

INCIDENCE OF ACUTE CORONARY SYNDROME AFTER TRAUMATIC BRAIN INJURY IN INTENSIVE CARE UNIT (ASSOCIATED FACTORS AND MORTALITY): A RETROSPECTIVE STUDY

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ABSTRACT

Background: Although the association between traumatic brain injury (TBI) and cardiac diseases were reported previously, the incidence of acute coronary syndrome (ACS) (per se) was not fully elucidated. The aim of this work was to estimate the incidence of ACS, associated factors and mortality during the first week of admission in patients with TBI admitted to intensive care unit (ICU). **Patients and Methods:** This retrospective study included all adult patients with TBI admitted to ICU (Al-Azhar University Hospital, Damietta), during (2016-2018). Patients with a history of cardiac co-morbidity, those who had associated chest, abdominal trauma, or bone fractures, were excluded. The collected data included: patient demographics, ICU clinical and laboratory data and history of chronic diseases. In additions, serum Troponin I, Glasco coma scale (GCS), electrocardiogram (ECG), echocardiographic examination, and patients' outcome were recorded. patients were divided into two groups according to the development of ACS the first included those who developed ACS and the second included those who did not develop ACS. **Results:** Of the 90 patients with TBI admitted to ICU, ACS was developed in (30.0%), age was (68.7±6.4), chronic diseases (40.7%). GCS was significantly lower in ACS group. Tachycardia, hypertension and hypernatremia was documented at admission. RBCs, hemoglobin and platelet count were significantly decreased while INR and PTT were elevated in ACS at admission and at 7th day. ECG changes in ACS group were in the form of ST elevation, ST depression and hyperacute T wave. Significant elevated troponin and abnormal echocardiographic findings were found in ACS group. Finally, significant increased mortality during the first week of admission in ACS compared to negative group (29.6% vs 3.2% respectively). **Conclusions:** These results documented the development of ACS after TBI and associated with older age, increased chronic disease, severity of trauma, hemodynamic instability, coagulopathy and increased ICU mortality. Search for ACS and identification of high-risk patients after

TBI are crucial to prevent cardiac morbidity and mortality. Otherwise, physicians could be exposed to medical negligence claims.

Keywords: Traumatic brain injury, ECG changes, Acute Coronary Syndrome, Cardiac Troponin I, Echocardiography.

INTRODUCTION

Traumatic brain injury (TBI) represent a significant etiology of death disability. About 13 million people live with disabilities due to TBI in Europe and United States of America. Severe TBI was confined to 10–15% of patients who need special care, and usually managed in the intensive care unit (ICU) with a combination of medical and surgical approaches (Stocchetti et al., 2017).

In TBI, the heart could be affected directly as a part of polytrauma, or indirectly by trauma-induced hypotension with coronary hypoperfusion. In addition, the stress of trauma induces catecholamine surge and release of inflammatory mediators that could harm cardiac myocytes (Salim et al., 2008).

In addition, TBI can disturb the autonomic nervous system, with subsequent cardiac dysfunction (Leitman, 2012).

The brain heart link could be explained by neurocardiac axis theory and neurogenic stunned myocardium phenomenon, with better understand and management of TBI (Cai et al., 2016).

The decreased blood to the coronaries with catecholamine surge may lead to acute coronary syndrome (ACS), which is a set of signs and symptoms due to harmful effect on cardiac muscle leading to its malfunction or even death (Amsterdam et al., 2014).

ACS usually presents by chest pain, radiating to the left shoulder, sense of tightness, with nausea and sweating.

Atypical presentation was reported especially in women old people, and patients with diabetes mellitus. The diagnosis depends on clinical presentation, changes in ECG, increased troponins and detection of wall abnormalities by echocardiography (Thygesen et al., 2012).

Although the association between TBI and cardiac diseases were reported previously, the incidence of ACS (per se) was not fully elucidated. The aim of the work to estimate the incidence of acute coronary syndrome, associated factors and mortality during the first week of admission in patients with traumatic brain injury admitted to intensive care unit.

