

ORIGINAL RESEARCH

ECG CHANGES AND CORRELATION WITH CARDIAC BIOMARKERS & ECHOCARDIOGRAPHY IN PATIENTS WITH TRAUMATIC BRAIN INJURY AT A TERTIARY CARE HOSPITAL IN NORTH INDIA

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Abstract:

Background: ECG changes are frequently seen in patients with brain injury. Cerebrovascular accidents are frequently associated with coronary artery disease and as a result ECG changes can be expected more frequently in such patients. However traumatic brain injury (TBI) is unrelated to atherosclerosis. This study was planned to study the ECG changes in TBI and to look for any associated changes in cardiac biomarkers and echocardiography in such patients.

Methods: A prospective analysis was done in 350 patients of TBI who were admitted in the ICU of Sharda Hospital, Greater Noida. ECG was done in all patients. In all the patients with abnormal ECG changes, quantitative Troponin I was estimation was done along with an echocardiography.

Results: ECG changes were found in more than 70% of patients. Cardiac biomarkers in the form of raised Troponin I were also elevated in more than 85% of the patients who had ECG changes. ST depression and persistent tachycardia were the most common ECG findings. Echocardiographic abnormalities were present in ~25% of patients with ECG changes. Most patients with echocardiographic abnormalities had moderate to severe TBI. Severe echo changes were almost exclusively seen in patients with severe TBI.

Conclusions: ECG changes are frequently associated with TBI. Troponin I levels are also frequently elevated in patients with ECG changes. However, echocardiography changes are not as common as a rise in cardiac biomarkers and are predominantly seen in elderly patients and in those with very high cardiac biomarker levels. Hence all patients with TBI should be evaluated with a baseline ECG and further investigations if the ECG is abnormal.

Key words: Cerebrovascular accidents, Echocardiography, Traumatic brain injury

Introduction:

Traumatic brain injury (TBI) is a major public health concern and a leading cause of traumatic death worldwide. Complications after TBI known as second insults can worsen neurologic and patient outcomes.¹ Clinically, post-TBI hypotension (systolic blood pressure <90 mmHg) has been directly linked to mortality and poor disposition. While the majority of patients experience a hypotensive episode during TBI surgery, the cause is often unknown, and treatment may be empiric. Cardiac dysfunction has been documented in TBI, and implicated as a cause for hypotension in other brain injury paradigms, where experimental evidence postulates that brain-heart-lung interactions may cause myocardial dysfunction in TBI.²

The most common non-neurologic site involved are the respiratory system followed by the cardiovascular system.³ The cardiovascular complications varied from extreme swings in blood pressure, repolarization abnormalities such as corrected QT (QTc) prolongation, ST-T wave changes and arrhythmias on electrocardiogram (ECG), the release of biomarkers of cardiac injury due to clinically occult myocardial necrosis to overt ventricular dysfunction on echocardiography. This is often associated with increased morbidity and mortality. The cause could be attributed to a surge in circulating catecholamines and a global inflammatory state.⁴ Head injury-related ECG abnormalities commonly include ST-segment changes, flat/inverted T waves, prominent U waves, and prolonged QTc interval. Repolarization abnormalities and QT prolongation were found to be independent prognostic factors for negative outcomes.⁵ Echocardiographic changes such as impairment in left ventricular contractile function, hypokinesia, and reduced ventricular ejection fraction (EF), and regional wall motion abnormality (Regional wall motion abnormalities [RWMA]) were seen in the setting of isolated TBI and were associated with increased in-hospital mortality.⁶ This study assessed ECG changes and correlation with cardiac biomarkers & echocardiography in patients with traumatic brain injury.

Materials & methods:

The present study comprised of 350 patients with traumatic brain injury reported to tertiary care hospital. It comprised of 252 males and 98 females. The approval for the study was obtained from ethical clearance committee. Written consent from relatives of patients was obtained.

