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An Assessment of ECG changes in Cases with Traumatic Brain Injury and their Correlation with Outcome

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ABSTRACT

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality and is a major public health issue. It has been suggested that electrocardiography (ECG) can be used as an inexpensive tool to identify high-risk patients who are at risk of developing cardiac dysfunction following TBI. 15 It has been unclear why ECG changes were, in fact, independently associated with the outcome of brain injuries. In the present article, we report our experience with the incidence of electro cardiographic changes in patients with TBI patients and their correlation with overall outcome. This analytical retrospective study involved Prior Consent from the patients and was found to be within ethical standards. All the patients who were admitted with the diagnosis of TBI under neurosurgery were included in the study. After obtaining the approval from Hospital Authorities / MS, data were retrieved from the case records of the patients in a predesigned pro forma. The details regarding age, gender, any history of hypertension and diabetes mellitus were collected. A total of 100 patients / case sheets were included in the study and the same number of admission ECGs was available for interpretation. The mean age was 36.69 years (minimum 2 years, maximum 74 years, standard deviation 14.72.) Mild head injury was most common (67%) followed by severe (18%) and moderate (15%) head injuries. Most of the patients had serum sodium and potassium in normal range (75% and 64%, respectively). ECG results were normal in 90 patients and were abnormal in 10 patients. Statistical analysis showed that the correlation among severity of the head injury, ECG results and outcome was significant. There was no significant correlation between QTc and outcome and correlation between severity of head injury and outcome. Neurogenic cardiac injury and associated electro cardiographic abnormalities can be associated with increased morbidity and mortality following TBI. The present study highlights the need to recognize the importance of ECG as a simple tool to identify the cardiovascular changes in patients with TBI.

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Key Words
Traumatic brain injury (TBI), ECG, GCS, cardiac injury

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INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality and is a major public health issue[1-4]. Many studies have identified cardiovascular abnormalities (particularly electro cardiographic changes) as a cause of poor outcome particularly in patients with severe brain injury. Electrocardiographic (ECG) abnormalities that occurred along with brain injuries were first described by Bramwell in 1934[1-4]. Since then increasing numbers of studies have focused on investigating the connection between acute brain injury (ABI) and the heart[1-5]. The incidence of ECG changes in these patients have been quoted as ranging from 12-99%[1-6]. Previous studies had described the associations between ECG changes and outcomes after brain injuries, but these studies had many limitations[5-14]. It has been suggested that electrocardiography (ECG) can be used as an inexpensive tool to identify high-risk patients who are at risk of developing cardiac dysfunction following TBI[15]. It has been unclear why ECG changes were, in fact, independently associated with the outcome of brain injuries. In the present article, we report our experience with the incidence of electrocardiographic changes in patients with TBI patients and their correlation with overall outcome.

MATERIALS AND METHODS

This retrospective study involved Prior Consent from the patients and was found to be within ethical standards. It was conducted among patients admitted to or attending to various Cardiac Units and Neurology /Neurosurgery units of local tertiary medical care institutes/hospitals. Data was obtained and studied retrospectively. All the patients who were admitted with the diagnosis of TBI under neurosurgery were included in the study. After obtaining the approval from Hospital Authorities/MS, data were retrieved from the case records of the patients in a predesigned pro forma. The details regarding age, gender, any history of hypertension and diabetes mellitus were collected. Clinical details including pulse, blood pressure, Glasgow coma scale score and serum levels of sodium, potassium, chloride were noted. Twelve lead ECG details for any ECG abnormalities in the form of rhythm abnormalities (atrial fibrillation, premature ventricular contraction, sinus arrhythmia, atrial flutter), conduction abnormalities (left bundle branch block, right bundle branch block, atrioventricular [AV] block), QRS ST complex abnormalities (old myocardial infarction [MI], acute MI, nonspecific ST changes, myocardial ischemia) and QT interval abnormalities (short QT interval, prolonged QT interval) were obtained.

Data was filled in Microsoft Excel and analysed using the Statistical Package for Social Sciences (SPSS) for Windows version 23 and a computer software Epi Info version 6.2 (Atlanta, Georgia, USA).Qualitative variables, expressed as numbers and percents, were compared by the Chi-square test. A p-value less than 0.05 was considered statistically significant.

RESULTS AND DISCUSSIONS

A total of 100 patients/case sheets were included in the study and the same number of admission ECGs was available for interpretation. The mean age was 36.69 years (minimum 2 years, maximum 74 years, standard deviation 14.72). Majority of the patients were young adult males with in the age group of 21-30 years (Table 1). Total 72% of the patients had heart rate in normal range, in 13% patient, it was less than 60 beats/min and in 15% patients, the heart rate was >100 beats/min (Table 2). Mild head injury was most common (67%) followed by severe (18%) and moderate (15%) head injuries (Table 2). Most of the patients had serum sodium and potassium in normal range (75% and 64%, respectively) (Table 3). ECG results were normal in 90 patients and were abnormal in 10 patients. The details are shown in (Table 4). Statistical analysis showed that the correlation among severity of the head injury, ECG results and outcome was significant (Table 5). There was no significant correlation between QTc and outcome and correlation between severity of head injury and outcome (Table 5).