PATIENTS AND METHODS

We retrospectively analyzed files of all adult patients (≥ 18 years) admitted to intensive care unit (Al-Azhar University Hospital, Damietta), during the last 3 years (2016-2018). Inclusion criteria specified adult patients of ≥ 18 years of age that have received a diagnosis of TBI verified by magnetic resonance imaging (MRI). Exclusion criteria included; Patients aged below 18 years, Patients with a history of cardiac co-morbidity (defined as documented preadmission coronary artery disease, myocardial ischemia/ infarction, arrhythmia, heart failure, untreated hypertension and cardiac pacemaker), a past medical history of cardio-thoracic surgery or significant cardiovascular stroke. Also, patients with extracranial injuries such as (those who

had associated chest, abdominal trauma, bone fractures) or an expected brain death were excluded from the study (**Cuisinier et al., 2016**). The study protocol was approved by the Local Research and Ethics Committee of Al-Azhar University (number: ADFM-IRB05082018). The collected data included the following: patient demographics (age and gender), history of chronic medical diseases (cancer, diabetes, hepatitis, and chronic kidney disease) and previous surgery. The severity of brain injuries assessed by Glasgow coma scale (GCS) at admission (**Esterov and Greenwald, 2017**).

In additions, results of clinical examination and ICU monitoring of heart rate, mean arterial blood pressure, and oxygen saturation during the first week of admission (at admission and at 7th day). Also, results of laboratory investigations such as complete blood count, liver function tests, kidney function tests and coagulation profile were searched for and documented. Finally, results of 12-lead ECG, serum troponin (at 24 hours and 72 hours) and trans-thoracic echocardiography were included in the assessment (**Hasanin et al., 2016**).

After collection and organization of data, patients were divided into two groups according to the development of acute coronary syndrome during ICU admission; the first included those who developed ACS and the second included those who did not develop ACS. Mortality during the first week were documented and included in the analysis.

Statistical analysis of data: the collected data were prepared and entered in an excel sheet; then coded and transferred to statistical package for social science (SPSS) program, version 18

(IBM® SPSS® Inc., USA). Quantitative data were expressed as mean \pm SD (Standard deviation), while qualitative data were presented as frequency (number) and percent distribution. For purpose of comparison between groups, independent samples student (t) test, and Chi square test were used for quantitative and qualitative data respectively. P value < 0.05 was considered significant. In the present work, the receiver operating characteristic (ROC) methodology was employed and the area under the ROC curve (AUC) was calculated to evaluate the predictive accuracy of ACS development and mortality in patients with (TBI). The higher AUC values can be interpreted as having a higher predictive accuracy. Additionally, sensitivity and specificity were measured (**Linden, 2006**). For evaluation of risk factors of ACS in TBI patients, a regression analysis was performed.

RESULTS

Acute coronary syndrome was developed in 27 patients (30.0%). Patients who developed ACS were significantly older in age when compared to negative group (68.7 ± 6.4 vs 61.3 ± 4.4 years respectively). Additionally, AUC of 0.85 for Age indicated a good predictors of ACS development in patients with (TBI) Table (1, 4). ACS was also associated with significant increased history of chronic disease (40.7% vs 20.6%) and GCS was significantly lower in ACS group (6.4 ± 1.2 vs 7.2 ± 0.9). However, TBI was associated with male gender where males represented 63% and 81% of ACS and negative groups respectively, with no significant difference between groups. In addition, there was no significant

difference between both groups regarding history of previous surgery (Table1).

Table (2) presented hemodynamic and laboratory data among studied populations and revealed that, ACS group had significantly faster heart rate and higher mean arterial pressure at admission, but at the 7th day both groups were comparable. In addition, there was statistically significant decrease of RBCs, hemoglobin, platelet count, at admission and at the 7th day in ACS group when compared to negative group. However, serum sodium was significantly increased in ACS group at admission, but not at 7th day. Furthermore, both INR and PTT were significantly increased in ACS at admission and at 7th day. AUC values for HR, HB and INR at admission were 0.892, 0.896 and 0.848 respectively which reflects a good predictive accuracy and the stronger the positive association between HR, HB, INR and ACS (Table 4).

