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Prevalence of Electrocardiographic Changes in Patients with Traumatic Brain Injury: A Prospective Hospital-based Study

Mohamed Hamila[1†*], Khaled Hussein[2], Mohamed Fatehy Ismail[3], Ahmed Kamal[2]

[1]Department of Critical Care Medicine, Beni Suef University, Beni Suef, Egypt; [2]Department of Critical Care Medicine, Cairo University, Cairo, Egypt; [3]Department of Critical Care Medicine, King Fahad Specialist Hospital, Dammam, Saudi Arabia
[†]This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

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***Correspondence:** Mohamed Hamila, Department of Critical Care Medicine, Beni Suef University, Beni Suef, Egypt. E-mail: hamila@hotmail.com
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Abstract

BACKGROUND: Head trauma and traumatic brain injury (TBI) are major causes of death and disability worldwide. TBI is associated with a variety of electrocardiographic (ECG) changes.

AIM: We aimed to evaluate the prevalence of ECG changes in TBI.

METHODS: Participants with TBI were included in the study, while participants with chest trauma or cardiovascular diseases were excluded from the study. A consecutive sample of 50 participants (mean age 37.8 ± 14.85 years, 80% males) was selected and referred for 12 lead ECG on admission, 24 h, and 72 h after admission.

RESULTS: The prevalence of sinus bradycardia versus sinus tachycardia, short PR interval, ST segment elevation, and inverted T wave in the study population was 18% versus 38%, 26%, 2%, and 16% in ECG on admission, 5% versus 22%, 14%, 0%, and 10% in ECG 24 h after admission, 5% versus 8%, 4%, 0%, and 8% in ECG 72 h after admission, respectively. Serial ECG was significantly associated with changes in heart rate (χ^2 [1] = 17.337, p = 0.002) and short PR interval (χ^2 [1] = 9.695, p = 0.008), respectively. There was a significant association between ECG changes and brain edema (χ^2 [1] = 4.131, p = 0.042), intracerebral hemorrhage (χ^2 [1] = 4.539, p = 0.033), and subarachnoid hemorrhage groups (χ^2 [1] = 5.889, p = 0.015), respectively.

CONCLUSIONS: ECG changes are prevalent in non-cardiac TBI patients. The significant association of serial ECG with changes in heart rate and short PR interval and the significant association of ECG changes with brain edema, intracerebral hemorrhage, and subarachnoid hemorrhage highlights the potential role of serial ECG as a screening tool for cardiac dysfunction in patients with TBI.

Introduction

Traumatic brain injury (TBI) is a major cause of morbidity and mortality worldwide with an incidence rate of 69 million (95% CI 64–74 million) individuals per year [1]. Acute myocardial damage and heart failure may occur in non-cardiac TBI patients [2]. Electrocardiographic (ECG) changes associated with TBI include disturbance of rate, rhythm, P wave, QRS complex, PR interval, T wave, ST segment, prolongation of QT interval, or myocardial ischemic-like ECG changes including ST-segment deviations, T wave inversion, and Q-waves [3]. In 2004, early serial ECG has been recommended by Stollberger and Finsterer for assessment of cardiac dysfunction in TBI [4]. Subgroup analysis of the TBI patient population showed that ECG changes occur most often in subarachnoid hemorrhage (49–100%), intracranial hemorrhage, ischemic stroke, cerebral venous thrombosis, head trauma, neurosurgical procedures, cryohypophysectomy, acute meningitis, intracranial space-occupying tumors, and epilepsy [5], [6]. We wanted to evaluate the prevalence of ECG changes in TBI, the association of serial ECG with cardiac dysfunction, and the association of ECG changes with certain types of TBI.

Methods

Study design

Our study was a 1-year prospective, open-labeled, non-randomized, single cohort study conducted at a single Intensive Care Unit (ICU) in a trauma care center. Investigators weren't blinded to the study group. The study design was approved by the hospital ethics committee review board, all study participants' substitute decision-makers for healthcare signed written informed consents, study procedures were carried out following the Code of Ethics of the World Medical Association (Declaration of Helsinki), all study data and ECGs were anonymized, and the privacy rights of the study participants were observed diligently.