Demographic profile such as name, age, gender etc. was recorded. GCS score was recorded as mild (Glasgow Coma Scale [GCS] 13-15), moderate (GCS 9-12) and severe (GCS 3-8). All patients underwent CT head examination. Changes in ECG and cardiac biomarker was recorded. Patients were managed with invasive intracranial pressure monitoring, maintaining intracranial pressure <20 mmHg, minimum cerebral perfusion pressure (CPP) of 50 mmHg, PaCO₂ 35-40 mmHg, SaO₂ >90% and maintaining core body temperature between 35°C and 37.5°C with antipyretics, cooling/warming blankets, or intravascular cooling devices if needed. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

Results:

Table I Baseline characteristics

Characteristic	Number (n=350)	P value
Age (mean)	38	
Gender		
Male	252 (72%)	0.01
Female	98 (28%)	

Table I shows that mean age was 38 years. Out of 350 patients, males were 252 (72%) and females were 98 (28%). The difference was significant (P< 0.05).

Table II GCS score at admission

GCS score at admission	Number (%)	P value
Mild TBI (13-15)	98 (28%)	0.05
Moderate TBI (9-12)	140 (40%)	
Severe TBI (3-8)	112 (32%)	

Table II, graph I shows that GCS was mild TBI in 98 (28%), moderate TBI in 140 (40%) and severe TBI in 112 (32%). The difference was significant (P< 0.05).

Graph IGCS score at admission

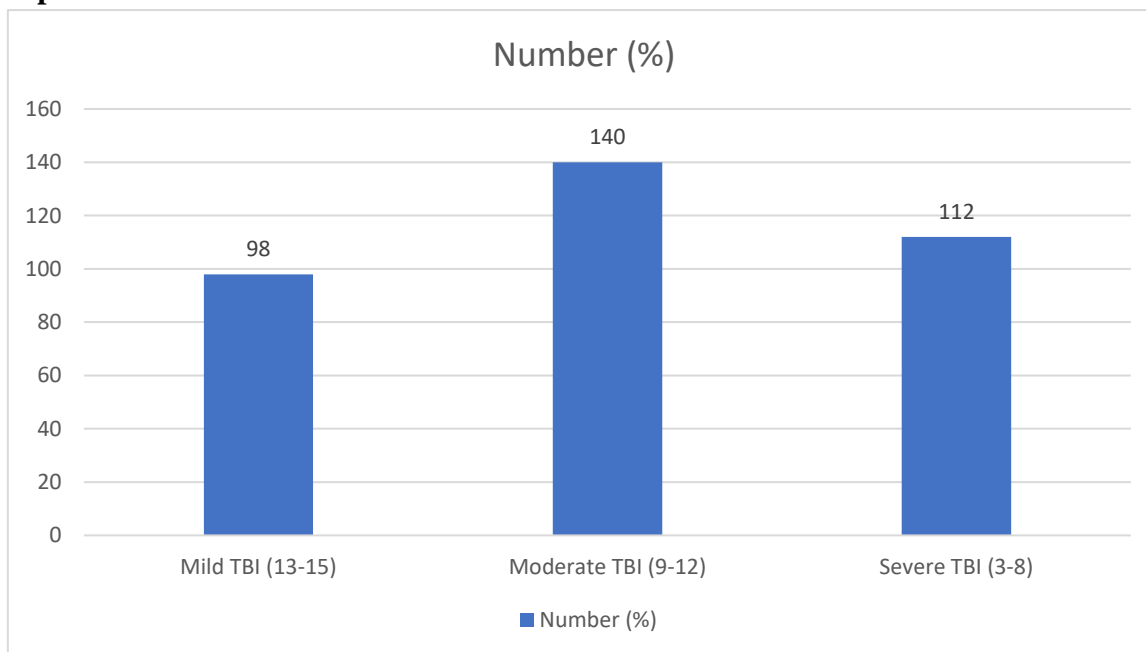


Table III Assessment of CT findings

CT findings	Number (%)	P value
Subdural hematoma	161 (46%)	0.04
Intraparenchymal hemorrhage	70 (20%)	
Subarachnoid hemorrhage	63 (18%)	
Contusions	49 (14%)	
Others	7 (2%)	

Table III, graph II shows that CT findings showed subdural hematoma in 161 (46%), intraparenchymal hemorrhage in 70 (20%), subarachnoid hemorrhage in 63 (18%) contusions in 49 (14%) and others in 7 (2%). The difference was significant ($P < 0.05$).