The p-values for the coefficients confirmed these results and revealed a statistically significant association between several variables and ACS in patients with TBI, including age, low GCS, low HB, higher mean arterial pressure, higher INR, higher sodium and

long PTT at admission suggesting the influence of those factors on the development of ACS in patients with TBI ($P < 0.05$) Table (6).

At admission, ECG changes in ACS group were in the form of ST elevation in (33.3%), ST depression in (55.6%) and hyperacute T wave in (11.1%). Elevated troponin was significantly increased at 24 and 72 hours in ACS group when compared to negative group (74.1% vs 30.2%) (81.5% vs 31.7%) respectively. In addition, abnormal Echocardiographic findings were significantly increased in ACS when compared to negative group (33.3% vs 6.3% respectively). Finally, mortality was reported in 10 patients (11.1%); eight of them were in ACS group and 2 in negative group with significant increase of mortality in ACS when compared to negative group (29.6% vs 3.2% respectively) (Table 3). The good predictors for mortality in ACS patients were age, HR, platelets and INR. The average values of AUC for the age, HR at admission, platelets at admission and INR were 0.868, 0.892, 0.892 and 0.901 respectively (Table 5).

Table (1): Demographic data, history of chronic disease, previous surgery and GCS of the studied patients with TBI and its Statistical significance tests (student t test and Chi square test).

Variable	ACS group (n=27)	Negative group (n=63)	Test	P value
Age (years)	68.7±6.4; 51-78	61.3±4.4; 53-76	6.36	<0.001*
Gender	Male	51(81.0%)	3.13	0.07
	Female	12(19.0%)		
History of chronic disease	11(40.7%)	13(20.6%)	3.90	0.048*
History of previous surgery	3(11.1%)	9(14.3%)	0.16	0.68
GCS	6.4±1.2; 4-8	7.2±0.9; 4-8	3.17	0.002*

* significant difference (P value < 0.05)

* GCS: (Glasgow coma scale)

*TBI: traumatic brain injury

Table (2): Hemodynamic parameters and laboratory investigations during the first week of admission in ICU of patients with TBI and their Statistical significance test (student t test).

		ACS group (n=27)		Negative group (n=63)		t	p
		Mean	S. D	Mean	SD		
Heart rate (HR) beats/min	At admission	105.0	12.2	94.1	11.0	4.18	<0.001
	At 7 days	90.4	8.2	86.9	4.7	1.14	0.25
Mean arterial pressure (mmHg)	At admission	91.9	4.2	87.5	5.6	3.68	<0.001
	At 7 days	83.2	3.2	85.5	4.8	1.105	0.27
*O2 saturation	At admission	96.7	0.9	96.8	0.8	0.56	0.57
	At 7 days	98.6	0.5	98.3	0.8	1.57	0.12
RBCs (10 ⁶ /cu mm)	At admission	3.3	0.2	3.4	0.2	3.97	<0.001
	At 7 th day	3.2	0.2	3.3	0.2	2.39	0.019*
Hemoglobin (HB) g/dL	At admission	11.9	0.4	12.4	0.2	6.91	<0.001
	At 7 th day	11.7	0.4	12.1	0.2	5.23	<0.001*
*WBCs (10 ³ /cu mm)	At admission	11.58	1.60	11.4	1.5	0.60	0.55
	At 7 th day	11.4	1.9	11.5	1.5	0.11	0.91
Platelets (10 ³ /cu mm)	At admission	182.5	46.0	223.6	15.7	6.30	<0.001
	At 7 th day	205.1	36.3	226.4	8.2	4.33	<0.001*
*ALT (U/L)	At admission	13.6	2.7	13.2	2.3	0.72	0.47
	At 7 th day	15.6	1.9	15.0	1.2	1.71	0.09
*AST (U/L)	At admission	19.4	4.6	18.1	2.5	1.73	0.09
	At 7 th day	17.1	1.8	17.4	1.9	0.66	0.50
*Total bilirubin (µmol/L)	At admission	1.2	0.4	1.1	0.2	1.36	0.17
	At 7 th day	0.9	0.2	0.9	0.1	0.36	0.71
*Direct bilirubin (µmol/L)	At admission	0.4	0.1	0.4	0.1	1.72	0.09
	At 7 th day	0.4	0.1	0.4	0.1	0.52	0.60
*Urea (mg/dl),	At admission	26.5	11.6	23.5	5.3	1.66	0.10
	At 7 th day	24.3	7.7	22.8	4.8	1.03	0.30
*Creatinine (mg/dl)	At admission	1.1	0.2	1.1	0.3	0.87	0.38
	At 7 th day	1.1	0.3	1.0	0.2	1.67	0.09
Sodium (mEq/L)	At admission	148.4	4.4	145.7	4.0	2.94	0.004
	At 7 th day	141.8	2.3	141.0	2.5	1.39	0.16
*Potassium (mEq/L)	At admission	4.0	0.3	4.0	0.3	0.28	0.77
	At 7 th day	4.1	0.3	4.0	0.3	0.66	0.51
INR	At admission	1.3	0.0	1.2	0.0	5.70	<0.001
	At 7 th day	1.2	0.0	1.2	0.0	4.11	<0.001*
PTT/ seconds	At admission	26.0	3.4	24.4	1.8	2.83	0.006
	At 7 th day	25.7	2.2	24.1	1.2	4.04	<0.001*