Study participants

Study participants were patients referred to the ICU. The study participants' family members were subjected to history taking and data collection for age, gender, risk factors including hypertension, and diabetes mellitus. The study participants' bystanders were asked about the mechanism of TBI. The study participants were subjected to comprehensive clinical examination including measurement of vital signs on admission (systolic and diastolic blood pressure, heart rate, respiratory rate, and body temperature), Glasgow Coma Scale (GCS) assessment, brain computerized tomography (CT) scan, complete blood count, prothrombin time, activated partial thromboplastin time, international normalized ratio, serum creatinine, sodium and potassium, aspartate aminotransferase and alanine aminotransferase, creatinine kinase and creatine kinase-MB, troponin, and serial 12 lead ECGs on admission, 24 h, and 72 h after admission. Data documented with serial ECGs included RR interval, P wave, PR interval, PR segment, QRS complex, ST segment, T wave, ST interval, and QT interval. Normal values for ECG waves and intervals were referenced to the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Electrocardiography) report [7]. TBI was categorized based on GCS into severe (GCS ≤ 8), moderate (GCS 9–12), and mild (GCS 13–14) [8]. Screened participants were enrolled if they had head trauma with CT brain showing brain edema, subdural hemorrhage, extradural hemorrhage, intracerebral hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage, or skull fractures. Screened participants with severe chest trauma, history of cardiovascular diseases, or on any cardiac medication were excluded from the study.

Study procedures

Fifty eligible participants were admitted to ICU, enrolled, consecutively assigned, and allocated in a single cohort. The enrolled study participants underwent serial ECG on admission, 24 h, and 72 h after admission.

End points

The study evaluated the prevalence of ECG changes in TBI, the association of serial ECG with cardiac dysfunction, and the association of ECG changes with certain types of TBI.

Statistical analysis

The ECG assessment outcomes were coded, and the data were analyzed with the Statistical Package for the Social Sciences software® version 20. Quantitative (continuous) data were expressed as means and standard deviations, while qualitative (categorial) data were expressed as frequencies and percentages. Comparisons between parametrically distributed quantitative variables were done with the Independent two-tailed t-test, between non-parametrically distributed quantitative variables with Mann-Whitney test, and between qualitative variables with Chi-square test, respectively [9], [10]. The confidence interval was set to 95% and the margin of error accepted was set to 5%. Any comparison considered statistically significant was at $p < 0.05$ or less.

Results

Study participants and procedures

We recruited 50 patients from one hospital in one country from August 2014 through April 2015. The study group was balanced with regards to baseline characteristics (Table 1) and risk factors (Figure 1). The key socio-demographic feature of the enrolled participants was male predominance (Mean age 37.84 ± 14.85 years, 80% males, 20% females) (Figure 2). All enrolled participants completed the study, and there were no withdrawals. Brain CT evaluation showed brain edema, subdural hemorrhage, extradural hemorrhage, intracerebral hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage, and fracture skull base in 38%, 4%, 20%, 36%, 26%, 10%, and 28% of the study population, respectively (Table 2).

Table 1: Patients characteristics and demographic data (n=50)

	Mean ± SD	
Age (Years)	37.84 ± 14.85	
	N	%
Age≤38 years	28	56
Age>38 years	22	44
Male	40	80.0
Female	10	20.0
Diabetes Mellitus	11	22.0
Hypertension	16	32.0
Glasgow Coma Moderate Brain Injury	23	46.0
Glasgow Coma Severe Brain Injury	27	54.0
	Mean ± SD	
Systolic Blood Pressure (mmHg)	126.00 ± 16.54	
Diastolic Blood Pressure (mmHg)	77.60 ± 10.21	
Respiratory Rate per Minute	20.36 ± 2.46	
Body Temperature (°C)	37.08 ± 0.32	

Prevalence of ECG changes in TBI

As per ECG assessment, the prevalence of sinus bradycardia versus sinus tachycardia, short PR interval, ST segment elevation, and inverted T wave in the study population was 18% versus 38%, 26%, 2%, and 16% in ECG on admission, 5% versus 22%, 14%, 0%, and 10% in ECG 24 h after admission, 5% versus

