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Correlation of head injury with ECG and echo changes

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ABSTRACT

Background: Abnormal electrocardiogram (ECG) findings can be seen in traumatic brain injury (TBI) patients. ECG may be an inexpensive tool to identify patients at high risk for developing cardiac dysfunction after TBI. This study aimed to examine abnormal ECG findings after isolated TBI and their association with true cardiac dysfunction based on echocardiogram.

Methods: This prospective observational study examined the data from adult patients with isolated and non-operated TBI between 2020 and 2021. Patients aged <18 years and >65 years with and presence of extracranial injuries including orthopedic, chest, cardiac, abdominal, and pelvis, pre-existing cardiac disease, patients who have undergone cardiothoracic surgery, with inotrope drugs, acute hemorrhage, and brain death were excluded from the study.

Results: We examined data from 100 patients with isolated TBI who underwent ECG and echocardiographic evaluation. ECG changes among 53% of mild cases showed a heart rate of 60–100/min, and 2% of cases showed more than 100/min. Prolonged pulse rate (PR) interval was observed in 8%, 11%, and 16% of mild, moderate, and severe cases, while no changes in PR interval were observed in 65% of cases. A prolonged QRS pattern was observed in 5%, 7%, and 15% of mild, moderate, and severe cases. A normal QRS complex was observed in 71% of cases. Prolonged QTc was observed in 3%, 10%, and 15% of cases in mild, moderate, and severe cases, respectively.

Conclusion: Repolarization abnormalities, but not ischemic-like ECG changes, are associated with cardiac dysfunction after isolated TBI. 12-lead ECG may be an inexpensive screening tool to evaluate isolated TBI patients for cardiac dysfunction.

Keywords: Cardiac function, Echocardiography, Electrocardiography, Traumatic brain injury

INTRODUCTION

Traumatic brain injury (TBI) is one of the major public health concerns and a leading cause of traumatic death all over the world.[23] Globally, TBI affects approximately about 1.8 million individuals[21] and is also a primary cause of brain death in intensive care units (ICUs).[24] Complications after TBI, referred to as second insults, can worsen neurologic as well as the outcome of the patient.[2,9] Post-TBI hypotension (systolic blood pressure [BP] <90 mmHg) has been associated directly with poor prognosis as well as mortality.[20,32] During TBI surgery, the majority of patients have a hypotensive episode.[28] The cause for this episode is unknown,

and treatment may be empirical. Cardiac dysfunction has been established in TBIs and has been linked as a source of hypotension in other brain injury paradigms, where experimental data suggest that brain-heart-lung interactions may produce myocardial dysfunction in TBIs.[6,26] This indicates that cardiac dysfunction may be one of the reasons for post-TBI hypotension. Brain injury, that is, TBI and subarachnoid hemorrhage (SAH), can initiate a strong neuroinflammatory response, which leads to the release of many immunologically active mediators into the systemic circulation.[10] This neuroinflammatory modulator activates a systemic inflammatory response syndrome, causing systemic organ system dysfunctions and ventricular arrhythmias after TBI.[10,16,30,34]

Although most of these abnormalities cause minimal clinical effects are reversible, and the management is mainly generalized supportive care and the treatment of the underlying brain injury. However, in severe cases, these can lead to pulmonary edema as well as cardiogenic shock.[27] These post-TBIs with cardiovascular abnormalities also have been recognized as an independent predictor of morbidity and mortality in these cases.[34] The spectrum of post-TBI cardiovascular abnormalities includes changes in hemodynamic pattern (hypertension and hypotension), arrhythmias, ECG changes, the release of many biomarkers (creatine phosphokinase-myocardial band CPK-MB, troponin-1, and brain natriuretic peptide), and ventricular dysfunction.[6,15] The exact incidence of arrhythmias following brain injury is not known, which includes premature atrial and ventricular contractions, sinus tachycardia, atrial fibrillation, and atrioventricular (AV) dissociation.[16] Electrocardiographic abnormalities have also been linked to neurologic hemorrhage, with ischemic-like changes as well as a variety of repolarization abnormalities.[4,10] In small series, children and adults with TBI have been characterized as autonomic instability, with variations in heart rate variability considered to be secondary to sympathetic over-activity and autonomic imbalance.[1,15] In addition to repolarization abnormalities as well as ischemic-like ECG changes, variability of heart rate may be lowered in patients with brain injuries, indicating dysfunction in the autonomic nervous system.[1,15] Hence, before ordering highly expensive and potentially more invasive tests, early ECG may serve as an inexpensive tool in screening cardiac dysfunction. With this background, this study aimed to assess the abnormalities in ECG findings after isolated TBI and their correlation with the incidence and impact of cardiac dysfunction.

MATERIALS AND METHODS

This prospective observational study was performed in the Department of Neurosurgery and Cardiology, Nizams Institute of Medical Sciences (NIMS), Hyderabad. After the approval from the NIMS Institutional Ethical Committee, study-related data were extracted, which includes complete admission and hospitalization records for all patients with a traumatic injury from the institution's trauma registry. The study was conducted between February 2020 and November 2021. Inclusion criteria included all adults aged 18–65 years, patients with isolated, and non-opened TBI. Patient age <18 years and >65 years and presence of extracranial injuries including cardiac, orthopedic, chest, abdominal, and pelvis, pre-existing cardiac disease (defined as documented preadmission untreated hypertension, heart failure, myocardial ischemia/infarction, arrhythmia, and cardiac pacemaker), patients who undergone cardiothoracic surgery, with inotrope drugs, acute hemorrhage, and brain death were excluded from the study. Demographic data were collected from the patient's case records in a pre-designed pro forma.