Quantitative data were expressed as mean ± SD (Standard deviation)

* significant difference (P value < 0.05)

*ICU: intensive care unit

*TBI: traumatic brain injury

*Normal value for; HR (60–100 beats/min), arterial pressure (90–140 mmHg), HB (12 -16 g/dL), RBCs (4.5-5.9 x 10⁶/cu mm), WBC (4.0-10.0 x 10³/cu mm), Platelets (150-450 x 10³/cu mm), AST (0 to 35U/L), ALT (7–56 U/ L), ALP (41 to 133U/L), total bilirubin (2 to 21µmol/L), direct bilirubin (less than 8µmol/L), Urea (5 to 20 mg/dl), Creatinine (0.6 to 1.2 mg/dl), Na (135 to 145 mEq/L), Potassium (3.5-5.0 mEq/L), international normalized ratio (INR) 1 to 2, Partial Thromboplastin Time (PTT 25-35 seconds).

Table (3): Cardiac troponin, ECG, Echo changes and mortality during the first week of admission in ICU of patients with TBI with their Statistical significance test (Chi square test).

		Positive (n=27)		Negative (n=63)		Total (n=90)		test	p
		0	%	0	%	0	%		
ECG	None	0	0.0%	63	100.0%	63	70.0%	90	<0.001*
	ST elevation	9	33.3%	0	0.0%	9	10.0%		
	ST depression	15	55.6%	0	0.0%	15	16.7%		
	Hyperacute T	3	11.1%	0	0.0%	3	3.3%		
Elevated Troponin(ng/mL)	At 24 hours	20	74.1%	19	30.2%	39	43.3%	14.84	<0.001*
	At 72 hours	22	81.5%	20	31.7%	42	46.7%	18.78	<0.001*
Abnormal echo findings		9	33.3%	4	6.3%	13	14.4%	11.13	0.002*
ICU mortality		8	29.6%	2	3.2%	10	11.1%	13.39	<0.001

* significant difference (P value < 0.05)

*ICU: intensive care unit

*TBI: traumatic brain injury

Table (4): Receiver operating characteristic (ROC) curve analysis to predict ACS development during the first week of admission in ICU of patients with TBI

Variable	AUC	Cut off	Sensitivity	Specificity
Age (years)	0.85	> 66 years	70.37	90.84
GCS	0.68	= < 6	51.9	79.4
HR at admission	0.828	> 91	96.3	74.6
Mean arterial pressure at admission	0.766	> 84	100.0	42.9
RBCs at admission	0.714	$\leq 3.2 \times 10^6$	62.96	73.02
HB at admission	0.848	≤ 12	66.67	93.65
Platelets at admission	0.729	≤ 210	55.56	98.41
Na at admission	0.68	>147	62.96	71.43
INR	0.848	>1.21	92.59	76.19
PTT	0.622	>26	40.74	85.71

AUC: area under the ROC curve. "Accuracy is measured by AUC *90-1 = excellent, *.80-.90 = good, *.70-.80 = fair, *.60-.70 = poor, *.50-.60 = fail.

