Brain CT scan was also performed for all patients and Fisher grade was assessed for SAH patients (Smith et al., 2005). A daily electrocardiogram was performed. Serum samples were taken from all patients at day one and day three.

Exclusion criteria

Patients with any cause of cardiovascular injury, thoracic trauma or history of cardiovascular diseases were excluded. Patients with vascular-related neurological coma, history of neurological, psychiatric disorders or alcohol or drug dependency were excluded from the study as well. Cases of elevated Creatine Kinase were not included to avoid the influence of skeletal muscle trauma.

Glasgow Coma Scale (GCS) (Mcnett, 2007)

Glasgow Coma Scale (GCS) is a standard neurological scale used to describe the conscious state of a patient; scores range from 3 to 15. A GCS score of 13 or greater correlates with mild brain injury; 9 to 12, with moderate brain injury; and 8 or less, with severe brain injury.

Fisher grading (Smith et al., 2005)

Fisher grading indicates the degree of blood evident on computed tomography scans: 0 = no scan done; 1=no blood detected; 2 = diffuse or vertical layers of blood 1 mm or thicker; 3 = localized clot and/or vertical layers of blood 1 mm or thicker; and 4 = diffuse or no SAH but intraventricular or intraparenchymal clot.

Serum samples

Peripheral blood samples were collected on first day of admission and at day 3. They were centrifuged for 10 minutes and serum obtained was stored until analysis. All samples were analyzed for S100B, cardiac troponin I and C-reactive protein.

S100 B protein

Serum concentration of protein S100B was measured by enzyme linked immunoassay by Biobryt. Laboratory reference range defines a normal S-100B concentration ranges from 0.02- 0.1 μg/L (Biberthaler et al., 2002)

Cardiac troponin I (cTnI)

Measurement of serum cTnI was done by (IMMULITE cTnI assay, Diagnostic Product Corporation, Los Angeles, CA). TnI level was categorized as normal (0 –1.2 μg/L), intermediate (1.3–5 μg/L), or high (>5 μg/L) (Martin et al., 2005).

C-reactive protein (CRP)

C-reactive protein was measured by latex-enhanced turbidimetric immunoassay (Roche Diagnostics--Integra 700). CRP less than 3 mg/L was considered normal (Ridker, 2003)

Glasgow Outcome Scale (GOS) (Jennett, 2005)

Glasgow Outcome Scale (GOS) obtained at time of death or after three months. Patients were contacted by the phone or visits at 3 months after injury. Some questions were asked for neuropsychatric disorders such as; amnesia, personality changes, depression and somatic disorder such as; headache, dizziness, seizure, sleep disturbance or post traumatic epilepsy.

Glasgow Outcome Scale (GOS) was categorized into:

- I: Death
- II: Persistent Vegetative State
- III: Severe Disability This applies to a conscious patient who is dependent for daily support from another person by reason of mental or physical disability, usually a combination of both.
- IV: Moderate Disability: These patients have some disability such as dysphasia, hemiparesis or epilepsy and/or deficits of memory or personality but are able to look after themselves. They may be able to work when special arrangements are made.
- V: Good Recovery: This implies a resumption of normal life with the capacity to work even if pre-injury status has not been achieved. Some of these patients have neurological or psychological deficits.

Data was analyzed using statistical package for social sciences (SPSS) version 18 for calculation of Arithmetic mean and standard deviation, median, Kruskal Wallis test and Mann Whitney test. Spearman coefficient was used to assess the degree of correlation between different variables as indicated (Field, 2005).

Results

Forty patients with traumatic head injuries of both sexes were included in this study; 20 patients of traumatic intracerebral hemorrhage (ICH) and 20 patients of traumatic subarachnoid hemorrhage (SAH) who were admitted to Critical Care Medicine department at the Alexandria Main University Hospital within 24 hours after trauma.

Intracerebral hemorrhage

Of the 20 patients suffering from traumatic ICH, 70% were males and 30% were females. Their ages ranged from 17 to 46 years with a mean age of 31.60±10.40 years.

CT findings: Patients with ICH was diagnosed according CT findings (Figure 1).

Clinical Findings and Outcome assessment

GOS was assigned to all patients on discharge or after three months and was divided according to the score into favorable outcome (scores IV and V) and unfavorable outcome (scores I, II and III) (Leitgeb et al., 2013).

Eight patients (40%) were classified as having mild head injuries, with a GCS of 13-15, all of them had a favorable outcome. Six patients (30 %) had a moderate GCS (score 9-12), 50 % of them had a favorable outcome. Cases with a GCS score of 3-8 constituted 30% (n=6) of all cases, one case died before third day. All cases with severe GCS had unfavorable outcome. (Table 1)

A significant correlation was found between the initial GCS score and GOS ($r= 0.883$, at $p\leq 0.001$).

S-100 B values and outcome

On the first day

On the first day, 50% of cases (four patients) of mild head injury had elevated S100 B, (reference range is from 0.02- 0.1 µg/L), while all cases of moderate and severe head injured patients had elevated S100B. A significant relation was observed between S100B level and GCS where P=0.001 (Table 2)

On dividing cases according to GOS into unfavorable and favorable outcome groups, the present data revealed a significant difference of S100 B level between both groups (1.66±0.79 versus 0.13±0.06) with higher levels in the unfavorable group, where $p<0.001$(table 3).