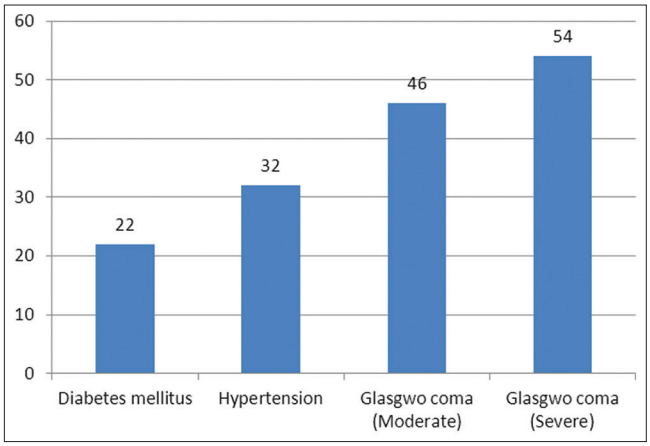

Figure 1: Risk factors distribution of the traumatic brain injury study group. Bar chart showing the percentage of the traumatic brain injury patients having diabetes mellitus (22%), hypertension (32%), and Glasgow coma moderate brain injury (46%) and severe brain injury (54%)

8%, 4%, 0%, and 8% in ECG 72 h after admission, respectively (Table 3).

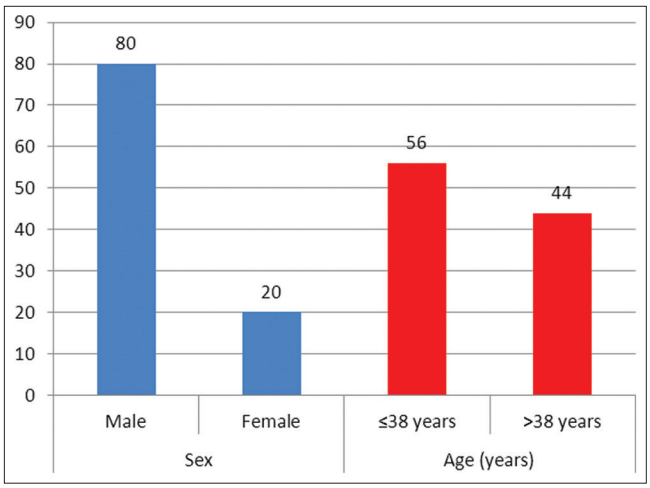

Figure 2: Demographic data distribution of the traumatic brain injury study group. Bar chart showing the percentage of male (80%) and female (20%) patients, those with age ≤ 38 years (56%) and >38 years (44%)

Association of serial electrocardiography with ECG changes in TBI

Serial ECG was significantly associated with changes in heart rate (χ^2 [1] = 17.337, p = 0.002) and short PR interval (χ^2 [1] = 9.695, p = 0.008), respectively (Table 4).

Table 2: Types of traumatic brain injury distribution in the study group (n=50)

	n	%
Brain edema	19	38
Subdural hemorrhage	2	4
Extradural hemorrhage	10	20
Intracerebral hemorrhage	18	36
Subarachnoid hemorrhage	13	26
Intraventricular hemorrhage	5	10
Fracture skull base	14	28

Table 2 showing the percentage of traumatic brain injury patients with brain edema (38%), subdural hemorrhage (4%), extradural hemorrhage (20%), intracerebral hemorrhage (36%), subarachnoid hemorrhage (26%), intraventricular hemorrhage (10%), and fracture skull base (28%), respectively.