Graph II Assessment of CT findings

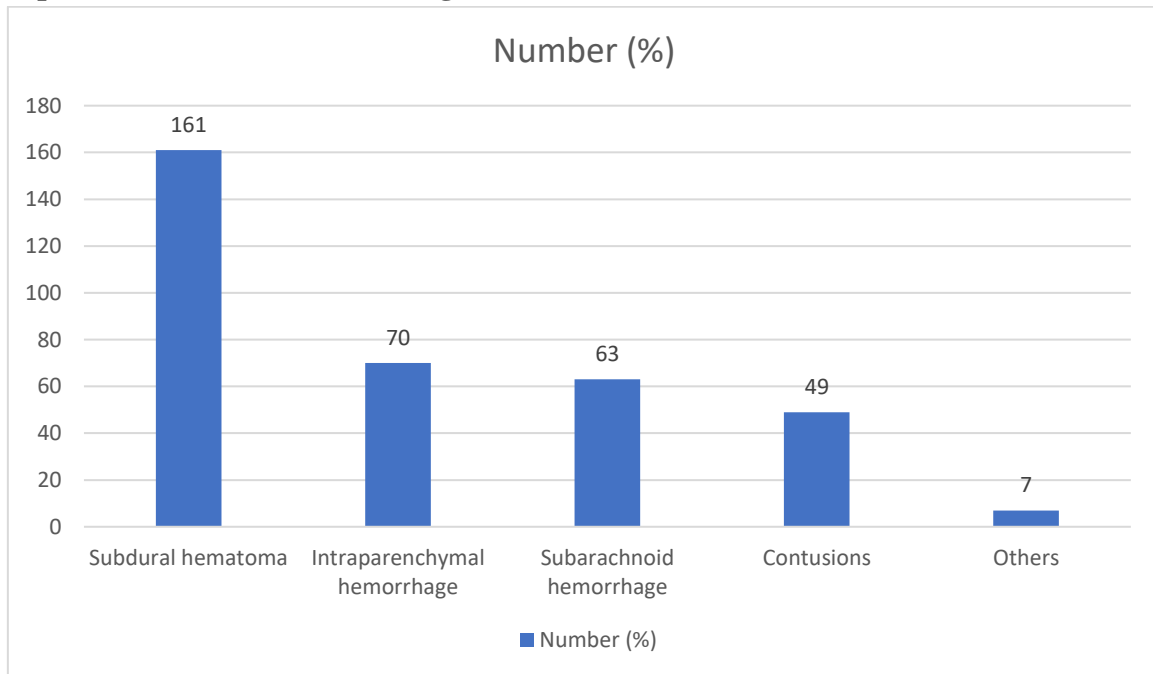


Table IV ECG changes

Characteristics	Number (n=252)	P value
ST depression	146 (57.9%)	0.01
QTc prolongation	106 (42%)	
Persistent tachycardia	96 (38.1%)	
Persistent bradycardia	38 (15.1%)	
T wave inversion	71 (28.2%)	
ST elevation	2 (0.8%)	
Troponin I elevated	215 (85.3%)	

Table IV shows that ECG changes was observed in 252 patients. ST depression in 146 (57.9%), QTc prolongation in 106 (42%), persistent tachycardia in 96 (38.1%), persistent bradycardia in 38 (15.1%), T wave inversion in 71 (28.2%) and ST elevation in 2 (0.8%). Troponin I elevation was seen in 215 (85.3%). The difference was significant ($P < 0.05$).

Table V Echocardiographic changes

Characteristics	N=252
Echo findings	
Normal study	191(75.7%)
LVEF 40-55%	42 (16.7%)
LVEF 30-40%	9 (3.5%)
LVEF <30%	10 (3.9%)

Table V shows that LVEF 40-55% was seen in 42 (16.7%), LVEF 30-40% in 9 (3.5%) and LVEF <30% in 10 (3.9%).

Table VI ECG and echo cardiography changes relation with severity of TBI

	Mild TBI (n=98)	Moderate TBI (n=140)	Severe TBI (n=112)
ECG changes (n=252)	46 (46.9%)	116 (82.8%)	90 (80.3%)
Raised Troponin I (n=215)	29	101	85
Echo findings			
Normal study	44	97	48
LVEF 40-55%	2	17	25
LVEF 30-40%	0	2	7
LVEF <30%	0	0	10

Table VI shows that more ECG and echo cardiography changes with severity of TBI.

