Electrocardiographic Changes in Patients with Isolated Traumatic Brain Injury and Their Correlation with Outcome

Veda Dhruthy Samudrala[1] Ashutosh Kumar[2] Amit Agrawal[1]

[1] Department of Neurosurgery, Narayana Medical College & Hospital, Chinthareddypalem, Nellore, Andhra Pradesh, India
[2] Department of Cardiology, Narayana Medical College & Hospital, Chinthareddypalem, Nellore, Andhra Pradesh, India

Address for correspondence Amit Agrawal, MCh, Department of Neurosurgery, Narayana Medical College & Hospital, Chinthareddypalem, Nellore, Andhra Pradesh 524003, India (e-mail: dramitagrawal@gmail.com; dramit_in@yahoo.com).

Indian J Neurotrauma 2016;13:70–74.

Abstract

Objectives Electrocardiography (ECG) can be used as an inexpensive tool to identify high-risk patients who are at risk of developing cardiac dysfunction following traumatic brain injury (TBI). 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 All the patients who were admitted with the diagnosis of TBI under neurosurgery were included in the study. Clinical details and 12 lead ECG details for any ECG abnormalities (rhythm abnormalities, conduction abnormalities, QRS ST complex abnormalities, nonspecific ST changes, QT interval abnormalities, and data regarding outcome) were recorded. The data were entered into a spreadsheet and analyzed using StatsDirect version 3 statistical analysis software. Data were expressed using descriptive statistics—frequency and percentage for categorical variables. Pearson chi-square test was used to identify significance. $p < 0.05$ was considered significant.

Results A total of 109 patients and same number of admission ECGs were available for interpretation. Mild head injury was most common (65.1%) followed by severe (18.3%) and moderate (15.6%) head injuries. ECG results were normal in 97 patients and were abnormal in 12 patients. Statistical analysis showed that the correlation among severity of the head injury, ECG results, and outcome was significant. However, there was no significant correlation between QTc and outcome, and correlation between severity of head injury and outcome.

Conclusion 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.

Keywords
- traumatic brain injury
- cardiac function
- electrocardiography
- QTc interval

received
August 12, 2015
accepted
June 23, 2016
published online
August 10, 2016

DOI http://dx.doi.org/10.1055/s-0036-1586237.
ISSN 0973-0508.

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 electrocardiographic changes) as a cause of poor outcome particularly in patients with severe brain injury.[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] 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

The present retrospective study was conducted at the Departments of Neurosurgery and Cardiology, Narayana Medical College & Hospital, Nellore. All the patients who were admitted with the diagnosis of TBI under neurosurgery were included in the study. After obtaining the institute ethical committee approval, 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.

Statistical Analysis

The data were entered into a spreadsheet and analyzed using StatsDirect version 3 (StatsDirect Ltd., Cheshire, United Kingdom) statistical analysis software. Data were expressed using descriptive statistics—frequency and percentage for categorical variables. Pearson chi-square test was used to identify significance; p-value < 0.05 was considered significant.

Results

A total of 109 patients were included in the study and the same number of admission ECGs was available for interpretation. The mean age was 34.39 years (minimum 3 years, maximum 70 years, standard deviation ± 15.4). Majority of the patients were young adult males (►**Table 1**). Total 72.5% of the patients had heart rate in normal range; in 14.7% patient, it was less than 60 beats/minute and in 12.8% patients, the heart rate was more than 100 beats/minute (►**Table 2**). Mild head injury was most common (65.1%) followed by severe (18.3%) and moderate (15.6%) head injuries (►**Table 2**). Most of the patients had serum sodium and potassium in normal range (73.4 and 59.6%, respectively) (►**Table 3**). ECG results were normal in 97 patients and were abnormal in 12 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**). However, there was no significant correlation between QTc and outcome, and correlation between severity of head injury and outcome (►**Table 5**).

Table 1 Age and gender distribution of traumatic brain injury patients ($n = 109$)

Age range (y)	Gender		Total
	Female	Male	
0–10	1	3	4
11–20	4	18	22
21–30	0	27	27
31–40	3	17	20
41–50	4	16	20
51–60	1	10	11
61–70	0	3	3
> 71	0	2	2

Discussion

A variety of neurological conditions and intracranial 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 neuroinflammatory response, releasing the immunologically active mediators (cytokines, adhesion molecules, and many multifunctional peptides) from brain to the systemic

Table 2 Details of heart rate and severity of the head injury of the patients ($n = 109$)

	Number of patients (%)
Heart rate (per min)	
< 60	16 (14.7)
61–100	79 (72.5)
> 100	14 (12.8)
GCS severity[a]	
Severe head injury (GCS = 3–8)	20(18.3)
Moderate head injury (GCS = 9–12)	17 (15.6)
Mild head injury (GCS = 13–15)	71 (65.1)

Abbreviation: GCS, Glasgow coma scale.
[a]Data for one patient were missing.