Table 3: Prevalence of ECG changes in traumatic brain injury study group (n=50)

Heart rate	Normal		Abnormal			
			Bradycardia		Tachycardia	
	n	%	n	%	n	%
Heart rate on admission	22	44	9	18	19	38
Heart rate 24 h after admission	34	68	5	10	11	22
Heart rate 72 h after admission	41	82	5	10	4	8
Rhythm	Regular		Irregular			
	n	%	n	%		
Rhythm on admission	50	100	0	0		
Rhythm 24 h after admission	50	100	0	0		
Rhythm 72 h after admission	50	100	0	0		
PR Interval	Normal		Short			
	n	%	n	%		
PR interval on admission	37	74	13	26		
PR interval 24 h after admission	43	86	7	14		
PR interval 72 h after admission	48	96	2	4		
QRS complex	Normal		Short			
	n	%	n	%		
QRS complex on admission	50	100	0	0		
QRS complex 24 h after admission	50	100	0	0		
QRS complex 72 h after admission	50	100	0	0		
QT interval	Normal		Short			
	n	%	n	%		
QT interval on admission	50	100	0	0		
QT interval 24 h after admission	50	100	0	0		
QT interval 72 h after admission	50	100	0	0		
ST segment	Normal		Elevation			
	n	%	n	%		
ST segment on admission	49	98	1	2		
ST segment 24 h after admission	50	100	0	0		
ST segment 72 h after admission	50	100	0	0		
T wave	Normal		Inversion			
	n	%	n	%		
T wave on admission	42	84	8	16		
T wave 24 h after admission	45	90	5	10		
T wave 72 h after admission	46	92	4	8		

Table 3 showing the prevalence rate of sinus arrhythmia, short PR interval, ST segment elevation, and inverted T wave in ECG on admission, 24 h, and 72 h after admission, respectively.

Table 4: Association of serial electrocardiography with electrocardiographic changes in traumatic brain injury study group

Electrocardiographic findings	On admission		24 h after admission		72 h after admission		Chi-square test	
	n	%	n	%	n	%	χ^2	p-value
Heart rate								
Normal	22	44	34	68	41	82	17.337	**0.002***
Bradycardia	9	18	5	10	5	10		
Tachycardia	19	38	11	22	4	8		
Rhythm								
Regular	50	100	50	100	50	100	-	-
PR interval								
Normal	37	74	43	86	48	96	9.695	**0.008***
Short	13	26	7	14	2	4		
QRS complex								
Normal	50	100	50	100	50	100	-	-
QT interval								
Normal	50	100	50	100	50	100	-	-
ST segment								
Elevated	1	2	0	0	0	0	2.013	0.365
Normal	49	98	50	100	50	100		
T wave								
Inverted	8	16	5	10	4	8	1.725	0.422
Normal	42	84	45	90	46	92		

*Significant

Table 4 showing significant association of serial ECG with sinus arrhythmia (χ^2 [1] = 17.337, p = 0.002) and short PR interval (χ^2 [1] = 9.695, p = 0.008), respectively.

Association of ECG changes with certain types of traumatic brain injury

The percent of patients in the TBI cohort who had ECG changes was 74%, while the percent of patients in the TBI cohort who had normal ECG was 26%, respectively. There was a statistically significant association between ECG changes and brain edema (χ^2 [1] = 4.131, p = 0.042), intracerebral hemorrhage

(χ^2 [1] = 4.539, p = 0.033), and subarachnoid hemorrhage groups (χ^2 [1] = 5.889, p = 0.015), respectively (Table 5).

Table 5: Association of electrocardiographic changes with certain types of traumatic brain Injury

Types of traumatic brain injury	ECG findings				Chi-square test	
	Normal (n=13)		Abnormal (n=37)			
	No.	%	No.	%	χ^2	p-value
Brain edema	5	38.46	26	70.27	3.892	**0.021***
Subdural hemorrhage	1	7.69	5	13.51	0.004	0.953
Extradural hemorrhage	4	30.77	15	40.54	0.084	0.772
Intracerebral hemorrhage	4	30.77	24	64.86	3.259	**0.027***
Subarachnoid hemorrhage	3	23.08	23	62.16	4.425	**0.011***
Intraventricular hemorrhage	0	0.00	2	5.41	0.043	0.972
Fracture skull base	5	38.46	15	40.54	0.040	0.842

*Significant

Table 5 showing 70.27% of abnormal ECG findings occur in TBI patients with brain edema, 13.51% of abnormal ECG findings occur in TBI patients with subdural hemorrhage, 40.54% of abnormal ECG findings occur in TBI patients with extradural hemorrhage, 64.86% of abnormal ECG findings occur in TBI patients with intracerebral hemorrhage, 62.16% of abnormal ECG findings occur in TBI patients with subarachnoid hemorrhage, 5.41% of abnormal ECG findings occur in TBI patients with intraventricular hemorrhage, and 40.54% of abnormal ECG findings occur in TBI patients with fracture skull base, respectively.