TBI was categorized based on Glasgow Coma scale (GCS) [25] as mild (13-15), moderate (9-12),severe (3-8). TBI was also categorized by head abbreviated injury scale (AIS): 1 = minor, 2 = moderate, 3 = serious, 4 = severe, 5 = critical, and 6 = not survivable,[23] as previously described. During the study period, patients were resuscitated according to institutional practice, consistent with the Brain Trauma Foundation guidelines.[23,24] Relevant to this study, practices included invasive intracranial pressure (ICP) monitoring, maintaining ICP for 90%, and maintaining core body temperature between 35°C and 37.5°C with antipyretics, cooling/warming blankets, or intravascular cooling devices if needed. Practices involving requests for echocardiography were not standardized during the time of this study, and echocardiography was requested at the discretion of the primary attending intensivist. All the patients were examined for pallor, icterus, cyanosis, clubbing, koilonychia, and lymphadenopathy. Pulse rate (PR), respiratory rate, GCS score, and BP monitoring were recorded regularly. Systolic and diastolic BP was measured in all of the subjects. The diagnosis of hypertension was based on a systolic BP of 140 mmHg or higher and a diastolic BP of 90 mmHg or higher. The pathological investigations, namely, hemoglobin, complete blood count, hemogram, and complete urine examination, were performed. The biochemical investigations, namely, serum blood urea, serum creatinine, serum electrolytes, serum calcium, and serum phosphorus, were investigated in a semi-automated analyzer. A 12-lead electrocardiogram (ECG) was performed within 24 hours of admission, and a blinded cardiologist interpreted the ECG findings. 2D Echo was done within 72 hours of admission. The parameters – chamber size, systolic function, diastolic function, left ventricular (LV) wall motion abnormalities, pericardial effusion, valvular abnormalities, and radiological imaging were examined. We also examined and noted the presence of cerebral contusion, intraventricular and

epidural hemorrhage, and its volume, which was measured as the largest width on axial cuts. We recorded the presence of high ICP, the period that the pressure was high, and its highest levels. The Marshall grade was measured by using the computed tomography (CT) scan's visible pathologies (e.g., edema, focal lesions, and intracranial hemorrhage). Any abnormal changes in ECG, any abnormal changes in echocardiogram, and any abnormal changes in cardiac biomarkers (CPK-MB, Troponin T [TROP T], and Troponin I [TROP I]) and in-hospital mortality were recorded.

Statistical analysis

The data were imported into MS Excel 2016 and analyzed using descriptive statistics – frequency and percentage for categorical variables – in the Statistical Package for the Social Sciences v 21 software. The Pearson Chi-square test was used to determine significance, and for pairwise comparison of categorical data (baseline compared with follow-up), the McNemar test was applied. $P < 0.05$ was considered statistically significant. Binary logistic regression was applied to assess the risk factors predicting the mortality (outcome), and only the significant ($P < 0.05$) factors were assessed to formulate the final model (outcome versus abnormal echocardiography).

RESULTS

We evaluated data of 100 patients with isolated TBI whose case records having an ECG within 24 h of admission and at least one echocardiography report within 72 h of admission.

Age and gender distribution

Age ranged from 18 to 65 years. The mean age of presentation to our institute was 43.88 ± 15.96 years. The largest cohort was between 41 and 50 years (n = 21, 21%), followed by 31–40 years (n = 20, 20%), 51–60 years (n = 19, 19%), the least number of patients were in 18–20 years (n = 10, 10%), and youngest and oldest patient were 18 years and 65 years, respectively. There were 86 males and 14 females in our study, and the male-to-female ratio was 6.1:1.

Distribution of head injury and its type based on GCS

The majority of the patients were mild (55%), moderate constituted 21% (21/100), while severe head injury was noted in 24% of cases (24/100). In our study, subdural hematoma (SDH) was the most common injury noted, which constituted 45% (45/100), followed by contusion in 20% (20/100) cases, extradural hematoma in 17% (17/100 cases), diffuse axonal injury (DAI) in 10% (10/100), SAH occupied 10% (10/100), and intraparenchymal hemorrhage was noted in 3% cases (03/100).

CT findings and heart rate of head injury patients

Based on CT findings, there was no compression on perimesencephalic cisterns in 24 cases, compression on cistern was noted in 40 cases, Midline shift (MLS) >5 mm was noted in 36 cases. ECG changes among 53% of mild cases showed a heart rate of 60–100/min, and 2% of cases showed more than 100/min. Among moderate cases, 16% of cases showed a heart rate of 60–100/min, and 5% showed more than 100/min. Among severe cases, 13% of cases showed a heart rate of 60–100/min, and 11% of cases showed more than 100/min.

