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Review Article

Evaluating the electrocardiographic abnormalities in traumatic brain injury: prevalence, severity correlation, and outcome prediction

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Abstract: Background: Traumatic brain injury (TBI) induces systemic responses, including neurogenic cardiac injury via the brain-heart axis, manifesting as electrocardiographic (ECG) abnormalities that may predict outcomes. Objective: This systematic review aimed to evaluate the diagnostic and prognostic utility of ECG monitoring in patients with TBI, identify knowledge gaps, and guide future research. Methods: Following PRISMA guidelines, we searched PubMed and Google Scholar (January 2020 - March 2025) for studies on adult patients with TBI (≥ 16 years) who underwent acute ECG assessment (≤ 72 hours post-injury). The inclusion criteria focused on observational/cohort studies that reported ECG changes, severity correlations, and outcomes. The exclusion criteria were pediatric cases, pre-existing cardiac conditions, and non-English articles. Data were extracted from the eligible studies. Results: Six studies (1,642 patients) revealed ECG abnormalities in 10-88% of cases, increasing with TBI severity (e.g., prolonged QTc in 3% of mild cases vs. 15% of severe cases). Common changes included repolarization issues (QTc prolongation and ST-segment/T-wave alterations), arrhythmias, and conduction disturbances. Abnormalities often resolved within days, improved post-neurosurgery (e.g., reduced QTc), and predicted mortality (e.g., QTc prolongation/ST depression as independent factors) and cardiac dysfunction. Conclusion: ECG changes are prevalent in TBI, correlate with severity, and have prognostic value for risk stratification. Routine monitoring is recommended, and larger, standardized studies are needed to optimize management.

Keywords: Traumatic brain injury, electrocardiogram, cardiac function, cardiac dysfunction

Introduction

Traumatic brain injury (TBI) is a major cause of mortality and disability worldwide. The estimated worldwide incidence of TBI is 69 million; however, this figure likely underrepresents the actual prevalence due to under-reporting [1, 2]. In addition to primary neurological damage, TBI induces complex systemic responses that significantly affect multiple organ systems, particularly the cardiovascular system [3].

The brain-heart connection, initially described by Bramwell in 1934, encompasses complex neurohormonal and inflammatory cascades that occur following acute brain injury [4]. The bidirectional physiological and pathological relationship between the central nervous system and the cardiovascular system is known as the brain-heart connection, or brain-heart axis. Inflammatory cascades, neurohormonal activation, and autonomic neural pathways mediate this relationship [5]. TBI triggers a robust neuroinflammatory response, resulting in the release of cytokines, adhesion molecules, and multifunctional peptides into systemic circulation. Although this response is initially protective in preserving cerebral perfusion, it can subsequently lead to cardiac neurogenic injury and systemic organ dysfunction [6, 7].

Electrocardiographic (ECG) changes following TBI have emerged as markers of neurogenic cardiac injury and potential predictors of patient outcomes. The reported incidence varies widely across studies, reflecting differences in study populations, injury severity, and diagnostic criteria [8, 9]. These abnormalities include rhythm disturbances (atrial fibrillation, sinus tachycardia), conduction abnormalities (bundle branch

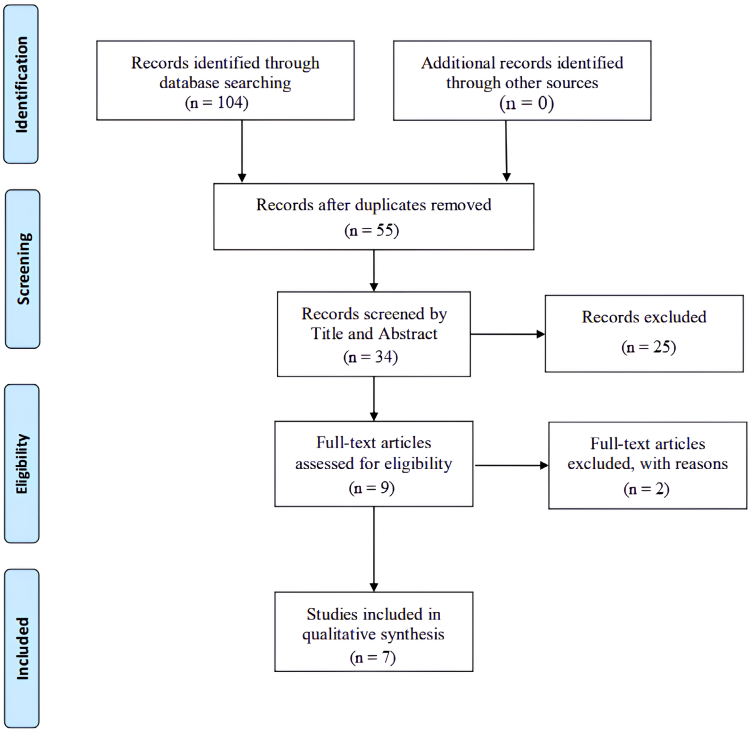

Figure 1. PRISMA flow diagram for enrollment of studies.

blocks, AV blocks), repolarization changes (ST-segment alterations, T-wave abnormalities, QT prolongation), and morphological changes (Q and U waves) [10].

