



Original Article

Perioperative cardiovascular changes in patients with traumatic brain injury: A prospective observational study

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ABSTRACT

Background: Traumatic brain injury (TBI) is an acutely stressful condition. Stress and conglomeration of various factors predispose to the involvement of other organ systems. The stress response from TBI has been associated with cardiovascular complications reflecting as repolarization abnormalities on electrocardiogram (ECG) to systolic dysfunction on echocardiography. However, the perioperative cardiac functions in patients with TBI have not been evaluated.

Methods: We conducted a prospective observational study 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 h.

Results: 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.

Conclusion: Cardiac dysfunction occurs frequently following TBI. Even patients with mild TBI had preoperative systolic dysfunction on echocardiography. Surgical intervention in the form of hematoma evacuation and decompression was associated with significant regression of both ECG and echocardiographic changes.

Keywords: Electrocardiography, Transthoracic echocardiography, Traumatic brain injury

INTRODUCTION

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. The most common non-neurologic site involved was the respiratory system (23%) followed by the cardiovascular system (18%).^[11,24] The cardiovascular complications varied from extreme swings in blood pressure, repolarization abnormalities such as corrected QT (QTc) prolongation,

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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.^[3,7] This is often associated with increased morbidity and mortality.^[19,24] The cause could be attributed to a surge in circulating catecholamines and a global inflammatory state.^[16,18]

The overwhelming focus has been on the clinical model of aSAH, electrocardiographic changes, and echocardiographic abnormalities in the literature.^[4,15,23] 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. Only recently the presence of such cardiac dysfunction in TBI has been described in a few reports and studies.^[2,6,7,19,20] Singla *et al.* found that ECG changes were present in almost all TBI patients (99%).^[20] Fan *et al.* 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.^[7] Others have shown that ECG changes do occur in children with a head injury.^[6] In a retrospective review from a Level 1 regional trauma center about 22% of patients have echocardiographic abnormalities.^[19]

Head injury-related ECG abnormalities commonly include ST-segment changes, flat/inverted T waves, prominent U waves, and prolonged QTc interval.^[7,20] Repolarization abnormalities and QT prolongation were found to be independent prognostic factors for negative outcomes. 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.^[19]

Few case reports have described the effect of surgical intervention on perioperative cardiovascular changes due to TBI.^[9,12,17] Intraoperative transthoracic echocardiographic done before decompressive surgery showed moderate basal hypokinesia with EF-35% unresponsive to vasopressors. Transthoracic echocardiography 5 min post decompression revealed resolution of basal hypokinesia with improvement in EF.^[12] In another case, multiple episodes of ventricular tachycardia were seen preoperatively which never reappeared following surgical evacuation of extradural hematoma.^[17] Jain *et al.* observed that the ECG changes reverted to normal after 48 h following hematoma evacuation in the severe head injury.^[9] However, the status of ECG and/or echocardiography abnormalities preoperatively and following surgical intervention has not been studied prospectively in patients with TBI. Hence, this study was designed to study the perioperative cardiovascular manifestations using the ECG as the basis for a definitive prospective registry

type study. We report both data of local and transthoracic echocardiography.

MATERIALS AND METHODS

This prospective, observational study was conducted on 60 consecutive isolated head injury patients of either sex between the age group of 10 and 70 years who underwent decompressive craniectomy over a period of 1½ year. All those patients who had valvular/coronary artery disease, or previous cardiac/lung surgery, and baseline electrolyte abnormality were excluded from the study. Before commencement of study, institute Ethics Committee approval (NK/1661/DM/10986) was sought and informed consent was obtained from either patients or next of the kin.

Cardiac evaluation

To carry out cardiac evaluation-ECG and transthoracic echocardiography were performed at two different points. Baseline ECG was obtained within the first 6 h after hospital admission, and then within 24–48 h after surgical intervention. Heart rate, rhythm, the QTc interval, and presence of repolarization abnormalities were computed and compared at two different time points.^[13] Abnormal ECG was considered if there were flattening or inversion of T-wave, ST-segment elevation, or depression (ST-segment changes > 2 mm), prolonged QTc interval >430 and >440 ms in male and female patients, respectively, pathologic Q-waves and U-waves (>1 mm), conduction defects, and rhythm disturbances (Bradycardias and tachycardias with HR < 60 beats/min and >100 beats/min, respectively).