Distribution of sinus rhythm rate, prolonged PR interval, prolonged QRS, and prolonged QTc

Only one mild case, three cases among moderate, and ten cases among severe cases showed abnormal sinus rhythm rate, and the left bundle branch block was the most common abnormality observed. A prolonged PR interval was observed in 8%, 11%, and 16% of mild, moderate, and severe cases, whereas no changes in PR interval were observed in 65% of cases. A prolonged QRS pattern was observed in 5%, 7%, and 15% of mild, moderate, and severe cases, respectively. A normal QRS complex was noted in 71% of cases. Prolonged QTc was noted in 3%, 10%, and 15% of cases in mild, moderate, and severe cases, respectively.

Distribution of left ventricular hypertrophy (LVH), ST-segment elevation, ST-segment depression, Q wave, and T wave among admitted cases

LVH distribution was observed in 15% of severe cases, 13% and 4% of moderate and mild cases, and ST-segment elevation was observed in 3%, 7%, and 12% of mild, moderate, and severe cases, whereas ST-segment depression was observed in 14% of severe cases, 9% and 5% of moderate and mild cases. Q wave distribution was observed in 5%, 8%, and 7% of mild, moderate, and severe cases, and T wave inversion was observed in 21%, 11%, and 12% of mild, moderate, and severe cases, respectively.

Distribution of ejection fraction, right and left dilation, aortic valve involvement, and mitral valve involvement among admitted cases

An ejection fraction of 55–70% was observed in 21% of severe cases, while 54% and 21% were mild and moderate cases. Right ventricle dilatation was observed in only 1% of severe cases, while 7% and 2% of mild and moderate cases. Left ventricle dilatation was found in only 1% of mild, moderate, and severe cases. Whereas aortic valve involvement was observed in 9% of severe cases, while 1% and 4% of mild and moderate cases. Mitral valve

involvement was found in 2% of severe cases, 25%, and 8% in mild and moderate cases.

Cardiac enzyme changes observed in head injury cases at admission (baseline) and management of head injury cases

At admission, elevated cardiac enzyme CPK-MB was observed in 12%, 5%, and 10% of mild, moderate, and severe cases, and a total of 27 cases had elevated CPK-MB at admission. TROP T was positive at admission and was observed in 2%, 3%, and 4% of mild, moderate, and severe cases. A total of nine cases had positive TROP T at admission. Elevated TROP I was observed in 39%, 17%, and 21% of mild, moderate, and severe cases, and a total of 74 patients had elevated TROP I valve at admission.

Intraoperative cardiac ECG changes, frequencies of intraoperative ECG changes, and mortality distribution among head injury patients

ST-segment depression was observed in 10% of severe cases, while 13% and 4% in mild and moderate cases. Inversion of T wave was observed in 14%, 7%, and 9% of mild, moderate, and severe cases. Improved QTc was observed in 2% of mild cases among 3% of preoperative cases, 1% of moderate cases among 11% of preoperative cases, and 4% of severe cases among 17% of preoperative cases. In our study, a total of 94 patients survived till the final follow-up, and in-hospital mortality was 6%. Persistent, prolonged QTc was observed in all mortality cases.

Distribution of ECG changes at follow-up

On follow-up, ST depression was persistent in 6% of severe cases, while 1% and 3% in mild and moderate cases, respectively. Inversion of the T wave was persistent in 2% of mild and moderate cases, respectively, and 4% in severe cases. Persistent QTc was observed in 1% of mild and 5% of moderate and severe cases, respectively.

ECG findings at admission and follow-up in different neurotrauma severity groups regional wall motion abnormality (RWMA)

In mild cases at admission, RWMA was noted in 11% of cases, and at follow-up, 4% of cases showed RWMA. In moderate cases, RWMA was noted in 6% of cases at admission and 4% of cases at follow-up. In severe cases at admission and at follow-up, 6% of cases showed RWMA.

Diastolic dysfunction (DD)

In mild cases, DD was noted in 20% of cases at admission, and at follow-up, 10% of cases showed DD. In moderate cases, DD was noted in 9% of cases at admission and 5% at follow-up. Whereas in severe cases at admission, DD was noted in 13% of cases, 10% of cases showed DD, and six deaths were recorded at follow-up.

Valvular dysfunction

In mild cases, valvular dysfunction was noted in 10% of cases at admission, and at follow-up, 5% of cases showed valvular dysfunction. In moderate cases at admission, valvular dysfunction was noted in 14% of cases, and at follow-up, 8% of cases showed valvular dysfunction. In severe cases at admission, valvular dysfunction was noted in 15% of cases, 12% of cases showed valvular dysfunction, and six deaths were noted at follow-up.

ECG findings among different head injury groups at admission (baseline) and follow-up

PR interval

In mild cases, prolonged PR interval at admission was noted in only 8% of cases. Intraoperatively, all 55% showed normal PR interval, and at follow-up, prolonged PR interval was noted in 3% of cases. In moderate cases, prolonged PR interval at admission was noted in 13% of cases. Intraoperatively and at follow-up, all 21 cases showed normal PR interval. Whereas in severe cases, prolonged PR interval at admission was noted in 18% of cases. Intraoperatively, all 24 cases showed normal PR interval, and at follow-up, six cases showed prolonged PR interval and 12 cases were normal.