The pathophysiology involves dysregulation of the autonomic nervous system, characterized by excessive sympathetic stimulation that results in a catecholamine surge and neurogenic stunned myocardium. This reversible cardiac injury is evident through abnormal ECG changes, arrhythmias, left ventricular dysfunction, and elevated cardiac biomarkers. The severity of TBI often correlates with the extent of brain damage, with more severe TBI linked to more pronounce cardiac abnormalities [11-13].

Recent studies have highlighted the prognostic significance of specific ECG parameters. Corrected QT interval (QTc) prolongation represents neurogenic cardiovascular dysfunction, whereas ST-segment depression is strongly associated with early mortality [8, 9, 14]. These findings suggest that ECG monitoring may serve as a valuable, noninvasive tool for risk stratification in TBI management.

The perioperative period is a critical window for cardiovascular monitoring in patients with TBI undergoing neurosurgical intervention. Surgical decompression may lead to significant improvements in ECG abnormalities and cardiac function, highlighting the dynamic nature of brain-heart interactions and the potential reversibility of neurogenic cardiac dysfunction [15, 16].

Despite the growing recognition of the clinical importance of ECG changes in TBI, knowledge gaps remain regarding their relationship with long-term outcomes, optimal monitoring protocols, and therapeutic implications. The integration of ECG parameters with existing prognostic tools requires further investigation.

Given the high prevalence of ECG abnormalities in TBI and their potential association with both cardiac and neurological outcomes, a systematic review of recent literature is warranted. Therefore, this systematic review aimed to clarify the diagnostic and prognostic utility of ECG monitoring in this population, identify gaps in knowledge, and inform future research directions to optimize the management and outcomes of patients with TBI.

Material and methods

Search strategy

For this systematic review, we conducted a comprehensive search to identify relevant studies examining the ECG changes in patients with TBI. The research was performed in compliance with the PRISMA criteria, Preferred Reporting Items for Systematic Reviews and Meta-Analyses, and the Flow Diagram is shown in **Figure 1**. The research was conducted in the PubMed and Google Scholar databases from January 2020 to March 2025. It used the Advanced Search Builder, and the keywords were searched in [Title OR Abstract]. We have filtered only research articles published in English language and using the terms '(Brain injuries, Traumatic [Mesh] OR Traumatic brain injury [Text word] OR Traumatic head injury

[Text word] AND (Electrocardiography [Mesh]) OR Cardiography [Text word] OR Electrocardiogram [Text word] OR Electrocardiograph [Text word])'.

Inclusion and exclusion criteria

Studies were eligible for inclusion if they involved adult patients aged ≥ 16 years with traumatic brain injury of any severity who underwent electrocardiographic evaluation within the acute phase of injury, defined as within 72 h post-trauma. Included studies were required to be prospective observational studies, retrospective cohort studies, or case-control studies that provided extractable data on ECG parameters and their association with TBI severity, clinical outcomes, or mortality. Studies comparing ECG findings between different TBI severities, examining pre- and postsurgical intervention changes, or contrasting TBI patients with control groups were included. The primary outcomes of interest included the prevalence of ECG abnormalities and their association with TBI severity and mortality, while the secondary outcomes encompassed specific ECG parameters, such as QTc prolongation, ST-segment changes, cardiac arrhythmias, and repolarization abnormalities.

Studies were excluded if they included patients with pre-existing cardiovascular disease, those taking cardiac medications before injury, or patients with significant chest trauma that could potentially confound cardiac findings. Pediatric studies focusing solely on patients aged < 16 years were excluded, as were case reports, case series, review articles, editorials, and conference abstracts. Studies that did not provide sufficient extractable data on electrocardiographic parameters or failed to meet the temporal criteria for ECG assessment were excluded. Additionally, studies not published in English were excluded to ensure consistent data interpretation and reduce potential translation bias that could affect the quality of the data extraction and analysis.

Data extraction and research indicators

Titles and abstracts were reviewed by A.M. After implementing inclusion and exclusion criteria, data from studies were extracted based on the requirements of the survey.

After scanning the references in previously published review articles, any relevant studies were included. We obtained six eligible published research articles in their final version. For some of them, we chose to include only the main findings that fit the purpose of this review.

Extracted information included general study characteristics such as the first author, year of publication, study design, and total sample size. Patient populations consisted of adult individuals with TBI who underwent ECG assessment within 72 hours of injury, as defined by the study inclusion criteria.

The primary research indicators focused on electrocardiographic abnormalities and their relationship to TBI severity and clinical outcomes. Data were extracted on the presence and prevalence of ECG abnormalities and on the specific types of changes reported in the included studies. These included repolarization abnormalities such as QTc prolongation and ST-segment or T-wave alterations, rhythm disturbances including atrial fibrillation and sinus tachycardia, and conduction disturbances. Where reported, quantitative information such as the proportion of patients with QTc prolongation in different TBI severity categories was also recorded to assess the relationship between ECG abnormalities and injury severity.