*ICU: intensive care unit. *ACS: acute coronary syndrome *TBI: traumatic brain injury

Table (5): Receiver operating characteristic (ROC) curve analysis for prediction of mortality during the first week of admission in ICU of patients developed ACS after TBI

Variable	AUC	Cut off	Sensitivity	Specificity
Age (years)	0.868	>67	80.00	86.25
GCS	0.712	≤6	60.00	73.75
HR at admission	0.892	>97	100.00	71.25
Mean arterial pressure at admission	0.688	>87	90.00	48.75
RBCs at admission	0.677	≤3.3 x 10 ⁶	70.00	60.0
HB at admission	0.741	≤12	70.00	81.25
Platelets at admission	0.892	≤150 x 10 ³	90.00	96.25
Na at admission	0.758	>149	70.00	88.75
INR	0.901	>1.28	70.00	97.50
PTT	0.719	>27	70.00	97.50

AUC: area under the ROC curve. *Accuracy is measured by AUC *90-1 = excellent, *

.80-.90 = good, *.70-.80 = fair, *.60-.70 = poor, *.50-.60 = fail.

*ICU: intensive care unit. *ACS: acute coronary syndrome *TBI: traumatic brain injury

Table (6): Regression analysis for associated risk factors for (ACS) in patients with (TBI).

Independent variables	Coefficient	P-value*
Age (years)	0.01376	0.0336*
Echo_12hours	0.1390	0.3889
GCS	0.1122	0.0009*
HB at admission	0.5120	0.0001*
Heart rate at admission	0.003443	0.5081
Mean arterial pressure at admission	0.02513	0.0008*
INR at admission	2.8687	0.0254*
History of chronic disease	0.03218	0.6979
Na at admission	0.02069	0.0222*
Platelets at admission	0.002419	0.1367
PTT at admission	0.07388	0.0019*
RBCs at admission	0.1066	0.5699

* significant difference (P value < 0.05)

*ACS: acute coronary syndrome *TBI: traumatic brain injury

DISCUSSION

In the present work, the acute coronary syndrome was reported in 27 patients (30.0%) as evidenced by significantly elevated Troponin I, abnormal ECG and echocardiographic examination. **Krishnamoorthy et al., (2014)** reported that, ACS developed in 13.6%. This percentage is lower than those of the present work. This could be explained by the severe TBI patients included in the present work as indicated

by low GCS (6.4±1.2) (**Prathep et al., 2014**).

In addition, **Gregory and Smith (2012)** reported that, ECG abnormalities were reported after TBI in 49-100%, which is so higher than the present work. On the other side, **Cuisinier et al., (2016)** denied any major myocardial dysfunction at the early phase of TBI. However, after speckle tracking echocardiography, they reported minor changes in cardiac muscle.

In the present study, ECG changes in ACS group were in the form of ST elevation in (33.3%), ST depression in (55.6%) and hyperacute T wave in (11.1%). **Gregory and Smith, (2012)** demonstrated that ECG abnormalities after TBI such as ST segment depression and abnormal T waves can be associated with the development of a delayed ischemic neurological deficit, poor outcome, and death.

Abnormal echocardiographic findings were significantly increased in ACS when compared to negative group (33.3% vs 6.3% respectively). **Hasanin et al., (2016)** reported abnormal echocardiographic examination in (28 %) of patients developed cardiac injury after TBI. In addition, **Prathep and colleagues (2014)** documented abnormal echocardiographic examination in (22.3 %) of TBI patients.