QRS

In mild cases, prolonged QRS at admission was noted in only 5% of cases. Intraoperatively, all 55% showed normal QRS, and at follow-up, prolonged QRS was noted in 2% of cases. In moderate cases, prolonged QRS at admission was noted in only 7% of cases intraoperatively, and at follow-up, all 21 cases showed normal QRS. In severe cases, prolonged QRS at admission was noted in 17% of cases; intraoperatively, all 24 cases showed normal QRS, and at follow-up, five cases showed prolonged QRS.

QTc

In mild cases at admission, prolonged QTc was noted in 3% of cases; intraoperatively, only one case showed prolonged QTc, and at follow-up, all 55 cases showed normal QTc. In moderate cases at admission, prolonged QTc was noted in 11% cases; intraoperatively, only one case showed prolonged QTc, and at follow-up, five cases showed normal QTc. In severe cases at admission, prolonged QTc was noted in 17% of cases; intraoperatively, four cases showed prolonged QTc, and at follow-up, seven cases showed prolonged QTc.

ST-segment depression

In mild cases at admission, ST depression was observed in 5% of cases; intraoperatively, it was detected in 11 cases, and at follow-up, only three cases showed ST depression. In moderate cases at admission, ST depression was noted in 9% of cases; intraoperatively, it was observed in four cases, and at follow-up, only one case showed ST depression. In severe cases at admission, ST depression was noted in 16% of cases; intraoperatively, it was observed in ten cases, and at follow-up, six cases showed ST depression.

Segment elevation

In mild cases at admission, ST elevation was noted in 3% of cases intraoperatively, and at follow-up, it was not noted in any case. In moderate cases at admission, ST elevation was noted in 7% of cases; intraoperatively, it was not observed in any case, and at follow-up, ST elevation was noted in 3% of cases. In severe cases, at admission, ST elevation was noted in 14% of cases; intraoperatively, it was not observed in any case, and at follow-up, ST elevation was noted in 3% of cases.

T wave inversion

In mild cases, at admission, T wave inversion was noted in 22% of cases; intraoperatively, it was noted in 14% of cases, and at follow-up, it was noted in only 3% of cases. In moderate cases, at admission, T wave inversion was noted in 11% of cases; intraoperatively, it was noted in 7% of cases, and at follow-up, it was noted in only 2% of cases. In severe cases, at admission, T wave inversion was noted in 12% of cases; intraoperatively, it was noted in 9% of cases, and at follow-up, it was noted in only 4% of cases.

Distribution of cardiac enzymes at admission and follow-up

CPK-MB

In mild cases, at admission, elevated CPK-MB was found in 9% of cases, and at follow-up, 3% of cases showed elevated CPK-MB. In moderate cases, at admission, elevated CPK-MB in 5% of cases, and at follow-up, 4% of cases showed elevated CPK-MB. In severe cases, at admission, elevated CPK-MB in 10% of cases, and at follow-up, 6% of cases showed elevated CPK-MB.

TROP T

In mild cases, at admission, elevated TROP T was noted in only 2% of cases, and at follow-up, elevated TROP T was not observed. In moderate cases, at admission, elevated TROP T was observed in 3% of cases, and at follow-up, elevated TROP T was not observed. In severe cases, elevated TROP T was noted in 3% of cases, and at follow-up, elevated TROP T was noted in 1% of cases.

TROP I

In mild cases, at admission, elevated TROP I was noted in 39% of cases, and at follow-up, elevated TROP I was noted in 12% of cases. In moderate cases, at admission, elevated TROP I was noted in only 17% of cases, and at follow-up, elevated TROP I was noted in 8% of cases. In severe cases, at admission, elevated TROP I was noted in only 21% of cases, and at follow-up, elevated TROP I was noted in 13% of cases.

Comparison of PR interval, QRS prolongation, QTc prolongation, ST-segment depression, ST-segment elevation, T wave changes at admission (baseline), and follow-up

A total of 35 cases had prolonged PR interval at baseline, whereas only seven cases were at follow-up. At baseline, 27 cases showed QRS prolongation, whereas only six cases showed QRS prolongation at follow-up. A total of 28 cases had QTc prolongation at baseline, whereas only 11 cases at follow-up. At baseline, 28 cases had ST-segment depression, whereas only 10 cases had ST-segment depression at follow-up. Altogether, 28 cases showed QTc prolongation at baseline, whereas only 11 cases showed QTc prolongation at follow-up. A total of 44 cases had T wave changes at baseline, whereas only seven cases were at follow-up. On performing the McNemar test, the difference between admission and follow-up among all the above parameters was found to be statistically significant ($P < 0.05$).

Comparison of head injury cases with RWMA, DD, valvular dysfunction at admission (baseline), and follow-up

A total of 23 cases had RWMA at baseline, whereas only 14 cases at follow-up. At baseline, 41 cases had DD, whereas only 25 cases were at follow-up. At baseline, 27 cases had CPK >6.3, whereas 15 cases at follow-up. Thirty-nine cases had valvular dysfunction at baseline, whereas 25 cases at follow-up. On performing the McNemar test, the difference between admission and follow-up among all the above parameters was found to be statistically significant ($P < 0.05$).