In addition, outcome-related indicators were extracted to evaluate the clinical significance of ECG changes in patients with TBI. These included reported associations between ECG abnormalities and mortality, the occurrence of cardiac dysfunction, and the temporal evolution of ECG changes during the acute phase after injury. Information describing the resolution of ECG abnormalities over time or improvement following neurosurgical intervention was also recorded when available. These indicators were subsequently synthesized to evaluate the frequency, patterns, and prognostic relevance of ECG changes in traumatic brain injury.

Results

Search and selection process

A comprehensive literature search conducted until March 2025 identified 104 articles. After duplicates removed and title and abstract

screening, 64 articles were excluded, and 9 underwent full-text review, with 3 further excluded. Ultimately, six studies met the inclusion criteria and were included in the final analysis.

Characteristics of included studies

The five included studies collectively analyzed 1,642 patients with TBI, encompassing a spectrum of severities from mild to severe. The study designs varied, including retrospective analyses, prospective cohort studies, and interventional studies, providing a robust foundation for evaluating ECG changes in this population (**Table 1**).

Prevalence and types of ECG abnormalities

The prevalence of ECG abnormalities in patients with TBI varies widely, reflecting the differences in injury severity and study design. Verma et al. [17] reported abnormalities in only 10% of cases, with 90% of patients maintaining normal electrocardiograms. In contrast, Lenstra et al. [9] observed abnormalities in 88% of patients with severe TBI, including ventricular repolarization disorders (57%, predominantly ST-segment alterations), conduction abnormalities (45%, chiefly QTc prolongation), and arrhythmias (38%, primarily supraventricular).

Ediga et al. [8] conducted a prospective observational study of 100 patients with isolated TBI and found ECG abnormalities distributed across all severity levels. They observed prolonged QTc intervals in 3% of mild, 10% of moderate, and 15% of severe cases. ST-segment elevation was noted in 7% of mild, 3% of moderate, and 14% of severe cases, while ST-segment depression occurred in 5% of mild, 9% of moderate, and 16% of severe cases. T-wave inversions were present in 22% of mild, 11% of moderate, and 12% of severe cases. Prolonged PR intervals were observed in 8% of mild, 11% of moderate, and 16% of severe cases, while prolonged QRS patterns were found in 5% of mild, 7% of moderate, and 15% of severe cases.

Srinivasaiah et al. [18] found ECG abnormalities in a substantial proportion of TBI patients undergoing surgical decompression within 48 hours, with prolonged QTc interval observed in 42% and morphologic end-repolarization abnormalities (MERA) in 47% of patients preoperatively. Lee et al. [14] identified QT prolongation and ST-segment depression as common findings, whereas Hamila et al. [19] noted sinus tachycardia (38% at admission), bradycardia (18%), shortened PR intervals (26%), inverted T waves (16%), and ST elevation (5%). Praveen et al. [20] (n=60, 73% moderate TBI) reported preoperative ECG abnormalities in 48.33% of patients who underwent decompressive craniectomy.

Association with TBI severity

A consistent association was observed between TBI severity and the prevalence of ECG abnormalities. Studies focusing on severe TBI, such as those by Lee et al. [14] and Lenstra et al. [9], reported higher rates (up to 88%), whereas Verma et al. [17] found a lower prevalence (10%) in a cohort with predominantly mild TBI (67% mild, 15% moderate, and 18% severe).

Ediga et al. [8] demonstrated a clear severity-dependent pattern, with repolarization abnormalities being more prevalent in severe cases: prolonged QTc (15% vs. 3% in mild cases), ST-segment elevation (14% vs. 7% in mild cases), and ST-segment depression (16% vs. 5% in mild cases). Their study population consisted of 55% mild, 21% moderate, and 24% severe TBI cases, with a mean heart rate of 89.55 ± 12.7 bpm.

Srinivasaiah et al. [18] demonstrated that a poor GCS motor score was an independent predictor of left ventricular dysfunction (OR 0.406, 95% CI 0.213-0.775), reinforcing the relationship between neurological severity and cardiac abnormalities. Lenstra et al. [9] identified significant associations between arrhythmias and higher grades of diffuse brain injury (P=0.042) and hyperosmolar therapy use (65%, P=0.022). Hamila et al. [19] further linked ECG changes to specific cerebral pathologies, including brain edema (P=0.043), intracerebral hemorrhage (P=0.033), and subarachnoid hemorrhage (P=0.015).

Temporal dynamics of ECG changes

Hamila et al. [19] characterized the temporal evolution of ECG abnormalities over 72 hours.