A significant negative correlation was found between the S-100 B level and each of GCS and GOS on the first day (where r = 0.949 and 0.900 at $p< 0.001$, respectively) (table 4 &5).

On the third day

37.5% of cases (three patients) of mild head injury showed elevated S100 B on the third day, while 83.3% of cases (five patients) of moderate and all severe head injured patients had elevated S100B. The highest level was observed in severe injury (1.62 ± 1.04) with a significant relation between S100B level and GCS where P=0.005 (table 2).

The present data revealed a significant difference of S100 B level on the third day between both unfavorable and favorable outcome groups (1.68±1.03 versus 0.11±0.07) with higher levels in the unfavorable group, where $p<0.001$ (table 3).

A significant negative correlation was found between the S-100 B level and each of GCS and GOS on the third day (where r = 0.894 and 0.964, respectively) (table 4 &5).

Troponin I values and outcome

15% of patients (three cases) with ICH presented with elevated cTnI. These patients showed ST-segment changes and T-wave abnormalities.

On the first day

On the first day, no cases of mild or moderate head injury had elevated troponin I, (reference range is from 0-1.2 μg/L), while 16.7% of cases (one case) of severe head injuries had elevated levels. However, the mean levels of troponin were within reference range (table 2).

The present data revealed a significant difference of troponin I level on the first day between both unfavorable and favorable outcome groups (0.70 ±0.96 versus 0.096±0.11) with higher levels in the unfavorable group, where p=0.006 (table 3).

A significant negative correlation was found between the troponin I level and each of GCS and GOS on the first day (where r =0.632 at p= 0.004 and 0.533 at p= 0.019, respectively) (tables 4 & 5).

On the third day

No cases of mild head injury had elevated troponin I on the third day, while one case with moderate and another one with severe head injuries had elevated levels but no significant relation was observed between cTnI level and GCS (table 2).

However, the present data revealed a significant difference of troponin I level on the third day between both unfavorable and favorable outcome groups (0.90 ±0.66 versus 0.035±0.37) with higher levels in the unfavorable group, where p=0.006 (table 3).

Non-significant correlation was found between the cTnI level and GCS (where r =0.430 at p= 0.075) and insignificant correlation was also noticed between cTnI level with GOS, where p= 0.084 (table 4 & 5).

CRP values and outcome

On the first day

All cases of mild, moderate and severe head injury had elevated CRP level, (reference range is less than 3mg/L). On the first day, the highest level was noticed in severe cases. A significant relation was observed between CRP and GCS where P= 0.001 (table 2).

The present data also revealed a significant difference of CRP level on the first day between both unfavorable and favorable outcome groups (76.44 ±23.22 versus 21.73 ±8.99) with higher levels in the unfavorable group, where p<0.001 (table 3).

A significant negative correlation was found between the CRP level and each of GCS and GOS on the first day (where r =0.890 and 0.837 at p< 0.001, respectively) (tables 4 &5).

On the third day

All cases of mild, moderate and severe head injury had elevated CRP level on the third day as well (table 2).

The present data revealed a significant difference of CRP level on the third day between unfavorable and favorable outcome groups (145.13 ±56.56 versus 31.27 ±11.0) with higher levels in the unfavorable group, where p <0.001 (table 3).

A significant negative correlation was found between the CRP level and each of GCS and GOS on the third day (where r =0.895 and 0.882 at p< 0.001 respectively) (table 4 & 5).

The highest correlation was observed between GOS and S100B level at day three followed by S100B level at day one as denoted in table 5.

Subarachnoid haemorrhage

Of the 20 patients suffering from traumatic SAH, 75% (15 patients) were males and 25% (five patients) were females. Their ages ranged from 17 to 47 years with a mean of 33.55±10.31 years.

Clinical Findings and Outcome assessment

Seven patients (35%) were classified as having mild head injuries, with a GCS of 13-15, all of them had a favorable outcome. Seven patients (35 %) had a moderate GCS (score 9-12), 57% of them (four patients) had a favorable outcome. Cases with a GCS score of 3-8 constituted 30% (n=6) of all cases. Two cases of severe brain injury died before the third day. All severe cases had unfavorable outcome. (Table 6)

A significant correlation was found between the initial GCS score and GOS (r= 0.913, at p≤ 0.001).

CT findings and outcome

All mild cases of head injuries had a fisher grade of II, while 71.4% of moderate head injuries were of grade II and the rest were of grade III. Regarding the severely head injured patients, 66.7% were of grade IV and the rest were of grade III. A significant relation was noticed between GCS and Fisher grade where P<0.001 (table 7 & figure 2).

The current results demonstrated a significant difference in Fisher grading between unfavorable and favorable outcome groups where p =0.001 (table 8).

Furthermore, a significant negative correlation was found between these CT findings and each of GCS and GOS (tables 9 & 10).

S-100 B values and outcome

On the first day

Four patients (57.1% of cases) of mild head injury showed elevated S100 B on the first day, while all cases of moderate and severe head injured patients had elevated S100B.The highest S100 B level was noticed in severe cases (1.20 ± 0.59) with significant relation between GCS and S100B level where p= 0.002 (table 11).