Comparison of CPK-MB (>6.3), TROP T, TROP I (>40) at admission (baseline), and follow-up

At baseline, 27 cases had CPK >6.3, whereas only 15 cases had CPK >6.3 at follow-up. A total of nine cases had positive TROP T at baseline, whereas only four cases had at follow-up.

Seventy-four cases had TROP I >40 at baseline, whereas only 31 cases had at follow-up. On performing the McNemar test, the difference between admission and follow-up among all the above parameters was found to be statistically significant ($P < 0.05$). None of the cardiac changes in the present study (logistic regression) was statistically significant in predicting mortality [Table 1].

Table 1: Logistic regression for risk factors and outcome (mortality).

Variable	OR	CI
RWMA	-	-
Diastolic dysfunction	0.25	0.029–2.299
Valvular disease	-	-
CPK >6.3	0.49	0.05–4.44
TROP T	-	-
TROP I >40	1.35	0.14–12.23
EF	1.04	0.90–1.19
Arrhythmias	1.14	0.12–10.53
PR Interval	1.68	0.32–8.81
QRS Prolongation	2.48	0.47–13.07
QTc Prolongation	2.35	0.44–12.39
ST-segment depression	1.17	0.20–6.80
ST-segment elevation	1.63	0.28–9.54
T wave changes	0.22	0.02–2.02

RWMA: Regional wall motion abnormality, PR: Pulse rate, CI: Confidence interval, OR: Odds ratio, CPK: Creatine phosphokinase, TROP T: Troponin T, TROP I: Troponin I, EF: Ejection fraction, QRS: Q wave, R wave, S wave; QTC: Q wave, T wave Corrected; ST: S wave, T wave

DISCUSSION

Cross-talks between the brain and the heart have been extensively explored in acute neurological insults such as SAH and TBI.[23] However, less attention has been paid to these interactions during TBI and other aspects of perioperative treatment, including the impact of surgical management, experience, and evidence gathered from SAH suggest that these changes are transient and revert to normalcy after successful management of aneurysm.[24] In this study, we observed ECG changes in nearly half of the patients with TBI, while 13% had associated echocardiographic changes. This has been mentioned in numerous case descriptions before.[11,15,30] The actual mechanism that causes these heart functions to improve after surgery is unknown. Reduced ICP following surgical decompression with or without clot removal may increase intracranial compliance, resulting in the reversal of catecholamine surge and its possible cause cardiovascular implications. Furthermore, postoperative intensive care reduces elevated ICP secondary to cerebral resuscitative measures (head-up, sedation, osmotic agents, etc.), which contributes to improved intracranial compliance and leads to a decrease in postoperative cardiovascular changes.

The awareness of cardiac injury possibilities as well as its impact on patients' outcomes might raise the importance of cardiac protection in high-risk cases. Unfortunately, there is no proper consensus on the effectiveness of any type of cardiac protection in neurosurgical patients at this time.[33] Neil-Dwyer *et al*.[19] stated that propranolol and phentolamine may have a role in cardiac protection, citing the absence of myocardial necrosis in postmortem specimens for patients with SAH who were treated with these drugs.[18] In acute central nervous system conditions, various theories have been proposed to describe stress-induced cardiac injury; the most commonly accepted one is the catecholamine-mediated direct cardiac injury – catecholamine hypothesis as a result of autonomic stimulation caused by direct brain injury. Various authors highlighted catecholamine elevated levels in the serum of patients with SAH[3,16] in an experimental model of SAH in dogs[29] as well as in cases of myocardial stunning after sudden emotional stress.[8]

The literature studies on age distribution have been compared with the present study [Table 2]. The mean age in the previous studies on TBI and cardiac changes ranged from 30.8 to 63 years. The mean age in our study was 43.88 ± 15.96, which is comparable to the previous studies.[7,12,21,22,25,32]

Table 2: Comparative studies related to age distribution.

Comparative studies	Mean±SD (years)
Samudrala *et al*., 2018	34.39±15.4
Prathep *et al*., 2014	58±20
Venkata *et al*., 2018	44.7±20.7
Hasanin *et al*., 2016	30.8±12
Praveen *et al*., 2021	39±13.43
Krishnamoorthy *et al*., 2014	63±2
Present study	43.88±15.96

SD: Standard deviation

In our study, predominantly males were presented with TBI (86%) when compared to females (14%) with a male: female ratio of 6.1:1. Krishnamoorthy *et al*.,[12] reported that 61% were male and 39% were females, and in the study conducted by Venkata *et al*.,[31] 36 (78%) were men and 10 (12%) were women. Praveen *et al*.,[22] reported 48 (80%) males and 12 (20%) females, and Samudrala *et al*.,[25] reported that 97 (88.9%) were male and 12 (11.9%) were female in their study.

The distribution of cases according to the severity in the present study was mild (55%), moderate (21%), and severe (24%). The literature on similar studies varies. Prathep *et al*., reported that the majority of the patients had severe TBI (56.1%) and mild TBI (36.7%).[21] In another study, Praveen *et al*., reported mild/moderate/severe TBI 12/44/4 (20/73.33/6.66%).[22] Similarly, Samudrala *et al*., reported as the most common head injury was mild (65.1%), followed by severe (18.3%) and moderate (15.6%).[25] This could be a part of referral bias.