Table 1. Summarized evidence from recent studies that evaluate the electrocardiogram abnormalities in patients with traumatic brain injury

Study	Year	Study Design	Sample Size	Mean of age, year ± SD	TBI severity (%)	Time points for ECG	Type of ECG changes	Conclusion
Verma et al.	2024	Retrospective observational study	100 patients	36.69	Mild: 67% Moderate: 15% Severe: 18%	On admission	-Conduction abnormalities: Right bundle branch block, AV block -QRS, ST complex abnormalities: Mostly nonspecific ST changes, old myocardial infarction, acute myocardial infarction -Rhythm abnormalities: Sinus tachycardia -QTc interval abnormalities: Prolonged QTc.	The study found a significant correlation among the severity of head injury, ECG results, and overall outcome.
Srinivasaiah et al.	2024	Prospective observational study	110 patients	43.5	Mild: 24.5% Moderate: 41.6% Severe: 33.6%	On admission and within 48 hours after surgical intervention	-Repolarization abnormalities, prolonged PR interval, QRS prolongation, QTc prolongation, ST-segment depression, ST-segment elevation, and T wave changes.	Preoperative poor LVEF (< 50%) and/or regional wall motion abnormality were associated with lower GCS at discharge.
Ediga et al.	2024	Prospective observational study	100 patients	43.88 ± 15.96	Mild: 55% Moderate: 21% Severe: 24%	On admission	-Prolonged PR interval, QRS prolongation, QTc prolongation, ST-segment depression, ST-segment elevation, and T wave changes.	ECG may serve as an inexpensive screening tool for cardiac dysfunction in isolated TBI patients.
Hamila et al.	2022	Prospective observational study	50 patients	37.8 ± 14.85	Moderate: 46% Severe: 50%	On admission, 24 and 72 hours after admission	-Sinus bradycardia, sinus tachycardia, short PR interval, ST segment elevation, inverted T wave. -Serial ECGs showed significant changes in heart rate and short PR interval over time.	Significant associations were found between ECG changes and brain edema, intracerebral hemorrhage, and subarachnoid hemorrhage.
Lee et al.	2022	Retrospective observational study	1024 patients	63.1	Moderate to severe TBI	On admission	-PR prolongation, QRS complex widening, QTP, ST-segment elevation, ST-segment depression.	The study assessed the association between ECG findings and early mortality, finding that QTP and ST-segment depression were independently associated with 48-hour mortality.
Praveen et al.	2021	Prospective observational study	60 patients	39 ± 13	Mild: 20% Moderate: 73.33% Severe: 6.66%	On admission and within 24-48 hours after surgical intervention	-Repolarization abnormalities, QTc prolongation.	Cardiac issues are common after TBI, with even those suffering from mild TBI showing preoperative systolic dysfunction on echocardiography.
Lenstra et al.	2021	Retrospective observational study	198 patients	40 ± 19	Exclusively severe TBI (median GCS=3)	Within 24 hours after ICU admission	-Ventricular repolarization disorders: Mostly ST-segment abnormalities and T-wave changes -Conduction disorders: Mostly QTc prolongation -Arrhythmias: sinus tachycardia and sinus bradycardia, atrial or AV junctional dysfunction, and ventricular arrhythmias.	In conclusion, a significant prevalence of ECG abnormalities was identified in patients with severe TBI during the acute phase after injury. However, no correlation was found between ECG abnormalities and the location of brain lesions or the presence of thoracic injuries.

AV: Atrioventricular, ECG: Electrocardiogram, GCS: Glasgow coma scale, ICU: Intensive care unit, LVEF: Left ventricular ejection fraction, SD: Standard deviation, TBI: Traumatic brain injury.

At admission, sinus tachycardia affected 38% of the patients, bradycardia 18%, shortened PR intervals 26%, inverted T waves 16%, and ST elevation 5%. By 24 hours, tachycardia decreased to 22%, bradycardia to 5%, PR interval abnormalities to 14%, and T-wave inversions resolved completely. By 72 hours, further reductions were observed: tachycardia to 8%, bradycardia to 5%, and shortened PR intervals to 4%.

Ediga et al. [8] provided insights into the natural history of ECG changes, noting that most abnormalities develop within days post-injury and typically resolve, though some can persist up to eight weeks. They observed that among mild TBI cases, 53% showed normal heart rates (60-100/min), while only 2% exhibited tachycardia (> 100/min). Their findings support the concept that ECG abnormalities are largely transient and improve following resolution of the inciting cerebral events.

Srinivasaiah et al. [18] provided additional insights into postsurgical changes, demonstrating that following surgical decompression, the QTc interval decreased significantly (P=0.01) from a median of 439.5 ms preoperatively to 428.5 ms postoperatively, while MERA abnormalities decreased from 47% to 29% (P= 0.002). Interestingly, the heart rate increased significantly postoperatively (P < 0.01), with new-onset tachycardia observed in 20% of patients (P=0.009). These findings suggest both the natural resolution of many ECG abnormalities over time and the specific effects of surgical intervention on cardiac parameters.

Prognostic value of ECG abnormalities

ECG abnormalities have varying prognostic significance. Lee et al. [14] identified QT prolongation (odds ratio [OR] 2.017, 95% confidence interval [CI] 1.203-3.382) and ST-segment depression (OR 8.428, 95% CI 5.019-14.152) as independent predictors of 48-hour mortality, observed in 8.7% of cases (n=89). The combined predictive accuracy of these abnormalities yielded an area under the curve (AUC) of 0.786 (95% CI 0.759-0.811), comparable to the Revised Trauma Score (RTS) (AUC=0.790, 95% CI 0.764-0.815) and outperforming the Injury Severity Score (ISS).