Table 12 shows a significant difference of S-100B level on the first day between both unfavorable and favorable outcome groups (0.92 ±0.64 versus 0.17

±0.12) with higher levels in the unfavorable group, where p =0.002 .

Significant negative correlation was found between the S100B level and each of GCS and GOS on the first day (where r =0.832and 0.758, respectively) (tables 9 & 10).

On the third day

42.9% of cases (three patients) of mild head injury had elevated S100 B on the third day, while all cases of moderate and severe head injured patients had elevated S100B. Again, the highest level was among severe cases with significant relation between GCS and S100B levels (table 11).

The present results demonstrated a significant difference of S100B level on the third day between both unfavorable and favorable groups (0.79 ±0.45 versus 0.11 ±0.07) with higher levels in the unfavorable group, where p≤ 0.001 (table 12).

A significant negative correlation was found between the S100B level and each of GCS and GOS on the third day (where r =0.881 and 0.955 at p≤ 0.001, respectively) (tables 9 & 10).

Troponin I values and outcome

10% of patients (two cases) with SAH presented with elevated cTnI. These patients also showed ST-segment elevation and T-wave abnormalities.

On the first day

No cases of head injury had elevated troponin I on the first day. Yet, a significant relation was observed between cTnI level and GCS (table 11).

The current data demonstrated a significant difference of troponin I level on the first day between both unfavorable and unfavorable groups (0.61 ±0.30 versus 0.15 ±0.21) with higher levels in the unfavorable group, where p= 0.002 (table 12).

A significant negative correlation was found between the troponin I level and each of GCS and GOS on the first day (where r =0.796 and 0.709 at p≤ 0.001 respectively) (tables 9 & 10).

On the third day

No cases of mild and moderate head injury had elevated troponin I on the third day, while two cases with severe head injuries had elevated levels. A significant relation was observed between GCS and cTnI (table 11).

The present data demonstrated a significant difference of troponin I level on the third day between unfavorable and favorable groups (1.0 ±0.52 versus 0.15 ±0.21) with higher levels in the unfavorable group, although it was within normal range (table 12).

A significant negative correlation was found between the troponin I level and each of GCS and GOS on the third day (where r =0.825 and 0.728 at p≤ 0.001, respectively) (tables 9 & 10).

CRP values and outcome

On the first day

All cases of mild, moderate and severe head injury had elevated CRP level on the first day with significant relation between GCS and CRP level with highest level in severe cases (table 11).

The current study demonstrated a significant difference of CRP level on the first day between unfavorable and favorable outcome groups (50.89 ±35.93 versus 16.36 ±10.59) with higher levels in the unfavorable group, where p=-0.042 (table 12).

A significant negative correlation was found between the CRP level and each of GCS and GOS on the first day (where r =0.763 and 0.660, respectively) (tables 9 & 10).

On the third day

All cases with different degree of severity regarding GCS had elevated CRP level on the third day with a significant relation with GCS, where P=0.006 (table 11).

The present data demonstrated a significant difference of CRP level on the third day between unfavorable and favorable outcome groups (87.11 ±38.34 versus 25.55 ±11.58) with higher levels in the unfavorable group, where p≤ 0.001 (table 12).

A significant negative correlation was found between the CRP level and each of GCS and GOS on the third day (where r =0.864 and 0.844, respectively) (tables 9 & 10).

The highest correlation was observed between GOS and S100B level at day three as denoted in table (10).

The present data also showed significantly higher levels of S100B among patients with ICH compared to SAH in moderate and severe injuries at day one and day three, where p=0.021 and 0.033, respectively.

Table (1): Distribution of the patients with intracerebral hemorrhage according to GCS and GOS (n=20)

GOS	Mild (13-15) (n=8)		Moderate (9-12) (n=6)		Severe (≤8) (n= 6)	
	n	%	n	%	n	%
Favorable outcome	8	100%	3	50%	-	-
Unfavorable outcome	-	-	3	50%	6	100%
total	8	100%	6	100%	6	100%

Table (2): Relation between each of S100 B, cardiac troponin I, C- reactive protein levels and GCS among patients with intracerebral hemorrhage at day one and three

Parameter		GCS			p
		Mild (13-15)	Moderate (9 - 12)	Severe (≤8)	
S100 B 1st day (µg/L) (n =20)	Min. – Max.	0.03 – 0.23	0.13 – 1.78	1.13 – 2.95	0.001*
	Mean ± SD	0.12 ± 0.07	0.58 ± 0.65	1.98 ± 0.64	
	Median	0.11	0.28	1.99	
S100B 3rd day (µg/L) (n =19)	Min. – Max.	0.01 – 0.20	0.08 – 2.90	0.47 – 2.60	0.005*
	Mean ± SD	0.10 ± 0.08	0.96 ± 1.18	1.62 ± 1.04	
	Median	0.08	0.33	2.0	
Troponin 1st day(µg/L) (n =20)	Min. – Max.	0.02 – 0.15	0.09 – 0.40	0.05 – 2.95	0.006*
	Mean ± SD	0.06 ± 0.04	0.26 ± 0.15	0.82 ± 1.11	
	Median	0.05	0.30	0.31	
Troponin 3rd day(µg/L) (n =19)	Min. – Max.	0.02 – 1.02	0.10 – 1.40	0.05 – 1.90	0.242
	Mean ± SD	0.39 ± 0.43	0.48 ± 0.52	0.92 ± 0.75	
	Median	0.15	0.30	1.15	
CRP 1st day(mg/L) (n =20)	Min. – Max.	10.0 – 35.0	21.0 – 72.0	56.0 – 118.0	0.001*
	Mean ± SD	19.63 ± 9.40	42.0 ± 18.63	86.33 ± 20.83	
	Median	15.50	38.0	88.50	
CRP 3rd day(mg/L) (n=19)	Min. – Max.	14.0 – 47.0	28.0 – 181.0	75.0 – 215.0	0.003*
	Mean ± SD	28.75 ± 11.17	85.17 ± 64.42	152.80 ± 59.92	
	Median	25.0	54.0	140.0	