Discussion:

Acute insults to the brain during aneurysmal subarachnoid hemorrhage (aSAH), traumatic brain injury (TBI), and stroke have shown organ dysfunction extraneous to the central nervous system.⁷The overwhelming focus has been on the clinical model of aSAH, electrocardiographic changes, and echocardiographic abnormalities in the literature. In contrast to the diverse literature on cardiopulmonary abnormalities associated with aSAH, there is scarce literature on the exact incidence of these complications after TBI.⁸Only recently the presence of such cardiac dysfunction in TBI has been described in a few reports and studies.

We found that mean age was 38 years. Out of 350 patients, males were 252 (72%) and females were 98 (28%). GCS was mild TBI in 98 (28%), moderate TBI in 140 (40%) and severe TBI in 112 (32%). Krishnamoorthy et al⁹examined abnormal ECG findings after isolated TBI and their association with true cardiac dysfunction, based on echocardiogram. Data from adult patients with isolated TBI between 2003 and 2010 was retrospectively examined. Inclusion criteria included the presence of a 12-lead ECG within 24 h of admission and a formal echocardiographic examination within 72 h of admission after TBI. Patients with preexisting cardiac disease were excluded. Baseline clinical characteristics, 12-lead ECG, and echocardiogram report were abstracted. Logistic regression was used to identify the relationship of specific ECG abnormalities with cardiac dysfunction. 13 (22%) patients had tachycardia (heart rate >100 bpm), 25 (42.4%) patients had a prolonged QTc, and 6 (10.2%) patients had morphologic end-repolarization abnormalities (MERA), with each having an association with abnormal echocardiographic findings: Odds ratios (and 95% confidence intervals) were 4.14 (1.02-17.05), 9.0 (1.74-46.65), and 5.63 (1.96-32.94), respectively. Ischemic-like ECG changes were not associated with echocardiographic abnormalities.

We found that CT findings showed subdural hematoma in 161 (46%), intraparenchymal hemorrhage in 70 (20%), subarachnoid hemorrhage in 63 (18%) contusions in 49 (14%) and others in 7 (2%). ECG changes was observed in 252 patients. ST depression in 146 (57.9%), QTc prolongation in 106 (42%), persistent tachycardia in 96 (38.1%), persistent bradycardia

in 38 (15.1%), T wave inversion in 71 (28.2%) and ST elevation in 2 (0.8%). Troponin I elevation was seen in 215 (85.3%). Praveen et al¹⁰ evaluated the perioperative cardiac functions in patients with TBI in 60 consecutive adult patients of either sex between the age of 10 and 70 years with an isolated head injury who were taken up for decompressive craniectomy as per institutional protocol. ECG and transthoracic echocardiography was carried out preoperatively and then postoperatively within 24–48 hours. The mean age of our study population was 39 + 13 years with a median Glasgow coma score of 11. A majority (73%) of our patients suffered moderate TBI. Preoperatively, ECG changes were seen in 48.33% of patients. Postoperatively, ECG changes declined and were seen only in 13.33% of the total patients. Similarly, echocardiography demonstrated preoperative systolic dysfunction in 13.33% of the total study population. Later, it was found that systolic function significantly improved in all the patients after surgery.

We observed that LVEF 40-55% was seen in 42 (16.7%), LVEF 30-40% in 9 (3.5%) and LVEF <30% in 10 (3.9%). Fan et al¹¹ showed that acute brain injury leads to myocardial damage and ECG changes (73.4%), and these changes had a significant association with the severity of TBI. Others have shown that ECG changes do occur in children with a head injury. In a retrospective review from a Level 1 regional trauma center about 22% of patients have echocardiographic abnormalities.¹²

Conclusion

Authors found that ECG changes are frequently associated with TBI. Troponin I levels are also frequently elevated in patients with ECG changes. However, echocardiography changes are not as common as a rise in cardiac biomarkers and are predominantly seen in elderly patients and in those with very high cardiac biomarker levels. Hence all patients with TBI should be evaluated with a baseline ECG and further investigations if the ECG is abnormal.

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