Ediga et al. [8] emphasized that prolonged QTc intervals after brain injury are associated with sudden cardiac death and correlate with worse neurological outcomes. They found that improvement in QTc interval favors a good prognosis, while persistent, prolonged QTc is a harbinger of ominous outcomes. Their study noted elevated intracranial pressure was linked to prolonged QTc intervals, and emergency decompressive craniectomy reduced QTc in 13 patients, with this effect being more remarkable in survivors.

Srinivasaiah et al. [18] identified a prolonged QTc interval as an independent predictor of left ventricular dysfunction (OR 1.016, 95% CI 1.004-1.030), and patients with preoperative left ventricular dysfunction had significantly lower GCS motor scores at discharge and prolonged hospital stays, although no association with in-hospital mortality was observed. Conversely, Verma et al. [17] found that QTc prolongation did not independently predict prognosis in milder TBI cases. Lenstra et al. [9] reported that arrhythmias were not independent predictors of in-hospital mortality (P= 0.097), with hypotension (P=0.029) and diffuse brain injury (P=0.017) driving a 30% mortality rate.

Impact of neurosurgical intervention

Praveen et al. [20] explored the impact of decompressive craniectomy on ECG abnormalities. Preoperative abnormalities, present in 48.33% of patients, decreased to 13.33% within 24-48 hours postoperatively. Concurrent echocardiographic assessments revealed preoperative systolic dysfunction in 13.33% of cases, with all affected patients showing significant improvement following surgical decompression.

Ediga et al. [8] corroborated these findings, reporting that more than half of their TBI cohort underwent surgery, with cardiac abnormalities recorded intraoperatively including ST-segment depression, T-wave inversion, and improved QTc intervals. They identified surgical decompression as the most important factor in improving cardiac function, with decompressive craniectomy effectively reducing QTc intervals in patients with elevated intracranial pressure. However, they noted that patients who did not survive showed significantly longer QTc intervals even after decompressive craniectomy, suggesting that intracranial hypertension may be an additional risk factor for prolonged QTc.

Srinivasaiah et al. [18] provided complementary evidence with their comprehensive evaluation of both ECG and echocardiographic parameters before and after the surgical decompression. They observed that left ventricular ejection fraction < 50% decreased from 9.8% preoperatively to 2% postoperatively, whereas regional wall motion abnormalities decreased from 10.8% to 4.9%. The mean ejection fraction improved significantly from 62.96 ± 8.55% to 65.58 ± 7.17% (P=0.006) following surgery. These findings collectively suggest that neurosurgical intervention may mitigate both electrical and mechanical cardiac dysfunction, supporting the hypothesis that surgical decompression and reduction of intracranial pressure can reverse neurogenic cardiac injury.

Discussion

The systematic review conducted henceforth carries the premise that ECG abnormalities in patients with TBI are common, with an occurrence range of 10-88%, depending on the study, and are strongly correlated with injury severity. Beyond the recorded incidence, these changes are often neurogenic cardiac injuries caused by mechanisms such as catecholamine surges, autonomic dysregulation, and raised intracranial pressure. In this context, our findings support the concept of a "brain-heart axis", in which the intensity of the cerebral insult is translated into transient but clinically relevant myocardial dysfunction rather than incidental ECG noise. Understanding these ECG changes could help in the management of patient care, as they can be used as noninvasive markers in the identification of individuals at high risk of contracting cardiac complications and timely intervention to optimize patient outcomes in neurology and cardiovascular fields. Hence, performing physician ECGs would significantly stratify risk, adapt multidisciplinary care plans, and introduce alerting mechanisms for the evolution of brain-heart interaction in this vulnerable population. Taken together, these observations suggest that ECG abnormalities should be regarded as integral features of TBI pathophysiology rather than incidental bystanders, and they may represent a potentially modifiable component of secondary injury if identified and managed early.

The prevalence of ECG abnormalities in patients with TBI differs widely depending on the study, ranging from 10% in Verma et al.'s population [17] of mostly mild TBI to 88% in Lenstra et al.'s group [9] of severe TBI with a median GCS score of 3. Other studies found middle values, such as 48.33% of Praveen et al.'s preoperative assessment patients [20] (mostly moderate TBI) and varying incidences reported by Hamila et al. [19] (38% sinus tachycardia at admission). Taken together, this gradient suggests that the burden of ECG abnormalities tracks not only with the binary presence of TBI but with its severity and the acuity of the intracranial process. The prospective study by Srinivasaiah et al. [18] adds to this variability, reporting QTc prolongation in 42% of patients undergoing surgery within 48 hours of injury, which falls within the moderate range of reported abnormalities. Similarly, Ediga et al. [8], in a prospective observational analysis of 100 patients with isolated TBI, observed ECG abnormalities in 53% of mild cases (predominantly normal heart rates of 60-100/min), escalating to higher rates in moderate and severe cases, including prolonged PR intervals (up to 16%), QRS durations (up to 15%), and QTc intervals (up to 15%). This demonstrates how the study design, severity of injury, and timing of assessment might affect the incidence, with higher incidence rates generally observed in the acute phase and severe cases. In clinical terms, this implies that a normal ECG in mild TBI should not be interpreted in the same manner as in severe TBI, and risk stratification should explicitly incorporate the severity of injury. Conversely, the presence of new or evolving abnormalities in milder TBI may indicate a level of systemic stress that is not widely recognized.