*p: p value for Kruskal Wallis test *; Statistically significant at p ≤ 0.05*

Table (3): Difference between unfavorable and favorable outcome regarding S100 B, cardiac troponin I and C-reactive protein levels among patients with intracerebral hemorrhage at day one and three

Parameter		GOS		P
		Unfavorable (poor) outcome	Favorable (good) outcome	
S100 B 1st day (µg/L) (n = 20)	Min. – Max.	0.35 – 2.95	0.03 – 0.23	<0.001*
	Mean ± SD	1.66 ± 0.79	0.13 ± 0.06	
	Median	1.78	0.13	
S100 B 3rd day (µg/L) (n = 19)	Min. – Max.	0.46 – 2.90	0.01 – 0.20	<0.001*
	Mean ± SD	1.68 ± 1.03	0.11 ± 0.07	
	Median	1.98	0.08	
Troponin 1st day (µg/L) (n = 20)	Min. – Max.	0.05 – 2.95	0.02 – 0.40	0.006*
	Mean ± SD	0.70 ± 0.96	0.096 ± 0.11	
	Median	0.31	0.06	
Troponin 3rd day (µg/L) (n = 19)	Min. – Max.	0.05 – 1.90	0.02 – 1.02	0.049*
	Mean ± SD	0.90 ± 0.69	0.35 ± 0.37	
	Median	1.15	0.20	
CRP 1st day (mg/L) (n = 20)	Min. – Max.	45.0 – 118.0	10.0 – 35.0	<0.001*
	Mean ± SD	76.44 ± 23.22	21.73 ± 8.99	
	Median	74.0	21.0	
CRP 3rd day (mg/L) (n = 19)	Min. – Max.	65.0 – 215.0	14.0 – 47.0	<0.001*
	Mean ± SD	145.13 ± 56.56	31.27 ± 11.0	
	Median	145.50	28.0	

*p: p value for Mann Whitney test *; Statistically significant at p ≤ 0.05*

Table (4): Correlation between GCS and S100 B, cardiac troponin I and C- reactive protein levels among patients with intracerebral hemorrhage at day one and three

Parameter	r	p
S100 B 1st day	-0.949*	<0.001*
S100 B 3rd day	-0.894*	<0.001*
Troponin 1st day	-0.632*	0.004*
Troponin 3rd day	-0.430	0.075
CRP 1st day	-0.890*	<0.001*
CRP 3rd day	-0.895*	<0.001*

rs: Spearman coefficient; Statistically significant at p ≤ 0.05*

Table (5): Correlation between GOS and S100 B, cardiac troponin I and C- reactive protein levels among patients with intracerebral hemorrhage at day one and three

Parameter	r_s	P
S100 B 1st day	-0.900*	<0.001*
S100 B 3rd day	-0.964*	<0.001*
Troponin 1st day	-0.533*	0.019*
Troponin 3rd day	-0.418	0.084
CRP 1st day	-0.837*	<0.001*
CRP 3rd day (n = 19)	-0.882*	<0.001*

rs: Spearman coefficient; Statistically significant at p ≤ 0.05*

Table (6): Distribution of the patients with subarachnoid hemorrhage according GCS and GOS (n=20)

GOS	Mild (13-15) (n=7)		Moderate (9-12) (n=7)		Severe (≤8) (n= 6)	
	n	%	n	%	n	%
Favorable outcome	7	100%	4	57%	-	-
Unfavorable outcome	-	-	3	43%	6	100%
total	7	100%	7	100%	6	100%

Table (7): Relation between Fisher grading and GCS among patients with subarachnoid hemorrhage (n=20)

Fisher grading CT	GCS						MCp
	Mild (13-15) (n = 7)		Moderate (9 - 12) (n = 7)		Severe (≤8) (n = 6)		
	No	%	No	%	No	%	
II	7	100.0	5	71.4	0	0.0	<0.001*
III	0	0.0	2	28.6	2	33.3	
IV	0	0.0	0	0.0	4	66.7	

MCp: p value for Monte Carlo test; Statistically significant at p ≤ 0.05*

Table 8: Difference between unfavorable and favorable outcome regarding Fisher grade among subarachnoid hemorrhage patients (n=20)

CT (Fisher grading)	GOS				FEp
	Unfavorable (n = 9)		Favorable (n = 11)		
	No.	%	No.	%	
II	1	11.2	11	100.0	<0.001*
III	4	44.4	0	0.0	
IV	4	44.4	0	0.0	

FEp: p value for Fisher Exact test for comparing between the two studied group; Statistically significant at p ≤ 0.05*