In the present study, **cardiac troponin I(cTnI)** was elevated in 43.3% at the first 24 hours and in 46.7% in the third day. These results are higher than that reported by **Cai et al., (2016)** who reported that, among 580 patients with TBI, 31% had detectable cTnI values at the time of admission. In addition, **Salim et al., (2008)** reported that, elevated cTnI were reported in 29.8%. The possible explanation for increased cardiac troponin in the present work could be attributed to increased rate of chronic diseases and increased severity of TBI. The degree of cTnI increase among patients with TBI is associated with an increased risk of death and poor functional outcome in survivors (**Tung et al., 2004**).

TBI was predominant in **males** and this result is comparable to previous work by **Cai et al., (2016)** reported that, eligible patients were largely males (70.8%). The

reason might be that males are more vulnerable to road injuries and disputes (**Munivenkatappa et al., 2016**).

This study revealed that patients who developed ACS were significantly older in age when compared to negative group (68.7±6.4 vs 61.3±4.4 years respectively). Advanced age is among the best independent predictors of worse outcome after TBI (**Cai et al., 2016**).

In this work, there was **hemodynamic instability** among studied patients. However, ACS group had significantly faster heart rate and higher mean arterial pressure at admission, but at the seventh day both groups were comparable. These changes are explained by dysfunction of autonomic nervous system (ANS) due to TBI. ANS activity, has been shown to correlate with an increased risk of cardiac complications, including an increased risk of arterial hypertension. (**Kenney and Ganta, 2014**).

Heart rate variability seems to play an important role in the development of ACS and this can explain the significant increase of heart rate in ACS group when compared to negative group. **Orso et al., (2009)** reported that, the pathophysiologic mechanism underlying the development of ACS is unique consisting in the instabilization of coronary atherosclerotic plaque and the final disruption of the fibrous cap. This harmful action leads to the activation of the coagulation cascade with an intravessel red thrombus formation with the consequence of a total or partial occlusion of the coronary vessel.

Lim and Smith, (2007); Krishnamoorthy (2017) demonstrated that the sympathetic hyperactivity associated with severe TBI causes direct injury to the myocardium. Catecholamine-

induced vasoconstriction is intense, leading to hypertension and tachycardia and a secondary increase in myocardial oxygen demand without simultaneous increase in myocardial oxygen delivery, resulting in impairment of ventricular function even in absence of atherosclerosis.

CBC findings of the present study revealed that, there was anemia and thrombocytopenia among studied populations. **Kramer et al., (2012)** reported that, in ICU patients with TBI, the prevalence of anemia is about 22 to 69%, depending if extracranial hemorrhage is present and the time of measurements. In addition, results of the present study agree with previous works (**Kunadian et al., 2014; Uscinska et al., 2015**).

Both **INR and PTT** were increased above normal values and platelet count was lower than normal values in the studied patients with significant increase in ACS reflected that, there is a stage of coagulopathy associated with traumatic brain injury. Different mechanisms were reported to explain such state of coagulopathy **Laroche et al., (2012)** reported that, platelet disorders (number and function), changes in anticoagulant factors, cell hypoperfusion and inflammation; all could contribute to the development of coagulopathy associated with TBI. Another mechanism is due direct brain tissue insults, which lead to the release of Tissue Factor (TF), which is a known activator of the extrinsic coagulation pathway. TF is liberated with acute structural brain lesions. Ischemia, secondary to hypercoagulation and microthrombi may also be a contributing factor (**Abdelmalik et al., 2016**).

Serum sodium was significantly increased in ACS group at admission, but not at 7th day. This result is consistent with **Maggiore et al., (2009)**, this hypernatremia could be attributed to the use of continuous-infusion hypertonic saline. **Kolmodin et al., (2013)** reported that hypernatremia is common following traumatic brain injury (TBI) and occurs from a variety of mechanisms, including hyperosmotic fluids, limitation of free water, or diabetes insipidus.

Our results also indicated that, it may be possible for clinicians to predict ACS in patient with TBI based on select clinical characteristics namely, age > 66 years, INR >1.21, HR at admission > 91 and HB at admission ≤ 12. These findings consistent with previous results (**Gaddam et al., 2015**).