These abnormalities can be classified into three major groups: repolarization abnormalities, rhythm disturbances, and conduction abnormalities. Repolarization abnormalities have been reported rather commonly, including QTc prolongation, ST-segment changes, and T-wave abnormalities [9, 14, 17-20]. Among these, QTc prolongation and ST-segment depression appear particularly important because they have repeatedly been linked to worse short-term outcomes, including early mortality, in larger cohorts. Srinivasaiah et al. [18] specifically identified MERA in 47% of their patients, including biphasic T-waves (24%) and two-peaked T-waves (7%), providing detailed characterization of these repolarization changes.

Ediga et al. [8] further corroborated this, noting repolarization issues such as ST-segment elevation/depression, T-wave inversions, and QTc prolongation in 3-15% of cases, with frequencies increasing alongside TBI severity; they emphasized that these repolarization changes, rather than ischemic-like patterns, were most closely linked to true cardiac dysfunction on echocardiography. This pattern supports the interpretation that many of the observed ECG changes reflect diffuse neurogenic myocardial injury rather than primary coronary occlusion, which has practical implications for deciding when to pursue invasive cardiac investigations versus conservative monitoring [21]. Our synthesis also suggests that repolarization changes convey more specific information about neurogenic myocardial injury than rhythm or conduction disturbances, which are more likely to be influenced by external factors such as drugs, electrolytes, or ventilatory status.

Rhythm disturbances ranged from bradyarrhythmias to tachyarrhythmias and premature complexes (the latter infrequently reported, though implied in the series of arrhythmias) [9, 19]. Conduction disturbances refer to situations including changes in the PR interval or QRS widening [9, 19], as seen in Ediga et al. [8] where prolonged PR intervals (8-16%) and QRS patterns (5-15%) were documented, again varying by injury severity. Although these abnormalities are generally less specific than repolarization changes, their presence may still indicate heightened autonomic instability and should prompt closer hemodynamic monitoring, especially in the perioperative period. This grouping depicts the multiphase behavior of ECG changes described as mostly resolving over time in Hamila et al.'s 72-hour serial monitoring [19]: a fall in tachycardia from 38% to 8%, bradycardia from 18% to 5%, and PR abnormalities from 26% to 4%, which is a hallmark of a transient neurogenic rather than a permanent insult. Such reversibility suggests that timely control of intracranial pressure and sympathetic overactivity might mitigate or even prevent sustained cardiac sequelae in a subset of patients [14, 22]. From a pragmatic standpoint, this means that in TBI care pathways, priority should be given to systematic evaluation of QTc and ST-T morphology, while rhythm and conduction changes should trigger targeted evaluation for potentially reversible systemic contributors.

A uniform correlation was observed between the severity of TBI and the degree of ECG abnormalities, with greater changes in more severe cases. Research such as Lenstra et al. [9] and Lee et al. [14], involving severe TBI, cited higher prevalence rates (up to 88%) than Verma et al.'s milder population (67% mild TBI, with only 10% abnormalities) [17]. Lenstra et al. [9] directly correlated arrhythmias with increased grades of diffuse brain injury and hyperosmolar therapy administration (65%), whereas Hamila et al. [19] correlated ECG changes with particular pathologies, such as brain edema, intracerebral hemorrhage, and subarachnoid hemorrhage. Srinivasaiah et al. [18] further strengthened this relationship by demonstrating that lower GCS motor scores independently predicted left ventricular dysfunction, with patients having severe TBI (GCS 3-8) showing 61.5% prevalence of cardiac dysfunction compared to 0% in mild TBI. This study also identified a prolonged QTc interval as another independent predictor of cardiac dysfunction, establishing a direct mechanistic link between specific ECG abnormalities and functional cardiac impairment. Ediga et al. [8] provided additional evidence, reporting a gradient of abnormalities - prolonged QTc in 3% of mild, 10% of moderate, and 15% of severe cases - directly associating repolarization changes with injury severity and echocardiographic evidence of cardiac dysfunction, such as RWMA in 23% and diastolic dysfunction in 45% of cases. This study also identified specific patterns like left ventricular hypertrophy and valvular involvement (e.g., mitral in 25% of mild cases, aortic in 9% of severe cases), reinforcing the link between severe TBI and multifaceted cardiac impairment. Interestingly, Lenstra et al. [9] did not find any correlation with lesion location, highlighting that the extent of injury could outweigh anatomical specificity. That being said, the relationship is not absolute; even mild TBI can have cardiac dysfunction, as witnessed in the findings of Verma et al. [17] where QTc prolongation was noted in some milder instances, albeit without significant prognostic correlations and in Ediga et al. [8] where mild cases showed preserved ejection fractions (55-70% in 54%) but subtle repolarization issues. This indicates that ECG changes can potentially occur along the spectrum of severity, possibly due to subtle autonomic disruption, although correlations with lower GCS scores (denoting severe injury) are stronger. From a clinical per-