SDH was the most common injury observed, which constituted 45% (45/100), followed by Contusion in 20% (20/100) cases, and other EDH etiology DAI and was noted in 27% of cases (27/100). The literature review of studies on assessing cardiac changes had similar heterogenecity with regard to the pathology of head trauma [Table 3].

In the present study, the mean heart rate was 89.55 ± 12.7; repolarization abnormalities most commonly detected were prolongation of QTc interval, inversion of T-wave, and changes in ST-segment. Prolonged QTc was noted in 17% of severe cases, 3%, and 11% in mild and moderate cases. ST-segment elevation was noted in 14% of severe cases, 7%, and 3% in moderate and mild cases. ST-segment depression was noted in 16% of severe cases, 9%, and 5% in moderate and mild cases. Q wave distribution was noted in 8% of severe cases, 5%, and 7% in moderate and mild cases. T wave inversion was noted in 12% of severe cases, 22% of mild cases, and 11% of moderate cases, respectively. In a study conducted by Krishnamoorthy *et al*., the median heart rate was 79 ± 3 bpm, with almost 80% being in a sinus rhythm.[12] Prolonged PR interval was found in 6.8% of patients, whereas QTc prolongation was identified in 42.4% of patients. Ischemic-like ECG variations (ST depression, ST elevation, and Q wave) developed in a small number of patients (3.4%, 3.4%, and 6.8%, respectively). Inverted T waves were found in 11.9% of patients, and morphological end repolarization abnormalities (MERA) were observed in 10.2% of patients. Thirteen (22%) patients had tachycardia, 25 (42.4%) patients had a prolonged QTc, and 6 (10.2%) patients had MERA, with each having a univariate association with cardiac dysfunction 3: 4.17 ([1.0217.05]; $P = 0.04$), 9.0 ([1.7446.65]; $P = 0.003$), and 5.63 ([1.9632.94]; $P = 0.03$), respectively. Hasanin *et al*., reported that the cardiac assessment revealed that among 50 patients, ECG abnormalities were observed in 31 (62%) patients, (abnormal QT intervals were found in 6 [12%] patients, tachyarrhythmia was in 29 [58%] patients and sinus bradycardia was developed in 5 [10%] patients).[7] Pathological Q-wave or U-wave was present rarely in 1% of patients. Prolongation of QTc (36%) was the most common conduction disorder, which was significantly identified more in men group ($P = 0.023$) (40 vs. 22%). QRS interval abnormalities were found in 11% of patients, and PR-interval abnormalities in 5% of patients. Cardiac arrhythmia was predominantly of supraventricular origin (95%) with 66% sinoatrial node (sinus tachycardia [35%] and sinus bradycardia [31%]) and 29% atrial or AV junctional dysfunction. Ventricular arrhythmias were present in 5% of patients.[14] In the study conducted by Venkata *et al*., the majority of these findings were nonspecific ST-T wave changes (8 patients).[31] These include T-wave flattening, ST repolarization changes, and minimal ST-segment depressions. Other abnormal ECG findings included right bundle branch block (three patients), LVH (one patient), and prolonged QT interval (one patient).[32]

Praveen *et al*., observed various abnormalities from sinus bradycardia/tachycardia to ventricular tachycardia.[22] Repolarization abnormalities were more common, such as prolongation of QTc interval, inversion in T-wave, and elevation/depression in the ST-segment. Post-surgery, statistically significant reduction was noted in repolarization abnormalities such as prolongation of QTc (58.62% vs. 13.79%, $P = 0.001$), variations in ST-segment (41.37% vs. 13.79%, $P = 0.008$), and variations in T wave (24.13% vs. 3.44%, $P = 0.031$), respectively. All patients with a baseline rate/rhythm disturbance displayed sinus rate/rhythm in the postoperative period (24.13% vs. 3.44%, $P = 0.002$).[22] Samudrala *et al*, reported various abnormal electrocardiographic findings following this intense neuroinflammatory response in patients with acute brain injury (including TBI).[25] Although the true incidence of the abnormal ECG findings is largely unknown, the main reported abnormalities were atrial fibrillation, premature atrial and ventricular contractions, sinus tachycardia, and AV dissociation. Other ECG abnormalities may include QT interval prolongation, abnormalities in the ST segment, flat or inverted T waves, U waves, peaked T waves, Q waves, and widened QRS complex.[25] Fan *et al*. described that ST-T changes (41.5%) were the most common ECG abnormality, which was noticed, followed by sinus tachycardia (23.6%) after an acute brain injury.[4] In the majority of the cases, once the management of TBI is initiated, the patient showed signs of recovery and even brain injury-related cardiac dysfunction also showed a spontaneous resolution.

Table 3: Comparative studies related to the type of injury distribution.