Transthoracic echocardiography was performed by cardiac anesthetist using P21x, 5-1MHz, 21 mm broadband, phased array probe of a Sonosite; Fujifilm Sonosite Inc. echocardiographic machine at baseline and then postoperatively within 24–48 h. Chamber quantification was performed as per the guidelines of the American Society of Echocardiography (ASE).^[15] RWMA were marked per the 16-segment model where each segment was graded as normal (score = 1), hypokinetic (score = 2), akinetic (score = 3), or dyskinetic (score = 4) and was calculated by averaging the score for each of the 16 segments. RWMA >1 was considered abnormal for analysis purpose. Global EF was determined using Simpson's method of discs. For study purposes, a left ventricular ejection fraction (LVEF) <50% was considered abnormal. Diastolic function was graded based on the transmitral Doppler, pulmonary venous Doppler, and principally the E/E' obtained at the mitral annulus.^[14] Right ventricular (RV) function was graded based on the Tricuspid Annulus Plane Systolic Excursion (TAPSE), the lateral tricuspid annular S' velocity on Tissue Harmonic Imaging and the fractional area change were additionally obtained on

a focused RV view as recommended by the ASE if the TAPSE was abnormal (<20 mm).

Perioperative management

Patients underwent a brief detailed preanesthetic check in the preoperative holding area. All patients received general anesthesia as per the discretion of the attending neuroanesthesiologist. Standard American Society of Anesthesiologist intraoperative monitors were applied. Following surgical intervention, patients were shifted to the intensive care unit.

Statistical analysis

Normality of quantitative data such as age, admission Glasgow coma score (GCS), ECHO parameters (Left ventricular end diastolic diameter/Left ventricular systolic end diameter/EF/Fractional shortening [FS]) was checked by measures of Shapiro–Wilk test of normality. For normally distributed data, mean \pm standard deviation (SD) whereas for skewed data, median, and interquartile range (IQR) has been reported. Paired *t*-test was applied to compare preoperative and postoperative normally distributed quantitative data and presented as mean \pm SD. Categorical variables such as sex (M/F), GCS (Mild/moderate/severe TBI), TBI Morphology (acute SDH/EDH/SDH with contusion), and ECG/ECHO changes presented as number and percentages. Chi-square test (or Fisher Exact test, whichever applicable) was applied to find out significant association between preoperative ECG/Echocardiography changes and grades of TBI. McNemar test was applied to find out significant changes in categorical variables from preoperative to postoperative. All calculations were two sided and were performed using Statistical Package for the Social Sciences (SPSS Inc. 2013, version 22.0 for Windows, Armonk, NY, USA). $P < 0.05$ considered statistically significant.

RESULTS

Sixty consecutive subjects with isolated TBI requiring neurosurgical intervention, who satisfied the inclusion criteria were studied. Baseline demographic and clinical-radiological parameters are shown in [Table 1]. The median GCS in our study was 11 and the majority of cases had moderate degrees of TBI (44/60, 73%). Preoperative occurrence of ECG/echocardiographic changes in various grades of TBI (classified on the basis of GCS) was not statistically significant ($P = 0.32$ and 0.51 , respectively) as described in [Table 2].

[Table 3] portrays various cardiovascular manifestations as measured by ECG. Approximately 48.33% of patients (29/60) had changes in baseline ECG which were reduced

Table 1: Baseline demographic and clinico-radiological parameter.

Baseline parameter	Values
Age in years	39 \pm 13.43
Sex (M/F)	48/12
Admission Glasgow coma score	11 (10–12)
Mild/Moderate/Severe TBI (n/%)	12/44/4 (20/73.33/6.66%)
TBI morphology (acute SDH/EDH/SDH with contusion)	13/22/25 (21.66/36.66/41.66%)

Values are mean \pm standard deviation, Median (Interquartile range), number (percentage). TBI: Traumatic brain injury

Table 2: Preoperative ECG/Echocardiography changes and grades of TBI.

	Mild/Moderate/Severe TBI (%)	P-value
ECG changes	4/22/3 (33.33/50/75)	0.32 ^{NS}
Echocardiography changes	2/5/1 (16.66/11.36/25)	0.51 ^{NS}

Values are described as numbers (percentages). $P < 0.05$ as statistically significant, NS: P value not significant. ECG: Electrocardiogram, TBI: Traumatic brain injury

Table 3: Comparison of preoperative and postoperative ECG changes.