spective, this endorses a stratified methodology wherein ECG abnormalities in severe TBI are regarded as high-risk indicators, while abnormalities in moderate TBI necessitate focused yet more cautious assessment. These converging lines of evidence underscore a significant conceptual transformation: rather than perceiving cardiac dysfunction as an isolated comorbidity in TBI, it may be more beneficial to regard it as an additional organ-level manifestation of overall injury severity, akin to pulmonary or renal involvement in multiorgan failure.

Surgical procedures also modulate this relationship, with Praveen et al. [20] showing a decrease in ECG abnormalities from 48.33% preoperatively to 13.33% postoperatively following decompressive craniectomy in moderate-to-severe TBI (median GCS=11), in concert with improvements in echocardiographic systolic dysfunction. Srinivasaiah et al. [18] provided more granular evidence of surgical effects, demonstrating that QTc prolongation significantly decreased from 42% preoperatively to 33% postoperatively, and MERA abnormalities decreased from 47% to 29%. Paradoxically, they also observed an increase in sinus tachycardia from 15% to 29% postoperatively, which they attributed to the unmasking of catecholamine surge effects following the intracranial pressure reduction. This nuanced finding suggests that while some ECG abnormalities improve with surgical decompression, others may temporarily worsen as the physiological response to brain injury becomes more apparent. Hamila et al. [19] also observed surgical effects on heart rate and PR interval, supporting the contribution of intracranial pressure reduction in decreasing severity-related cardiac impacts. Ediga et al. [8] aligns with these observations by noting that repolarization abnormalities, prevalent in non-operated isolated TBI cases, warrant surgical consideration for risk mitigation, though their study focused on non-surgical patients and highlighted ECG's role in early screening. Clinically, these findings suggest the inclusion of ECG trends in perioperative decision-making. If repolarization abnormalities persist or worsen despite adequate decompression, it could indicate a particular group with a high risk, which could benefit from increased cardiology involvement and extended intensive monitoring.

The integration of ECG findings with echocardiographic assessment provides a more comprehensive evaluation of neurogenic cardiac injuries. Srinivasaiah et al. [18] uniquely combined both modalities and revealed that 10% of patients had reduced left ventricular ejection fraction (< 50%) and 10.8% had RWMA preoperatively, with both parameters improving following surgical decompression. This study demonstrated that patients with preoperative cardiac dysfunction had significantly lower GCS scores at discharge (median, 9 vs. 12) and longer hospital stays (median, 5 vs. 2 days), establishing cardiac dysfunction as a marker of overall injury severity and recovery potential. Ediga et al. [8] extended this integration, reporting normal ejection fractions (55-70%) in most cases but identifying RWMA in 23%, diastolic dysfunction in 45%, and valvular dysfunction in 39%, with repolarization ECG changes (e.g., prolonged QTc) strongly associated with these echo findings. The combined ECG-echocardiographic approach in Ediga et al. [8] and Srinivasaiah et al. [18] showed that prolonged QTc intervals and reduced GCS motor scores could predict functional cardiac impairment with high accuracy (up to 89.5%), providing a practical screening tool for identifying high-risk patients, particularly those with isolated TBI or subarachnoid hemorrhage. In practice, this indicates that the systematic integration of ECG and targeted echocardiography during the initial phase of TBI may facilitate the triage of patients into various monitoring and treatment protocols (e.g., intensive telemetry versus normal ward care). This combined ECG-echocardiography technique, in our opinion, is especially appealing in poor and middle-income countries where advanced neuromonitoring may be unavailable but basic ultrasound and ECG are available.

While ECG parameters are promising for evaluating the possibilities of death and morbidity in patients with TBI, there remains inconsistency. In a large retrospective study conducted by Lee et al. [14], QTc prolongation and ST-segment depression were said to independently predict death within 48 hours (observed in 8.7% of cases), with a combined AUC of 0.786, which is similar to the RTS (AUC=0.790) and superior to the ISS. Hence, repolarization abnormalities compete with well-recognized trauma scoring systems in predicting early adverse outcomes