Table (9): Correlation between GCS and Fisher grading, S100 B, cardiac troponin I and C- reactive protein levels among patients with subarachnoid hemorrhage at day one and three

Parameter	r_s	p
CT (Fisher grading)	-0.870*	<0.001*
S100 b 1st day	-0.832*	<0.001*
S100 b 3rd day	-0.881*	<0.001*
Troponin 1st day	-0.796*	<0.001*
Troponin 3rd day	-0.825*	<0.001*
CRP 1st day	-0.763*	<0.001*
CRP 3rd day	-0.864*	<0.001*

rs: Spearman coefficient; Statistically significant at p ≤ 0.05*

Table (10): Correlation between GOS and S100 B, cardiac toponine I and C- reactive protein levels and Fisher grading among patients with subarachnoid hemorrhage at day one and three

Parameter	r_s	p
CT (Fisher grading)	- 0.876*	<0.001*
S100 b 1st day	-0.758*	<0.001*
S100 b 3rd day	-0.955*	<0.001*
Troponin 1st day	-0.709*	<0.001*
Troponin 3rd day	-0.728*	0.001*
CRP 1st day	-0.660*	0.002*
CRP 3rd day	-0.844*	<0.001*

rs: Spearman coefficient; Statistically significant at p ≤ 0.05*

Table (11): Relation between each of S100 B, cardiac troponin I , C- reactive protein levels and GCS among patients with subarachnoid hemorrhage at day one and three

Parameter		GCS			p
		Mild (13-15)	Moderate (9 - 12)	Severe (≤8)	
S100 B 1st day (µg/L) (n=20)	Min. – Max.	0.06 – 0.50	0.14 – 0.58	0.20 – 2.0	0.002*
	Mean ± SD	0.16 ± 0.15	0.25 ± 0.16	1.20 ± 0.59	
	Median	0.12	0.19	1.30	
S100 B 3rd day (µg/L) (n=18)	Min. – Max.	0.02 – 0.18	0.12 – 0.53	0.50 – 1.54	0.003*
	Mean ± SD	0.09 ± 0.07	0.28 ± 0.16	1.06 ± 0.43	
	Median	0.05	0.20	1.10	
Troponin 1st day (µg/L) (n=20)	Min. – Max.	0.02 – 0.08	0.09 – 0.70	0.40 – 1.10	<0.001*
	Mean ± SD	0.04 ± 0.02	0.32 ± 0.21	0.76 ± 0.25	
	Median	0.03	0.25	0.79	
Troponin 3rd day (µg/L) (n=18)	Min. – Max.	0.01 – 0.08	0.07 – 0.63	0.50 – 1.80	0.002*
	Mean ± SD	0.04 ± 0.02	0.44 ± 0.24	1.19 ± 0.56	
	Median	0.04	0.55	1.22	
CRP 1st day (mg/L) (n=20)	Min. – Max.	3.0 – 27.0	10.0 – 34.0	23.0 – 95.0	0.005*
	Mean ± SD	12.29 ± 9.93	18.43 ± 8.73	70.50 ± 26.05	
	Median	10.0	14.0	73.0	
CRP 3rd day (mg/L) (n=18)	Min. – Max.	6.0 – 42.0	26.0 – 62.0	25.0 – 134.0	0.006*
	Mean ± SD	22.14 ± 13.13	42.43 ± 14.58	102.17 ± 38.99	
	Median	19.0	36.0	114.50	

p: p value for Kruskal Wallis test; Statistically significant at p ≤ 0.05*

Table (12): Difference between unfavorable and favorable outcome regarding Fisher grading, S100 B, cardiac troponin I and C- reactive protein levels among patients with subarachnoid hemorrhage at day one and three

Parameter		GOS		P
		Unfavorable (poor outcome)	Favorable (good outcome)	
S100 B 1st day (µg/L) (n = 20)	Min. – Max.	0.14 – 2.0	0.06 – 0.50	0.002*
	Mean ± SD	0.92 ± 0.64	0.17 ± 0.12	
	Median	1.01	0.14	
S100 B 3rd day (µg/L) (n = 18)	Min. – Max.	0.35 – 1.54	0.02 – 0.20	<0.001*
	Mean ± SD	0.79 ± 0.45	0.11 ± 0.07	
	Median	0.53	0.15	
Troponin 1st day (µg/L) (n = 20)	Min. – Max.	0.25 – 1.10	0.02 – 0.70	0.002*
	Mean ± SD	0.61 ± 0.30	0.15 ± 0.21	
	Median	0.57	0.07	
Troponin 3rd day (µg/L) (n = 18)	Min. – Max.	0.50 – 1.80	0.01 – 0.60	0.002*
	Mean ± SD	1.0 ± 0.52	0.15 ± 0.21	
	Median	0.82	0.06	
CRP 1st day (mg/L) (n = 20)	Min. – Max.	10.0 – 95.0	3.0 – 34.0	0.041*
	Mean ± SD	50.89 ± 35.93	16.36 ± 10.59	
	Median	67.0	14.0	
CRP 3rd day (mg/L) (n = 18)	Min. – Max.	25.0 – 134.0	6.0 – 42.0	0.001*
	Mean ± SD	87.11 ± 38.34	25.55 ± 11.58	
	Median	105.0	26.0	