ECG changes	Preoperative n (%)	Postoperative n (%)	P-value (McNemar test)
Overall changes	29	9	0.001*
Corrected QT prolongation	17	4	0.001*
ST segment changes	12	4	0.008*
T wave changes	7	1	0.03*
Arrhythmias (Brady/Tachycardias/Atrial or Ventricular premature complex)	16	0	0.002*

Values are number (percentages). * $P < 0.05$ as statistically significant, NS: P value not significant. ECG: Electrocardiogram

to 13.33% (8/60) postoperatively. These changes varied from benign sinus bradycardia/tachycardia to malignant ventricular tachycardia. Repolarization abnormalities were more common such as QTc interval prolongation, T-wave inversion, and ST-segment elevation/depression. Following surgery there was statistically significant reduction from baseline parameters in repolarization abnormalities such as QTc prolongation (58.62% vs. 13.79%, $P = 0.001$), ST-segment changes (41.37% vs. 13.79%, $P = 0.008$), and T wave changes (24.13% vs. 3.44%, $P = 0.031$), respectively. Furthermore,

all patients who had a baseline rate/rhythm disturbances displayed sinus rate/rhythm in the postoperative period (24.13% vs. 3.44%, $P = 0.002$).

[Tables 4 and 5] describe echocardiographic changes in TBI patients. In this study, echocardiographic abnormality primarily included left ventricular systolic dysfunction quantified by the decrease in EF (<50%), FS (<25%), or global hypokinesia as seen in eight patients (13.33%). Among these eight patients, mild systolic dysfunction was seen in five patients while three patients had moderate dysfunction. None of these patients had severe systolic dysfunction. EF and FS significantly improved within 48 h of surgery from baseline in all these eight patients following surgery. Diastolic dysfunction was determined by the altered E/A ratio and four patients showed diastolic dysfunction. Right heart function was normal in all the patients as assessed by the TAPSE. None of the patients showed valvular abnormalities. Global systolic dysfunction was seen in three patients; however, no distinct segmental wall motion changes were observed in any case.

A majority of patients with an ECG abnormality did not have a corresponding echocardiographic disturbance. Seven of the eight patients who had echocardiographic changes showed ECG changes. None of our patients received vasopressors/inotropes postoperatively. Intraoperative hypotension was treated by phenylephrine boluses which occurred in few cases and were not statistically significant.

Table 4: Comparison of preoperative and postoperative systolic function in echocardiography in all patients ($n=60$).

Echocardiographic parameters	Preoperative function (Mean±SD)	Postoperative function (Mean±SD)	P-value (Paired t-test)
LVEDD (mm)	4.14±25	4.16±0.24	0.06 ^{NS}
LVSED (mm)	3.01±0.22	2.98±0.19	0.10 ^{NS}
EF (%)	52.67±5.41	55.20±2.50	0.001*
FS (mm)	27.22±2.76	28.32±1.92	0.001*

* $P < 0.05$ as statistically significant, NS: P -value not significant.
LVEDD: Left ventricular end diastolic diameter, LVSED: Left ventricular systolic end diameter, EF: Ejection fraction, FS: Fractional shortening

Table 5: Comparison of preoperative and postoperative systolic function in echocardiography in patients with preoperative systolic dysfunction ($n=8$).

Echocardiographic parameters	Preoperative function (Mean±SD)	Postoperative function (Mean±SD)	P-value (Paired t-test)
EF (%)	41.77±7.19	51.99±1.20	0.01*
FS (mm)	21.93±2.38	26.21±0.86	0.008*

* $P < 0.05$ as statistically significant, NS: P -value not significant.
EF: Ejection fraction, FS: Fractional shortening

DISCUSSION

The brain-heart cross-talks have widely been studied in acute neurological insults such as aSAH and stroke.^[1,5] The mechanisms suggested have ranged from excessive circulating catecholamines to widespread neuroinflammation, both triggered by the original insult to the central nervous system. Numerous case reports and few studies have suggested an association of similar cross-talk in TBI patients.^[2,3,6,14,19,22] However, there has been a relatively scarce focus on these interactions during TBI and the allied aspect of perioperative care including the effect of the surgical management, although studies are emerging on aSAH showed improvement in cardiac functions following surgical clipping of aneurysm.^[10]

In this prospective study, it was found that 48.33% of patients with TBI have cardiovascular changes in a preoperative period as seen in ECG and 13% of patients have echocardiographic disturbances. The probable mechanism could be excessive circulating catecholamines to widespread inflammation, triggered by the neurological insult as suggested by the literature. Later, there was a gross decline in the cardiovascular changes in the postoperative period. Only 13.3% of patients showed ECG changes while none have echocardiography changes, respectively. This has been previously reported in various case-descriptions.^[9,12,17] The exact mechanism responsible for the improvement of these cardiac function following surgery is unknown. Intracranial pressure (ICP) reduction following surgical decompression with or without clot evacuation may improve intracranial compliance contributing to the reversal of catecholamine surge and thereby its possible adverse effects on the cardiovascular system. Furthermore, intensive care in the postoperative period further decreases raised ICP secondary to cerebral resuscitative measures (head-up, sedation, osmotic agents, etc.) which adds to the improvement of intracranial compliance and leads to a decline in cardiovascular changes in the postoperative period.