in TBI patients. Srinivasaiah et al. [18] extended this prognostic utility by showing that preoperative cardiac dysfunction was associated with poorer neurological outcomes at discharge, but not with in-hospital mortality in their cohort. Ediga et al. [8] similarly positioned repolarization changes as predictors of cardiac dysfunction rather than mortality, noting their resolution potential and utility in risk stratification. This suggests that ECG abnormalities may be more useful for predicting functional recovery than acute mortality in certain populations. Conversely, Verma et al. [17] denied any independent prognostic role of QTc prolongation in milder TBI, while Lenstra et al. [9] determined that arrhythmias were not independently related to in-hospital mortality, with factors such as hypotension and diffuse injury claiming a 30% mortality rate. Overall, although some ECG abnormalities, such as QTc prolongation and ST-segment depression, have great potential for predicting short-term mortality, their role in assigned disparity (e.g., long-term cardiac dysfunction) has yet to be validated, especially in serial monitoring. The available data indicate a dual use for ECG: repolarization markers like QTc and ST depression may aid in forecasting early mortality in severe injuries, while comprehensive ECG-echo patterns may provide greater insight into functional recovery and long-term cardiac risk. Subsequent prospective studies must to concentrate on the standardized assessment of QTc and ST segment variations over time, as well as on correlating these trajectories with both cardiac and neurological outcomes.

ECG monitoring provides a cheap, noninvasive, and readily accessible screening procedure for cardiac dysfunction in victims of TBI, thus complementing more expensive modalities such as echocardiography. Routine ECG screening can identify higher-risk patients early, such as those exhibiting QTc prolongation or ST-segment depression, both of which are associated with an increased risk of mortality and functional cardiac impairment [14, 18]. The practical application of ECG screening is further enhanced by the finding that simple parameters, such as the QTc interval and GCS motor score, can predict cardiac dysfunction with high accuracy, making this approach feasible even in resource-limited settings. This would occur mainly in resource-limited settings, where ECG can herald the presence of subtle neurogenic cardiac injury before clinical decompensation becomes evident. Combined with trauma scoring systems, including the ISS and RTS, ECG findings improve diagnostic precision above and beyond that provided by repolarization abnormalities alone for risk stratification and triage of acute TBI care. Based on this, we suggest that ECG results should be included to existing trauma ratings instead of being utilized on their own. Even simple composite indices that combine GCS, QTc, and essential radiologic aspects could be better than single-modality instruments and should be tested in the future.

The high prevalence and dynamic nature of ECG abnormalities necessitate routine cardiac monitoring in patients with TBI, particularly in the acute phase (within 72 hours post-injury), to track temporal changes and guide interventions. Serial ECG assessments can serve as a surrogate for neurological recovery and inform ongoing management, especially for patients with severe TBI or specific pathologies (e.g., brain edema or hemorrhage), where changes correlate with injury progression [9, 19]. The findings of Srinivasaiah et al. [18] and Ediga et al. [8] support the utility of postoperative ECG monitoring, as they demonstrated differential patterns of improvement and deterioration in various ECG parameters following surgical intervention. In terms of anesthetic and surgical management, ECG abnormalities have direct implications, such as the heightened risk of arrhythmias during procedures. Studies by Praveen et al. [20] and Srinivasaiah et al. [18] and Ediga et al. [8] highlighted how surgical interventions (e.g., decompressive craniectomy) can mitigate cardiac risks by reducing preoperative abnormalities and associated systolic dysfunction, suggesting that timing and intracranial pressure reduction are key; thus, anesthesiologists should prioritize ECG-guided optimization, including electrolyte correction and beta-blocker use for tachyarrhythmias. Perioperative care should incorporate multidisciplinary strategies, such as continuous ECG monitoring in intensive care units, tailored hemodynamic support (e.g., for bradycardia or QTc prolongation), and vigilant screening for confounders, such as medications or electrolyte imbalances. The identification of specific predictors, such as prolonged QTc intervals and low GCS motor

scores, can help clinicians implement enhanced hemodynamic monitoring protocols for high-risk patients, potentially improving both cardiac and neurological outcomes. These considerations could reduce morbidity, particularly in high-risk cohorts, by preventing cardiac complications that exacerbate the neurological outcomes.

However, our systematic review has some limitations. Methodological limitations among the studies, including variations in design, variations in sample sizes (n=50-1,024 patients), and injury severities, also cause inconsistencies in the findings. Different ways to account for confounders, such as pre-existing cardiac conditions, electrolytes, or medications, further complicate interpretations. Another hindrance is the outcome measure employed and estimation of the sample size. In addition, we relied on recently published studies in adults and in English, which may introduce publication and language bias and limit generalizability to pediatric or low-resource settings. Future research should incorporate standardized ECG protocols, predefined cardiac endpoints, and longer follow-up to more clearly delineate which ECG patterns are transient epiphenomena and which truly modify prognosis. Such data would ultimately allow clinicians to move from descriptive use of ECG patterns toward evidencebased, protocolized management strategies in TBI.

Conclusion

In conclusion, ECG abnormalities are prevalent in TBI, with their occurrence dependent on the severity of the injury and significant prognostic implications. Notably, repolarization changes, such as QTc prolongation and ST-segment depression, are critical predictors of mortality. The temporal resolution of these abnormalities and their responsiveness to interventions underscore the potential of integrated brain-heart management strategies. Future research should focus on the development of standardized protocols, inclusion of larger cohorts, and mechanistic studies to further elucidate the role of ECG in optimizing outcomes for patients with TBI.

Disclosure of conflict of interest

None.

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