p: p value for Mann Whitney test;Statistically significant at p ≤ 0.05*

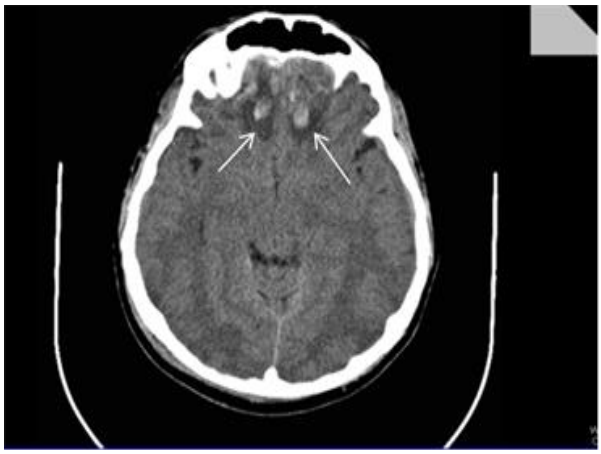

Figure 1: CT scan showing well defined hypodensity seen involving both frontal lobes with evidence of effacement of the related sulci ,Intra-parenchymal hemorrhages

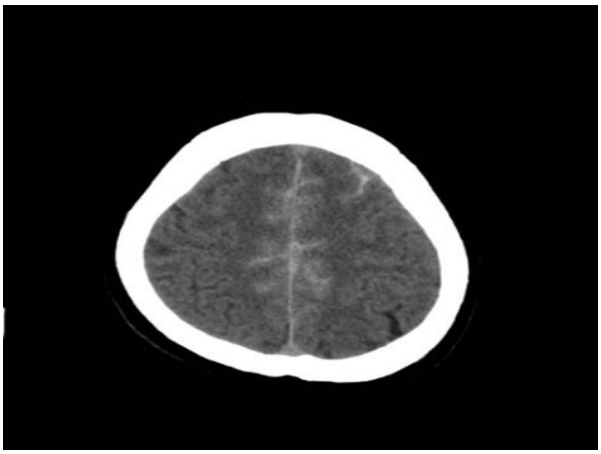

Figure 2: Hyperdensity is seen casting the cerebral sulci, fissures and cisterns impressive of subarachnoid hemorrhage

Discussion

Traumatic brain injury (TBI) comes to the attention of the forensic team and has had a thorough medical workup with various opinions given about causation and prognosis. Occasionally, the workup is incomplete and the forensic team is able to add important dimensions to understand the individual's prognosis and treatment (Raffle, 2012).

Assessment of TBI still remains less than optimal. Predicting prognosis on the basis of the original insult to the brain is difficult. A modality that has been of considerable interest is the assessment of certain biochemical markers in serum after TBI (Mehta, 2010). Furthermore, in analyzing prognostic data, it has been found that multifactorial analyses seem to allow for better outcome prediction accuracy (Zasler ND and Cantor IV, 2004).

Moreover, precise diagnosis and prediction of outcome may solve many problems of medico legal importance as the impact of malpractice on doctors due to delay in interference or absence of facilities.

Therefore, the present study aimed to assess the value of biochemical markers in serum; S100B, cardiac troponin I and C-reactive protein, to predict the outcome among patients with traumatic intracerebral and subarachnoid hemorrhage.

Patients with any cause of cardiovascular injury or thoracic trauma were not included in the present study to avoid the influence of cardiac abnormalities on cTnI level.

Moreover, it is difficult to interpret the significance of S100B elevations in patients with brain injury because variable amounts of skeletal muscle injury may influence the elevations. Yet, the release of S100B from injured skeletal muscle has been found to be short lived and returned to normal value within 20 hours. Therefore, serum CK was simultaneously measured in all patients in the present study to distinguish the cerebral component of S100B from the skeletal muscle component (Bloomfield et al., 2007).

Intracerebral hemorrhage

The present data showed a significant relation between GCS and S100B level both in first and third day. Early S100B elevation after trauma could be explained by astrocytic activation that has been found immediately following primary brain injury. S100B protein is involved in the astrocytes reaction to injury by regulating the Calcium influxes and stimulating astrocytic proliferation via interaction with transcription factors (Bloomfield et al., 2007).

With evaluation of outcome of the studied cases using GOS, The outcome was grouped into the favorable outcome (good recovery and moderate disability) and the unfavorable outcome (ranged from severe disability to death) (Gaetani et al., 1995; Jennett, 2005). In the current study, among patients with ICH, a significant difference was observed regarding S100 B levels in both groups. Unfavorable outcome group demonstrated significantly higher S100 levels than those of good outcome group at day 1 and day 3, in agreement with Yoon et al study (2008), that have demonstrated similar findings but in spontaneous intracerebral hemorrhage (Yoon et al.,2008).

The present data also revealed significant negative correlation of S100B with each of GCS and GOS. In accordance with the present study, several investigations have reported that S100B serum level elevation reflects severity of brain injury and that the extent of S100B elevation can predict outcomes (Dimopoulou et al., 2003; Rothoerl et al., 2000; Townend and Ingebrigtsen, 2006; Böhmer et al., 2011; Madkour et al., 2005). Yet, some of these studies measured S100B only within 24 hours after injury (Rothoerl et al., 2000; Madkour et al., 2005).