Table 3 Details of the serum sodium and potassium values of the patients ($n = 109$)

Serum electrolytes (mEq/L)	Number of patients (%)
Sodium range	
< 135	12 (11)
136–145	80 (73.4)
> 145	8 (7.3)
Missing	9 (8.3)
Potassium range	
< 3.5	21 (19.3)
3.6–4.5	65 (59.6)
> 4.5	13 (11.9)
Missing	10 (9.2)

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 intracranial 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 biomarkers.[7] Several electrocardiographic abnormalities have been recognized following this intense neuroinflammatory response in 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, 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]

Table 4 Details of ECG findings and ECG results of the traumatic brain injury patients ($n = 109$)

ECG findings	Number of patients (%)
Conduction	
Normal	101 (92.7)
Right bundle branch block	4 (3.7)
AV block	1 (0.9)
Other	3 (2.8)
QRS ST complex	
Normal	92 (84.4)
Nonspecific ST changes	9 (8.3)
Old MI	5 (4.6)
Acute MI	1 (0.9)
Other	2 (1.8)
Rhythm	
Normal sinus	106 (97.2)
Sinus tachycardia	3 (2.8)
QTc interval	
Normal	99 (90.8)
Prolonged	10 (9.2)
ECG results	
Normal	97 (89)
Abnormal	12 (11)

Abbreviations: AV, atrioventricular; ECG, electrocardiography; MI, myocardial infarction.

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		Total	*p*-Value[a]
		Dead	Alive		
GCS severity[b]	3–8	8	12	20	0
	9–12	3	14	17	
	13–15	1	70	71	
ECG results	Normal	7	90	97	0
	Abnormal	5	7	12	
QTc interval	Normal	10	89	99	0.341
	Prolonged	2	8	10	
GCS severity	3–8	16	4	20	0.136
	9–12	15	2	17	
	13–15	67	4	71	

Abbreviations: ECG, electrocardiography; GCS, Glasgow coma scale.
[a]$p < 0.05$ was considered significant.
[b]Data for one patient were missing.

Prognosis

Although many studies describe the correlation between severity of the brain injury and electrocardiographic 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 electrocardiographic 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.