Discussion

Head trauma and TBI is associated with mortality and morbidity [11]. Post TBI hypotension (systolic blood pressure <90 mmHg) can worsen neurologic and patient outcomes [12]. Autonomic instability leading to cardiac dysfunction has been suggested as a probable mechanism for post TBI hypotension [13], [14]. ECG changes suggestive of autonomic instability and cardiac dysfunction include myocardial ischemic-like changes and repolarization abnormalities [15]. A retrospective cohort study by Fan, *et al*. showed ST-T wave changes in 41.5% of acute brain injury patients [16]. Another retrospective cohort study published in 2014 demonstrated prevalence of ECG changes after head trauma including prolonged PR interval (6.8%), prolonged QT interval (42.4%), Q-wave (6.8%), ST depression (3.4%), ST elevation (3.4%), inverted T-wave (11.9%), and morphologic end-repolarization abnormalities (10.2%), respectively [17]. Our prospective study's results demonstrated prevalence rate of 18% for sinus bradycardia versus 38% for sinus tachycardia, 26% for short PR interval, 2% for ST segment elevation, and 16% for inverted T wave in ECG on admission, 5% for sinus bradycardia versus 22% for sinus tachycardia, 14% for short PR interval, 0% for ST segment elevation, and 10% for inverted T wave in ECG 24 h after admission, and 5% for sinus bradycardia versus 8% for sinus tachycardia, 4% for short PR interval, 0% for ST segment elevation, and 8% for inverted T wave in ECG 72 h after admission, respectively. In addition, our prospective study demonstrated significant association of serial ECG with changes in heart rate (χ^2 [1] = 17.337, p = 0.002) and short PR interval (χ^2 [1] = 9.695, p = 0.008), respectively.

Most of the ECG changes occurring in TBI are related to subarachnoid hemorrhage, intracranial hemorrhage, ischemic stroke, cerebral venous thrombosis, head trauma, neurosurgical procedures, cryohypophysectomy, acute meningitis, intracranial space-occupying tumors, and epilepsy [5], [6]. Our prospective study's results showed that 70.27% of ECG changes occur in TBI patients with brain edema, 13.51% of ECG changes occur in TBI patients with subdural hemorrhage, 40.54% of ECG changes occur in TBI patients with extradural hemorrhage, 64.86% of ECG changes occur in TBI patients with intracerebral hemorrhage, 62.16% of ECG changes occur in TBI patients with subarachnoid hemorrhage, 5.41% of ECG changes occur in TBI patients with intraventricular hemorrhage, and 40.54% of ECG changes occur in TBI patients with fracture skull base, respectively.

Strengths and limitations

Our study did not have missing data allowing robust per protocol analysis and the investigators who analyzed and reported the anonymous ECG changes were blinded to the identity and clinical data of the study participants and hence minimizing observer bias. On the other hand, the study has important limitations. It was a single centered study with a small sample size. Being a short prospective study with a lack of lengthy follow-up did not allow us to investigate the chronological relationship between the ECG changes and the clinically driven outcomes in the TBI patient population.

Conclusions and Recommendations

ECG changes are prevalent in non-cardiac TBI patients. The significant association of serial ECG with changes in heart rate and short PR interval and the significant association of ECG changes with brain edema, intracerebral hemorrhage, and subarachnoid hemorrhage highlights the potential role of ECG as a screening tool for cardiac dysfunction in patients with TBI. Large prospective studies are warranted to correlate ECG changes with cardiovascular clinical outcomes in patients with TBI, especially in patients with brain edema, intracerebral hemorrhage, and subarachnoid hemorrhage.

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