In the present study, the higher correlation of S100 B with GOS was observed at day three, that was attributed to some change in S100B level; increase of S100B level with poor outcome and the decrease of

S100 B level with good outcome. It may reflect the importance of repeated assay of this marker.

This information may be helpful in guiding clinicians in choosing the most appropriate treatment. If the levels reach a threshold that reliably predicts poor outcome despite aggressive therapy, these treatments could be avoided and other measures considered.

The brain-heart connection is corner stone to maintain normal cardiovascular function. This relationship concerns the central and autonomic nervous systems, and their impairment can adversely affect the cardiovascular system and induce stress-related pathology. Enhanced sympathetic tone induces endogenous catecholamine's stimulation of the myocardium (Samuels, 2007; Bybee and Prasad, 2008; Richard, 2011).

Serum levels of cardiac troponin (Tn I), a small regulatory protein of heart muscle, are the most sensitive and specific markers of myocardial cell damage currently in widespread use. cTnI level was categorized as normal (0 –1.2 μg/L), intermediate (1.3–5 μg/L), or high (>5 μg/L) (Martin etal., 2005).

In the present study, troponin levels showed an increase above 1.2 μg/L, in three cases, in moderate (one patient) and severe cases (two patients). One case showed elevated cTnI at first day and died before an ECG on third day. Cases presented with elevated cTnI also showed ST-segment changes and T-wave abnormalities, reflecting ischemic heart changes.

A significantly higher cTnI levels was demonstrated in poor outcome group compared to good outcome group at day 1and day 3, although the mean values were within reference laboratory range. It was attributed to the small number of cases showing elevated cTnI.

At day one, a significant correlation was observed between troponin levels and each of GCS and GOS in accordance with Hays and Diringer (2006) and Salim et al (2008) who concluded that the level of TnI correlates with the severity of head injury and is a predictor of adverse outcomes in traumatic brain injury.

It has been long known that tissue damage occurs following trauma, and as a result, liver releases acute phase reactant (APR) into blood stream. One of the better known APR is C-reactive protein (CRP) (Gabbe et al., 2003).

The current results revealed high levels of CRP reflecting the severity of GCS. Similar results were obtained by Meisner et al (2006) who found that initial CRP levels increased at three levels above 10 mg/l, above 50 mg/l. and higher concentrations did not exceed 365 mg/l during severe trauma.

The present study also revealed significant correlation between CRP levels and each of GCS and GOS. The correlation was higher on day three as well.

In agreement with Lee et al (2005) and Sogut et al., (2010), they concluded that the high serum level of CRP was more in patients with low GCS and in patients with poor outcome.

Among patients with traumatic ICH, in the current study, the highest correlation was demonstrated between S100B level on third day and GOS, followed by S100B level on first day.

Subarachnoid haemorrhage

Regarding S100B levels among patients with traumatic SAH, similar results were obtained and significant correlation to GCS and GOS at day one and three were observed. In contrast to the present data, Yoon et al (2008) concluded that, SAH patient was relatively steady during the first 3 days, where as in ICH patient showed abrupt S100 surge on admission suggesting brain damage at the time of bleeding in ICH patient. Yet, this study included patients with spontaneous hemorrhage rather than traumatic causes.

In the present study, a significant correlation was observed between cTnI level and each of GCS and GOS. Two cases of severe injury (GCS < 8) showed an elevated cTn level on third day with ST-segment changes and T-wave abnormalities. None of the dead cases was associated with elevated cTnI level. Further investigations of lager sample size with various head injury severity are needed.

Various studies were in consistent with the present findings, concluded that an increased cTnI level detected in cases with traumatic SAH considered as a predictive of the outcome (Sogut et al., 2010; Baffoun et al., 2011). The increase of serum TnIc level and T-wave changes on third day, suggesting that daily assaying of this myocardial enzyme must be systematically performed in all patients with tSAH during at least the seven first days. Similar to the present findings, Baffoun et al (2011) demonstrated higher level of cTnI on third day.

However, the clinical relevance of these findings is questionable. Browers et al., (1989) suggested that poor outcome amongst patients with more severe ECG irregularities was due to the fact that ECG abnormalities were indicators of severe intracranial disease.

Other studies referred the myocardial injury to sympathetic changes and catecholamine release that concomitantly contributed to the development of the ischemic changes of myocardium. Martin et al (2005) speculated that, increased serum TnI after trauma is related to the degree of overall injury and physiologic stress and intermediate and high TnI increases are associated with poor outcome.

Pollick also had observations that compromised hypothalamic circulation lead to a derangement in autonomic function (Pollicket al., 1988).

The present data may suggest the need for more than one parameter for precise outcome prediction or evaluation of traumatic cases.

The present study revealed that at all degrees of severity had the mean values of CRP more than 3 mg⁄L with significant negative correlation with each of

GCS and GOS. The correlation was higher also on third day.

In the present study, Fisher grading correlation with GCS was higher than CRP. Lee et al., (2005) concluded that in TBI, CRP alone is not adequate to assess the severity of TBI but it can be useful in cases in which imaging studies are usually less optimal to reveal the severity of TBI.

Among patients with traumatic SAH, the highest correlation with GOS was noticed with S100B levels on third day, followed by CT finding. Moreover, the third correlated parameter was CRP on third day.