References

1 Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. J Head Trauma Rehabil 2006;21(6):544–548
2 Sosin DM, Sniezek JE, Waxweiler RJ. Trends in death associated with traumatic brain injury, 1979 through 1992. Success and failure. JAMA 1995;273(22):1778–1780
3 Agrawal A, Coronado VG, Bell JM, et al. Characteristics of patients who died from traumatic brain injury in two rural hospital emergency departments in Maharashtra, India, 2007-2009. Int J Crit Illn Inj Sci 2014;4(4):293–297
4 Agrawal A, Galwankar S, Kapil V, et al. Epidemiology and clinical characteristics of traumatic brain injuries in a rural setting in Maharashtra, India. 2007-2009. Int J Crit Illn Inj Sci 2012;2(3): 167–171
5 Calvo-Romero JM, Fernández De Soria-Pantoja R, Arrebola-García JD, Gil-Cubero M. [Electrocardiographic abnormalities in subarachnoid hemorrhage]. Rev Neurol 2001;32(6):536–537
6 Hirashima Y, Takashima S, Matsumura N, Kurimoto M, Origasa H, Endo S. Right sylvian fissure subarachnoid hemorrhage has electrocardiographic consequences. Stroke 2001;32(10): 2278–2281
7 Gregory T, Smith M. Cardiovascular complications of brain injury. Contin Educ Anaesth Crit Care Pain 2012;12:67–71
8 van der Bilt IAC, Hasan D, Vandertop WP, et al. Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: a meta-analysis. Neurology 2009;72(7):635–642
9 Zygun D. Non-neurological organ dysfunction in neurocritical care: impact on outcome and etiological considerations. Curr Opin Crit Care 2005;11(2):139–143
10 Schulte Esch J, Murday H, Pfeifer G. Haemodynamic changes in patients with severe head injury. Acta Neurochir (Wien) 1980; 54(3–4):243–250
11 Dash M, Bithal PK, Prabhakar H, Chouhan RS, Mohanty B. ECG changes in pediatric patients with severe head injury. J Neurosurg Anesthesiol 2003;15(3):270–273
12 Bhagat H, Narang R, Sharma D, Dash HH, Chauhan H. ST elevation —an indication of reversible neurogenic myocardial dysfunction in patients with head injury. Ann Card Anaesth 2009;12(2): 149–151
13 Hüttemann E, Schelenz C, Chatzinikolaou K, Reinhart K. Left ventricular dysfunction in lethal severe brain injury: impact of transesophageal echocardiography on patient management. Intensive Care Med 2002;28(8):1084–1088
14 Agrawal A, Reddy GV. Cardiovascular abnormalities in patients with traumatic brain injury. Cardiology Today 2014;XVIII:14–16
15 Krishnamoorthy V, Prathep S, Sharma D, Gibbons E, Vavilala MS. Association between electrocardiographic findings and cardiac dysfunction in adult isolated traumatic brain injury. Indian J Crit Care Med 2014;18(9):570–574
16 Shanlin RJ, Sole MJ, Rahimifar M, Tator CH, Factor SM. Increased intracranial pressure elicits hypertension, increased sympathetic activity, electrocardiographic abnormalities and myocardial damage in rats. J Am Coll Cardiol 1988;12(3):727–736
17 Shivalkar B, Van Loon J, Wieland W, et al. Variable effects of explosive or gradual increase of intracranial pressure on myocardial structure and function. Circulation 1993;87(1):230–239
18 Schrader H, Hall C, Zwetnow NN. Effects of prolonged supratentorial mass expansion on regional blood flow and cardiovascular parameters during the Cushing response. Acta Neurol Scand 1985;72(3):283–294
19 Di Angelantonio E, Fiorelli M, Toni D, et al. Prognostic significance of admission levels of troponin I in patients with acute ischaemic stroke. J Neurol Neurosurg Psychiatry 2005;76(1):76–81
20 James P, Ellis CJ, Whitlock RM, McNeil AR, Henley J, Anderson NE. Relation between troponin T concentration and mortality in patients presenting with an acute stroke: observational study. BMJ 2000;320(7248):1502–1504
21 Song H-S, Back J-H, Jin D-K, et al. Cardiac troponin T elevation after stroke: relationships between elevated serum troponin T, stroke location, and prognosis. J Clin Neurol 2008;4(2):75–83
22 Kolin A, Norris JW. Myocardial damage from acute cerebral lesions. Stroke 1984;15(6):990–993
23 Myers MG, Norris JW, Hachinski VC, Weingert ME, Sole MJ. Cardiac sequelae of acute stroke. Stroke 1982;13(6):838–842
24 Parr MJ, Finfer SR, Morgan MK. Reversible cardiogenic shock complicating subarachnoid haemorrhage. BMJ 1996;313(7058): 681–683
25 Dupuis M, van Rijckevorsel K, Evrard F, Dubuisson N, Dupuis F, Van Robays P. Takotsubo syndrome (TKS): a possible mechanism of sudden unexplained death in epilepsy (SUDEP). Seizure 2012; 21(1):51–54
26 Wittstein IS, Thiemann DR, Lima JAC, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005;352(6):539–548
27 Prathep S, Sharma D, Hallman M, et al. Preliminary report on cardiac dysfunction after isolated traumatic brain injury. Crit Care Med 2014;42(1):142–147
28 Frangiskakis JM, Hravnak M, Crago EA, et al. Ventricular arrhythmia risk after subarachnoid hemorrhage. Neurocrit Care 2009;10(3):287–294
29 Lim HB, Smith M. Systemic complications after head injury: a clinical review. Anaesthesia 2007;62(5):474–482
30 Nguyen H, Zaroff JG. Neurogenic stunned myocardium. Curr Neurol Neurosci Rep 2009;9(6):486–491
31 Tung P, Kopelnik A, Banki N, et al. Predictors of neurocardiogenic injury after subarachnoid hemorrhage. Stroke 2004;35(2): 548–551

32 Nguyen M-D, Giridharan G, Prabhu SD, Sethu P. Microfluidic cardiac circulation model (microCCM) for functional cardiomyocyte studies. Conference proceedings: Annual International Conference of the IEEE Engineering in Medicine and Biology Society. IEEE Engineering in Medicine and Biology Society. Annual Conference; 2009: 1060–1063
33 Macmillan CSA, Grant IS, Andrews PJD. Pulmonary and cardiac sequelae of subarachnoid haemorrhage: time for active management? Intensive Care Med 2002;28(8):1012–1023
34 Chang PC, Lee SH, Hung HF, Kaun P, Cheng JJ. Transient ST elevation and left ventricular asynergy associated with normal coronary artery and Tc-99m PYP myocardial infarct scan in subarachnoid hemorrhage. Int J Cardiol 1998;63(2): 189–192
35 Jachuck SJ, Ramani PS, Clark F, Kalbag RM. Electrocardiographic abnormalities associated with raised intracranial pressure. BMJ 1975;1(5952):242–244
36 Collier BR, Miller SL, Kramer GS, Balon JA, Gonzalez LS III. Traumatic subarachnoid hemorrhage and QTc prolongation. J Neurosurg Anesthesiol 2004;16(3):196–200
37 Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. J Am Coll Cardiol 1994;24(3):636–640
38 Fan X, Du FH, Tian JP. The electrocardiographic changes in acute brain injury patients. Chin Med J (Engl) 2012;125(19):3430–3433
39 Hersch C. Electrocardiographic changes in head injuries. Circulation 1961;23:853–860