In our study, repolarization abnormalities most commonly detected were QTc interval prolongation, T-wave inversion, and ST-segment changes. Krishnamoorthy *et al.* reported prolonged QTc, morphologic end repolarization abnormalities in 42.4% and 10.2% isolated TBI while ST changes, inverted T waves in 6.8% and 11.9% patients, respectively.^[12] The overall incidence of abnormalities detected by our study was lower than previously cited; this could be attributed to our methodology of sub-stratifying ECG changes such as those in the heart rate alone from repolarization abnormalities. As such, changes in the heart rate alone could have other causes such as a different level of sedation, pain from other injuries, and so on. Further, most of these changes regressed and a majority of the patients with ECG change did not have a corresponding echocardiographic

correlate, testifying to our choosing to differentiate between sheer changes in rhythm and repolarization abnormalities.

In the reported literature, only a couple of studies have described the occurrence of echocardiographic changes in TBI patients. A study conducted by Krishnamoorthy *et al.* showed early systolic dysfunction in 22% moderate-severe TBI patients which improved over their 1st week while none in the mild TBI group demonstrated any changes.^[12] In line with this study, a similar percentage of patients showed echocardiographic changes in moderate-severe TBI in our study.

Prathep *et al.* in a retrospective analysis reported cardiac dysfunction in 22.3% of patients with TBI using echocardiography during the first 2 weeks. Reduced LVEF and RWMA were documented in 12% and 17.5% of patients, respectively.^[19] Their overall median time to echocardiography was 3 days after TBI, somewhat similar to our timings of postoperative echocardiography. However, in our study, abnormal findings were seen in a smaller proportion of patients in the preoperative period (13.33%) and normal echocardiography in this subset after surgical intervention. This could probably due to the relatively younger study population with a mean age of 39 + 13.43 years in our study. Furthermore, we did not specifically model our findings to the nature and severity of TBI, since ours is perhaps the first description of a study proximate to hospital admission it may more reliably separate the effects of TBI alone from other causes such as sepsis, fluid management, and quasi-phenomena native to intensive care.

Venkata *et al.* found cardiac dysfunction in 13% patients (as mild-to-moderate reduction in LVEF) in moderate to severe TBI patients in the intensive care unit and patients who developed cardiac dysfunction had a higher proportion of ECG abnormalities.^[21]

Hasanin *et al.* showed abnormal ECG changes in 62% and abnormal echocardiography findings in 28% patients in severe TBI.^[8] A majority of patients with an ECG abnormality did not have a corresponding echocardiographic disturbance. In line with the other perioperative literature on the subject and outside of causal hypotheses relating neural injury to cardiac disturbance, the ECG is a poor marker of cardiac risk in the perioperative period. Conversely, a majority of patients with abnormal echocardiography did have corresponding repolarization abnormalities on ECG (7/8, 87.5%) as displayed in our study. Larger studies are, therefore, warranted to build a case for routine echocardiographic screening before anesthesia in patients with TBI.

Limitations

Given the small number of patients in our study, a definitive conclusion will likely need replication in a larger subset of

patients. Our study was not designed to build a regression model of the effect of surgery on non-neurological organ (systemic) effects of TBI; we can only suggest that this interesting phenomenon needs more detailed study. It further raises the interesting hypothesis that besides offering a therapeutic tool to address the primary insult, active surgical intervention may limit or treat secondary organ effects of neural injury. Nonmeasurement of cardiac enzymes was one of the few limitations of our study.

CONCLUSION

Cardiovascular abnormalities on ECG occurred in 48.33% and transthoracic echocardiographic changes in 13.33% of patients with TBI. Even patients with mild head injury develop echocardiographic changes indicating possible sudden ICP surge as the cause. Surgical decompression significantly improves cardiac functions. Considering that no in-hospital mortality occurred among any of our patient's timed surgical intervention is a must. However, further study with a larger sample size is required to substantiate the same.

Ethics approval and consent to participate

Clearance to conduct the study was obtained from the Institute Ethics Committee in accordance with the Helsinki Declaration of 1975, as revised in 2000. Written informed consent was obtained from either patients or next of the kin all patients who participated in the study.

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Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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