As regard mortality, it was reported in 10 cases during the first week (11.1%), 8 in ACS and 2 in negative group which is comparable to **Krishnamoorthy et al., (2015)** who reported that, in-hospital mortality occurred 10.2 % of their studied subjects. In addition, **Urdaneta et al., (2017)** reported that, 16 (11.5%) patients died during the hospitalization.

However, **Jochems et al., (2018)** reported a mortality rate of 33.0% during hospitalization. In addition, **Cai et al. (2016)** reported a mortality rate of 49.5%. This higher rate of mortality could be attributed to different sample size, the type of trauma or the pre-existing cardiac disease. The autonomic dysfunction has also been shown to correlate with increased morbidity and mortality in moderate and severe TBI (**Hendén et al., 2014**).

Current study also documented a significant increased incidence of in-

hospital mortality in ACS patients when compared to negative group (29.6% vs 3.2% respectively). Our findings are consistent with **Prathep et al., (2014); Hasanin et al., (2016)**, who concluded that cardiac injury was a mortality risk factor in patients with TBI.

The results of this study identified the increased age of patients, markers of coagulopathy, (prolonged INR & thrombocytopenia) and increased HR at admission, as significant predictor factors for mortality in patients developed ACS after (TBI) and admitted to the ICU. These findings are in line with results of previous study (**Lim and Smith 2007; Lin et al., 2015**).

Our findings have several potential clinical implications in both intensive care and anesthetic practice in cases of severe TBI. The possible occurrence of ACS in the setting of TBI and its impact on patient outcome suggest that the clinicians need to keep in mind the probability of cardiovascular changes in patients with TBI. Echocardiography and search for ACS must be a routine in ICU after TBI. Otherwise, clinicians could be blamed for medical malpractice or negligence. Identification of high-risk patients after TBI is important to be able to arrange appropriate cardiac monitoring, an effective management of associated cardiac dysfunction and also to prevent cardiac morbidity and mortality, thus serving like a "shield" to medical-legal claims in addition to suits.

CONCLUSIONS

Results of the present work documented the development of acute coronary syndrome after traumatic brain injury. The development of the disease

was associated with older age, increased chronic disease, severity of trauma, hemodynamic instability, anemia, coagulopathy and associated with increased ICU mortality. suggesting the influence of those factors on the development of ACS in patients with TBI. Watching those factors might amend the development of ACS in patients with TBI in clinical treatment and may prove useful in checking ACS after brain injuries.

RECOMMENDATIONS

- Further research is necessary to assess cardiac dysfunction broadly in traumatic head injuries, to recognize the impact of cardiac problems on patient outcomes, and to develop medical strategies that may either intercept or minimize the development of ACS and thus serving like a "shield" to medical-legal claims and suits.
- Enhancements in completeness and high quality of epidemiological data are needed for the detection of associated high-risk populations and recognition of key targets regarding improved prevention and management of ACS due to TBI.
- Legal issues regarding traumatic brain injuries and the diagnosis and management of them should be recognized and appreciated by medical providers and used to balance the applicable medical and legal risks associated with their practices.
- Clinicians must know the efficient ways to translate the development in basic and scientific research into clinical practice and public.
- An adequate funding is required in the long-term TBI research to recognize

best practices in addition to get the best outcomes.

- Implementation of prevention strategies and provision of optimum medical care for TBI patients in ICU should become a priority for physicians

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مدى حدوث الإصابة بمتلازمة الشريان التاجي الحادة بعد إصابة الدماغ في وحدة العناية المركزة (العوامل المرتبطة والوفيات): دراسة بأثر رجعي

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الملخص العربي

خلفية البحث: على الرغم من أن العلاقة بين إصابات الدماغ وأمراض القلب ذكرت سابقاً ، إلا أنه لم يتم توضيح حدوث متلازمة الشريان التاجي الحادة (في حد ذاتها) تماماً. لذلك كان الهدف من هذا العمل هو تقدير معدل حدوث متلازمة الشريان التاجي الحادة والعوامل المرتبطة بها والوفيات خلال الأسبوع الأول في المرضى الذين يعانون من إصابات الدماغ وتم ادخالهم وحدة العناية المركزة.