Type of injury distribution	Krishnamoorthy *et al*. (%)	Venkata *et al*. (%)	Hasanin *et al*. (%)	Prathep *et al*. (%)	Praveen *et al*. (%)	Present study
SDH	35 (59.3)	11 (24)	10 (20)	87 (62.6)	13 (21.6)	45 (45)
SAH	8 (13.6)	11 (24)	5 (10)	23 (16.5),	22 (36.6)	10 (10)
IPH	7 (11.9)	4 (8.6)	4 (8)	6 (4.4),	-	3 (3)
CONTUSION	6 (10.1)	8 (17.4)	-	12 (8.6)	21 (41.6)	20 (20)
EDH-DAI (others)	3 (5.1)	12 (26)	5 (10)	11 (7.9)	-	27 (27)

SDH: subdural hematoma, SAH: Subarachnoid hemorrhage, IPH: intraparenchymal hemorrhage, EDH: Extradural hematoma, DAI: Diffuse axonal injury

ECG morphological changes

In the present study, the most common findings were changes in ST segment, flat or inverted T waves, prominent U waves, as well as QTc interval prolongation. These findings can be corroborated and explained by the hypothesis of circulating catecholamine levels and ECG abnormalities and increased secondary to myocardial injury by activating the local sympathetic network. Even though it is widely recognized that neurogenic ECG variations are unrelated to cardiac hypoperfusion, distinguishing them from an acute ischemic coronary event can be challenging. ECG abnormalities develop mostly in the first few days after the injury which are temporary because repolarization normalizes as the neurological insult resolves. However, in certain cases, it might persist for up to 8 weeks. Although most neurogenic ECG variations are asymptomatic, abnormalities such as depression in the ST segment and T wave abnormalities have been linked to the development of delayed ischemic neurological impairment, poor outcome, as well as death after a TBI.

After a brain injury, increased QTc interval prolongation can lead to sudden cardiac death.[5] It is of note that after TBI, prolongation of the QTc interval continues to occur in patients with an unfavorable outcome, but it gradually improves in those patients who have a good outcome. The magnitude of ECG abnormalities appears to be linked to the brain injury severity, with greater ST and QT variations being linked to an adverse neurologic outcome. ECG changes seem to correlate with the severity of the brain injury, with greater ST and QT changes associated with a worsened neurologic outcome. Prolongation of QTc occurs in traumatic SAH as well, as it does in non-traumatic SAH, with greater prolongation as the severity exacerbates.[5] There can be varied causes of elevations in the ST segment in the ECG of a head injury patient. ST elevations were noted initially, which normalized subsequently. Elevations in the ST segment have also been seen in patients with neurogenic stunned myocardium (NSM).[5] There were ST-segment elevations in the ECG and hypotension which returned to normal, corresponding to improvement in neurological status. The present study believes that ST-segment elevations in the ECG of our patients following the neurological sequelae as a case of reversible neurogenic myocardial dysfunction.

2D echo findings in head injury cases

In the present study, an ejection fraction of 55–70% was noted in 21% of severe cases, 54% of mild, and 21% of moderate cases, respectively. Right ventricle dilation was noted in only 1% of severe cases and 7% and 2% of mild and moderate cases, respectively. Left ventricle dilation was noted in only 1% of severe, mild, and moderate cases. Aortic valves are involved in 9% of severe cases, 1%, and 4% of mild and moderate cases. The mitral valve was involved in 2% of severe cases, 25% of mild, and 8% of moderate cases. Pulmonary valves were involved in 5% of severe cases 10% of mild, and 3% of moderate cases, respectively. The tricuspid valve was involved in 5% of severe cases, 16% of mild, and 9% of moderate cases, respectively. RWMA was observed in 23% of cases, DD was noted in 45% of cases, and valvular dysfunction was noted in 39% of cases.

Hasanin *et al*. observed that in 14 (28%) patients, an echocardiographic assessment for contractility findings showed abnormalities of which 5 (10%) of them were global hypokinesia. Sixteen (32%) patients showed systemic arterial hypotension.[7] The cardiac injury score was 0 in 16 (32%) patients, the score was 1 in 15 (30%), followed by 2 in 14 (28%) patients, and the score was 3 in 5 (10%) patients. Overall, in Krishnamoorthy *et al*., 15.3% of patients showed a reduced LV ejection fraction, and 13.6% developed a documented RWMA.[12] In the reported literature, very few studies have explained the development of echocardiographic changes in TBI patients. Krishnamoorthy *et al*. described early systolic dysfunction in 22% of moderate-to-severe TBI patients, which improved over 1st week, but none of the mild TBI group showed any variations. In line with this study, we observed DD in 10% of cases of mild TBI, 6% of cases of moderate TBI, and 10% of cases of severe TBI.[12] In a study conducted by Prathep *et al*., overall, 22.3% of TBI patients with echocardiograms showed cardiac dysfunction; 12% of patients showed reduced left ventricular ejection fraction (LVEF 43% ± 8%), and 17.5% of patients were documented with RWMA.[21] In Venkata *et al*., study, 13% of patients with moderate-to-severe TBI developed cardiac dysfunction in the ICU (as the mild-to-moderate reduction in LVEF) and showed ECG abnormalities in a higher proportion.[32] Hasanin *et al*. reported ECG abnormalities in 62% as well as echocardiography abnormalities in 28% of patients with severe TBIs.[7] The majority of patients having abnormal ECG findings did not show equivalent echocardiographic abnormalities. The ECG is a poor cardiac risk marker in the perioperative period, according to the existing studies and outside of causal hypotheses relating neural injury to cardiac abnormalities. In contrast, as shown in our study, the majority of individuals with abnormal echocardiograms also had comparable repolarization abnormalities on ECG. Therefore, to make a rationale for routine echocardiographic screening before anesthesia in patients with TBI, larger studies are needed.[25]