A variety of neurological conditions and intra cranial lesions have been shown to be the cause of cardiac dysfunction and myocardial damage (both in clinical and experimental models) with increased mortality[16-26]. The effect of TBI on cardiovascular functions and its correlation with outcome in humans largely remains unknown and under investigation[27]. Acute brain injury including TBI can activate an intense neuro-inflammatory response, releasing the immunologically active mediators (cytokines, adhesion molecules, and many multifunctional peptides) from brain to the systemic circulation[10,28-32]. This mechanism is a protective phenomenon which is primarily meant for maintenance of cerebral perfusion particularly in the presence of raised intra cranial pressure in severe brain injury patients[7]. In unfavorable circumstances, this response can initiate a systemic inflammatory response syndrome potentially responsible for systemic organ system dysfunction (including cardiac arrhythmias) and multiple organ failure[10,28-32]. This intense systemic response can result in neurogenic stunned myocardium responsible for a reversible neurologically mediated cardiac injury which can be characterized by abnormal ECG changes, cardiac arrhythmias, left ventricular dysfunction and increased serum levels of cardiac bio-markers[7]. Several electro cardiographic abnormalities have been recognized following this intense neuro-inflammatory response in

Table 1: Age and gender distribution of traumatic brain injury patients (n = 100)

	Gender		
Age range (y)	Female	Male	Total
0-10	1	2	03
11-20	3	16	19
21-30	2	22	24
31-40	3	17	20
41-50	5	16	21
51-60	1	09	10
61-70	0	2	02
>71	0	2	01

Table 2: Details of heart rate and severity of the head injury of the patients (n = 100)

Heart rate (per min)	Number of patients (%)
< 60	15%
61–100	72%
> 100	13%
GCS severitya	
Severe head injury (GCS ¼ 3-8)	18%
Moderate head injury (GCS ¼ 9-12)	15%
Mild head injury (GCS ¼ 13-15)	67%

GCS, Glasgow coma scale.

Table 3: Details of the serum sodium and potassium values of the patients (n =100)

Serum electrolytes (mEq/L)	Number of patients (%)
Sodium range	
< 135	10
136-145	75
> 145	7
Missing	8
Potassium range	
< 3.5	18
3.6-4.5	64
> 4.5	10
Missing	08

Table 4: Details of ECG findings and ECG results of the traumatic brain injury patients (n = 100)

ECG findings	Number of patients (%)
Conduction	
Normal	92
Right bundle branch block	04
AV block	01
Other	03
QRS ST complex	
Normal	82
Nonspecific ST changes	10
Old MI	05
Acute MI	01
Other	02
Rhythm	
Normal sinus	96
Sinus tachycardia	04
QTc interval	
Normal	91
Prolonged	09
ECG results	
Normal	90
Abnormal	10

patient with acute brain injury (including TBI). Although the true incidence of the ECG abnormalities is largely unknown, main abnormalities reported are sinus tachycardia, atrial fibrillation, premature atrial and ventricular contractions and AV dissociation[33]. Other ECG abnormalities may include prolongation of the QT interval, ST segment abnormalities, flat or inverted T waves, U waves, peaked T waves, Q waves and widened QRS complex[34-37] Fan *et al*.[38] noted that ST-T changes (41.5%) were the most common ECG abnormality following acute brain injury followed by sinus tachycardia (23.6%). In majority of the cases,

Table 5: Details of correlation among severity of head injury, ECG results, QTc interval and outcome and correlation between severity of head injury and QTc interval

		Outcome			
		Dead	Alive	Total	p-Value[a]
GCS					
severity	3-8	5	13	18	<0.001
	9-12	3	13	16	
	13-15	1	65	66	
ECG					
results	Normal	7	83	90	<0.001
	Abnormal	3	07	10	
QTc interval	Normal	10	80	90	0.32
	Prolonged	02	8	10	
GCS					
severity	3-8	14	3	17	0.14
	9-12	13	2	15	
	13-15	65	3	68	

Abbreviations: ECG, electrocardiography, GCS, Glasgow coma scale.
ap <0.05 was considered significant.

once the management of TBI is instituted and it shows signs of recovery, brain injury-related cardiac dysfunction also show spontaneous resolution[7,29]. The patients with abnormal ECG changes can be followed up at regular intervals[7]. Life-threatening arrhythmias (although uncommon) may need special attention and specific management as if left untreated, these arrhythmias can result in sudden cardiac death[34]. Without detail investigations, it is difficult to implicate TBI as the sole cause of ECG changes and to differentiate a pure neurogenic events from a cardiac events (to exclude coronary artery disease), there shall be a need for further investigations (i.e., coronary angiography) particularly in high-risk group patients for cardiac disease[30]. Although many studies describe the correlation between severity of the brain injury and electro cardiographic changes[36,38] it is unclear whether it is the severity of the brain injury or it is neurogenic cardiac injury which is mainly responsible for poorer outcome[8,14,39]. Gregory and Smith 7 have reported that prolongation of the QTc interval can be a manifestation of neurogenic cardiovascular dysfunction and it is not clear whether it is life threatening on its own or rather it is the severity of the underlying brain injury which is fatal. We also observed prolonged QTc in patients with TBI in our study, however, there was no statistically significant correlation between prolonged QTc and outcome. However, as we observed in the present study that the overall outcome of these patients is determined by the severity of the underlying TBI[3,4,8,9].

CONCLUSION

Neurogenic cardiac injury and associated electro cardiographic abnormalities can be associated with increased morbidity and mortality following TBI. The present study highlights the need to recognize the importance of ECG as a simple tool to identify the cardiovascular changes in patients with TBI. However, there is a need to conduct further prospective studies to supplement these findings with changes in the levels of cardiac enzymes or associated echocardiography

abnormalities and their correlation with ECG findings and overall outcome.

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