منهجية البحث: أجريت هذه الدراسة بأثر رجعي وشملت جميع المرضى البالغين الذين يعانون من إصابات في الدماغ في وحدة العناية المركزة (مستشفى الأزهر الجامعي، دمياط)، خلال (2016-2018). وقد تم استبعاد المرضى الذين لديهم تاريخ مرضي سابق في القلب، أو أولئك الذين لديهم إصابات بالصدر أو البطن أو كسور بالعظام. وتضمنت البيانات التي تم جمعها: الخصائص الديموغرافية للمرضى، البيانات السريرية والمخبرية وتاريخ الأمراض المزمنة. بالإضافة إلي ذلك، تم تسجيل مصل تروبونين 1 في الدم، مقياس غلاسكو للغيوبية (GCS) ، تخطيط كهربية القلب (ECG) ، والفحص بتخطيط صدى القلب، وسجلت نتائج وأثار ذلك علي المرضى. وقد تم تقسيم المرضى إلى مجموعتين وفقاً لظهور متلازمة الشريان التاجي الحادة من عدمه.

النتائج: أظهرت النتائج أن متلازمة الشريان التاجي الحادة حدثت في (30.0%) من بين 90 مريضاً لديهم إصابات في الدماغ بوحدة العناية المركزة، وكان متوسط عمر المرضى (68.7±6.4)، ونسبة الأمراض المزمنة لديهم (40.7%) مع ارتفاع ضغط الدم وعدم انتظام نبضات القلب وارتفاع نسبة الصوديوم في الدم في اليوم الأول لدخول العناية المركزة، كذلك كان مقياس غلاسكو للغيوبية (GCS) أقل في هذه المجموعة مقارنة بالمجموعة السلبية. كما أشارت نتائج هذه المجموعة الي حدوث انخفاض ذو دلالة إحصائية في عدد كرات الدم الحمراء والهيموغلوبين وعدد الصفائح الدموية في حين ارتفع مؤشر سيولة الدم "النسبة المعيارية الدولية" INR ووزن الثرموبلاستين الجزئي (PTT) في اليوم الأول والسابع. اسفرت النتائج كذلك أن المجموعة التي ظهر بها متلازمة الشريان التاجي الحادة حدثت بها تغييرات في تخطيط كهربية القلب في شكل ارتفاع أو انخفاض في (ST) وكانت موجة تي (T wave) شديدة الحدة مع زيادة ذات دلالة إحصائية في مصل تروبونين 1 في الدم بالتوافق مع تغييرات غير طبيعية في تخطيط صدى القلب، كما اقترن ذلك بزيادة كبيرة في معدل الوفيات خلال الأسبوع الأول مقارنة مع المجموعة السلبية (29.6% مقابل 3.2% على التوالي).

الاستنتاجات: هذه النتائج وثقت حدوث متلازمة الشريان التاجي الحادة بعد إصابات الدماغ والتي ارتبطت مع التقدم في العمر، وزيادة الأمراض المزمنة، شدة إصابة الدماغ وعدم ثبات واستقرار الدورة الدموية، أمراض تجلط الدم وكذلك زيادة معدل الوفيات في وحدة العناية المركزة. لذلك يعد البحث عن متلازمة الشريان التاجي الحادة واكتشاف حدوثها بعد إصابات الدماغ وتحديد المرضى المعرضين لحدوثها من الأمور الهامة للوقاية من الأمراض والوفيات القلبية. خلاف ذلك، يمكن أن يتعرض الأطباء للمساءلة القانونية بسبب الإهمال الطبي.

الكلمات المفتاحية: إصابات الدماغ، تغييرات تخطيط القلب، متلازمة الشريان التاجي الحادة ، تروبونين القلب 1 ، تخطيط صدى القلب.