Venkata *et al*., investigated 46 moderate-to-severe TBI patients, of which 6 (13%) of them noted cardiac dysfunction, mildly reduced LVEF in four patients, and moderate reduction in LVEF in two patients. TBI severity was evaluated using a GCS score that was linked to the onset of cardiac dysfunction. There was no association between the occurrence of heart

dysfunction and adverse clinical outcomes.[32] Venkata *et al*. did not notice any correlation between TBI severity as well as the occurrence of cardiac dysfunction. The mean GCS scores of patients who had cardiac dysfunction as well as those who did not (5.5 vs. 5.6, $P = 0.95$) were not statistically significant.[32] The present data on cardiac dysfunction after a TBI are restricted to a very limited number of studies, with conflicting findings on the incidence as well as its clinical outcomes. In a retrospective analysis of 139 cases with isolated TBIs who received echocardiography within 2 weeks of their hospital stay, around 31 cases (22%) demonstrated the development of cardiac dysfunction, as defined by reduced LV systolic function or the presence of regional wall motional abnormalities. It was also noted that abnormalities in echocardiogram were associated independently with increased in-hospital mortality.[32] A small prospective study on 49 individuals with moderate-to-severe TBI found that none of them showed depressed LVEF, and 4 (8%) patients developed regional wall motional abnormalities with preserved LVEF.[17]

In the present study, ventricular dysfunction (right and left ventricle dilatation in 10% and 3% cases), hypokinesia, impaired LV contractility, and low ejection fractions are allied with the NSM syndrome.[17] This reflects the sympathetic nerve distribution rather than specific vascular territories in line with the known etiology of NSM. After a TBI, left ventricular dysfunction happens in around 3% of the patients, usually within 3 days of the ictus, but the dysfunction degree is usually mild. Even though usually left ventricular dysfunction is transient, it is linked to an increased risk for mortality after TBIs.[17] In this study, DD was found much more common after TBI, was noted in around 71% of the patients, and was linked with pulmonary edema at a higher incidence rate.

Role of surgery in relieving cardiac dysfunction in TBI

More than half of the cohorts with acute TBI underwent surgery, and (due to brain death, 6 patients have died before 28 days of treatment) cardiac abnormalities were recorded intraoperatively, which included ST-segment depression wave inversion improved QTc. Surgical decompression was the most important factor in improving the patient's cardiac function.[13] This study reported the role of decompressive craniectomy (DC) on variables of electrocardiography. In our study, elevated ICP was linked to a prolonged QTc interval, and emergency DC reduced the QTc in 13 patients. This effect was more remarkable in the survivors. Increased spatial QRS-T angle and QTc interval have long been recognized as major markers of life-threatening cardiac arrhythmias and sudden cardiac death.[13] In the present study, we reported a considerable positive association between ICP as well as QTc interval. DC generally performed for ICP reduction and shortening QTc interval, However, after DC, a significantly longer QTc was observed in six patients who did not survive. Hence, considering these findings, ICH is likely to be an additional risk factor for prolonged QTc.

Cardiac enzymes in head injury patients

In the present study, 46 (46%) had elevated CPK MB, TROP I elevated in 53 (53%), and TROP T elevated in 16 (16%). There was no association between the severity of TBI at admission and follow-up, but TROP T findings at follow-up were statistically significant with a *P*-value (<0.003). CPK MB and TROP I findings at admission and follow-up were statistically significant with *P*-values (<0.01 and <0.001). Cardiac enzymes were analyzed in a few studies like in Hasanin *et al*. study, TROP I enzyme levels were found to be increased in 27 (54%) of patients on day 1.[7] Among 24 (22.4%) of 107 patients tested for TROP, I showed increased levels, but there was no association between TBI severity (admission GCS or head AIS scores) and elevated cardiac enzymes, according to Prathep *et al*.[21]

Biomarkers of cardiac injury

In the present study, after TBI, cardiac TROP I (cTnI) levels have been found to rise in 20–68% of patients (mean incidence 36%) and usually spikes within 24–36 h. For detecting LV dysfunction, cardiac TROP, I is more sensitive than creatine phosphokinase-myocardial fraction (CK-MB). Cardiac TROP I has a sensitivity of 100% and specificity of 86% compared with phosphokinase-myocardial fraction, which is 29% sensitive and 100% specific, respectively. Although the peak concentration is typically below the diagnostic threshold for myocardial infarction, it has been linked to mild, transient ventricular dysfunction in 50% of the patients. Initial brain injury severity influences the degree of rise in cTnI levels. A highly positive response is an independent predictor of acute RWMAs and is associated with an increased risk of death and poor functional outcomes in survivors.

CONCLUSION

TBI incites electrocardiographic and echocardiographic abnormalities irrespective of the severity of the head injury, and without pre-existing cardiac elements necessitates adequate focus and attention as these the reversible following the resolution of inciting cerebral events. Improvement in QTc interval favors a good prognosis, while persistent, prolonged QTc is a harbinger of ominous outcomes.

Ethical approval

The research/study approved by the Institutional Review Board at Nizam's Institute of Medical Sciences, number No.EC/NIMS/2637/2020, dated August 28, 2021.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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