Due to the large number of patients sustaining head injury, repetitive CT scanning and the need for repetitive neurological supervision may generate serious financial and logistic burdens, especially in areas where trauma incidence is high and medical resources are limited (Figueiredo et al., 2006). Therefore, early and sensitive screening tests suspecting the outcome in presence of intracranial lesions in patients is very much needed to provide rapid and sufficient health care prevent complication and protect the physician from malpractice claim.

The highest correlation, in both ICH and SAH, was noticed between S100B levels on third day and GOS. It could be explained as S100B might be useful enough to identify the early stages of secondary CNS injury so that it could be a good predict clinical outcome. Furthermore, S100 B is a constitutive protein of glial cells. Due to specificity of its cellular expression, S100B protein is a useful biological marker of acute neurological disorders (Bloomfield, 2007).

On comparing ICH and SAH, S100B was higher in ICH, in agreement with Yoon et al (2008) who demonstrated similar findings. It could be explained as the degree of brain damage was more severe in ICH compared to SAH group based on the S100 level.

In conclusion, serum S100B, cTnI and CRP levels were negatively correlated with the severity of GCS and GOS among patients with ICH and SAH, except the level of cTnI level on third day among patients with ICH. Furthermore, patients with unfavorable outcome had significantly higher serum S100B, cTnI and CRP levels than favorable outcome. The highest correlation of all biomarkers, in both ICH and SAH, was noticed between S100B level on third day and GOS.

In brain injury, there are consequences to the patient, the family and the healthcare providers. Hopelessness and despair are common emotions felt by these persons. In this framework, ethical and moral dilemmas arise. In the background of the dilemmas which arise in such cases, the clinician should be honest and direct in conveying diagnostic and prognostic information to the family (Zasler ND and Cantor IV, 2004).

Recommendation

The usefulness of using more than one parameter may increase the accuracy of prognostic prediction allowing a faster and more valid prediction of outcome during the early period after trauma.

To achieve the clinical utility of these parameters, it requires more information describing what the threshold for poor outcome really is**.** There is a need of further study of large population with different grades of TBI severity.

These parameters are suggested for studying their usefulness in the postmortem examination as markers of brain damage severity.

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الملخص العربى

تقييم الدور المحتمل لمستوي كل من اس ١٠٠ ب و التروبونين وبروتين سي التفاعلي كدلالات تنبؤية في حالات النزيف الإصابي بالمخ وتحت العنكبوتية

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تعد الإصابات بالرأس من اهم اسباب الوفيات والاعتلال في العالم. حتى الإصابات الطفيفة يمكن ان تؤدي الي بعض الاضرار العصبية النفسية. ويمكن استخدام بعض الدلائل الكيميائية الحيوية في تنبؤ النتائج. حيث يمكن ان يعكس مستوى إس ١٠٠ ب درجه اصابه الجهاز العصبي. كما أن التروبونين آي يعد من الدلالات الإصابات لعضلة القلب، وكذلك فإن بروتين سي التفاعلي يزيد في حالات الإصابات والالتهابات. ولذلك هدفت هذه الدراسة لتقييم الدور المحتمل لمستوى كل من إس ١٠٠ ب و التروبونين آي و بروتين سي التفاعلي كدلالات تنبؤية في حالات النزيف الإصابي بالمخ وتحت العنكبوتية.

وقد تم اجراء هذه الدراسة علي ٤٠ حالة من الاناث و الذكور تم تشخيصهم كحالات نزيف إصابي بالمخ (٢٠حالة) و حالات نزيف إصابي تحت العنكبوتية (٢٠حالة) في خلال ٢٤ ساعة من النزيف الإصابي. وتم تقييم مؤشر جلاسجو عند الدخول بالمستشفى لكل الحالات، كما ان درجة فيشر تم تقييمها لحالات النزيف الإصابي تحت العنكبوتية، و كذلك تم استخدام رسام القلب الكهربائي لكل الحالات. وتم استخدام المصل في اليوم الاول و الثالث لقياس مستوى إس ١٠٠ ب و التروبونين آي و بروتين سي التفاعلي. وتم تقييم مؤشر جلاسجو التنبؤي عند الوفاة او بعد ثلاثة أشهر.

وقد أظهرت النتائج أن مستوى إس ١٠٠ ب و التروبونين آي و بروتين سي التفاعلي كان له علاقة تبادلية سلبية مع درجة خطورة مؤشر جلاسجو و مؤشر جلاسجو التنبؤي بين حالات النزيف الإصابي بالمخ وتحت العنكبوتية ماعدا مستوى التروبونين آي في اليوم الثالث. كما ان الحالات ذات النتائج غير المرضية كانت ذات مستوى مرتفع لكل من اس ١٠٠ ب و التروبونين آي و بروتين سي التفاعلي. وقد لوحظ ان أعلى علاقه تبادلية كانت بين مستوى إس ١٠٠ ب في اليوم الثالث و مؤشر جلاسجو التنبؤي.

ولذا نستخلص ان مستوى إس ١٠٠ ب في اليوم الثالث أظهر أعلى علاقة تبادلية مع مؤشر جلاسجو التنبؤي بين حالات النزيف الإصابي بالمخ وتحت العنكبوتية.

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