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Incidence and Clinical Impact of Myocardial Injury Following Traumatic Brain Injury: A Pilot TRACK-TBI Study

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Abstract

Background: Traumatic brain injury (TBI) is a major global health problem. Little research has addressed extracranial organ dysfunction following TBI, particularly myocardial injury. Using a sensitive marker of myocardial injury - high sensitivity troponin (hsTn) - we examined the incidence of early myocardial injury following TBI and explored its association with neurologic outcomes following moderate-severe TBI.

Methods: We conducted a pilot cohort study of 133 adult (age > 17 years) subjects enrolled in the TRACK-TBI 18-center prospective cohort study. Descriptive statistics were used to examine the incidence of myocardial injury (defined as hsTn > 99th percentile for a standardized reference population) across TBI severities, and to explore the association of myocardial injury with 6-month extended Glasgow outcome score (GOS-E) among patients with moderate-severe TBI.

Results: The mean (standard deviation) age of the participants was 44 (17) years, and 87 (65%) were male. Twenty-six patients (20%) developed myocardial injury following TBI; myocardial injury was present in 15% of mild TBI patients and 29% of moderate-severe TBI patients (p=0.13). Median (interquartile range) hsTn values were 3.8 ng/L (2.1, 9.0), 5.8 ng/L (4.5, 34.6) and 10.2 ng/L (3.0, 34.0) in mild, moderate and severe TBI participants, respectively (p=0.04). 11 % of participants with moderate-severe TBI and myocardial injury experienced a good outcome (GOS-E 5) at six months, compared with 65% in the group that did not experience myocardial injury (p=0.01).

Conclusions: Myocardial injury is common following TBI, with a likely dose-response relationship with TBI severity. Early myocardial injury was associated with poor six-month clinical outcomes following moderate-severe TBI.

Keywords

myocardial injury; trauma; traumatic brain injury; high sensitivity troponin; outcome

Introduction:

Traumatic brain injury (TBI) is a major cause of death and disability in the United States¹ and globally. While significant research has examined the consequences of primary injury on the brain, comparatively little research has been undertaken to understand the impact of TBI on extracranial organ dysfunction, which commonly occurs in patients with moderate and severe TBI²⁻⁴. Among the spectrum of extracranial organ dysfunction, cardiac dysfunction, hypotension and circulatory shock occurs in over 50% of patients with severe TBI, which reduces cerebral blood flow if autoregulation is impaired, and leads to poor clinical outcomes^{5,6}. The mechanism by which circulatory shock occurs is largely unknown, but potentially involves a fulminant surge in catecholamine levels resulting in myocardial injury and cardiac dysfunction^{7,8}.

While prior studies have examined acute myocardial injury following TBI^{9,10}, these have primarily focused on severe TBI patients and used conventional troponin assays which limit investigation of subclinical myocardial injury. The recent introduction of high sensitivity troponin (hsTn) assays provides a new tool for improved characterization of myocardial injury¹¹ and an opportunity to investigate the relevance of subclinical myocardial injury (identified using hsTn) on clinical outcomes following TBI. High sensitivity troponin assays measure up to 10-fold lower concentrations of troponin than conventional troponin assays, with greater precision^{12,13}. They allow improved detection of myocardial injury in non-cardiac conditions¹⁴, and afford an opportunity to more accurately quantify myocardial injury following TBI. The burden of myocardial injury across the spectrum of TBI severities, as well as its association with clinical outcomes, represents a gap in the TBI literature. To address this knowledge gap, we conducted a pilot study to: 1) examine the incidence of myocardial injury across TBI severities, and 2) explore the association between myocardial injury and neurologic outcomes following moderate-severe TBI.

Methods

The study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and was approved by the Institutional Review Board at Duke University (Pro00100061).

Study design, database and population

We conducted a pilot cohort study by performing a secondary analysis of 133 adult (age > 17 years) subjects enrolled in the Transforming Clinical Research and Knowledge in TBI (TRACK-TBI) study. TRACK-TBI is an 18-center prospective cohort study of patients evaluated in participating level 1 trauma center emergency departments within 24 hours of experiencing blunt TBI, and for whom a clinically indicated head computerized tomography (CT) scan was obtained by the treating clinician¹⁵. In addition to the collection of detailed hospital encounter data, the study also collected baseline (and serial) blood samples and a multidimensional outcome assessment battery across the year post-injury. Subjects were excluded from TRACK-TBI if they had significant extracranial injury or significant history of pre-existing conditions that would interfere with follow-up and outcome assessment. In addition, subjects were also excluded if they were prisoners or patients in custody, pregnant, on psychiatric hold, had major debilitating baseline mental health disorders or major debilitating neurologic disease, were participants in an interventional trial, or had penetrating TBI or spinal cord injury with American Spinal Injury Association impairment score of C or worse. All data were obtained from participants by trained research coordinators using structured data collection tools recommended as part of the National Institute of Neurological Disorders and Stroke Common Data Elements for TBI studies.¹⁶

Exposure, outcomes, and covariates

For our first research aim, the exposure was TBI severity and outcome was myocardial injury. TBI severity was categorized as mild (defined as Glasgow Coma Scale [GCS] score 13 after resuscitation), moderate (GCS 9–12 after resuscitation), and severe (GCS 8 after resuscitation). Myocardial injury was defined as a single hsTn value (obtained either

at study enrollment or within four hours following enrollment) greater than the upper reference limit, corresponding to > 99% for a standardized reference population. For our second research aim, we restricted the population to subjects with moderate-severe TBI. The exposure was the presence of myocardial injury, and the outcome of interest was 6-month extended Glasgow Outcome Score (GOS-E), with good outcome defined as GOS-E 5–8 and poor outcome defined as GOS-E 1–4. We also examined additional covariates, including demographic (age, gender, race) and clinical (injury mechanism, injury severity score, initial head CT findings, and Rotterdam score based on initial head CT) parameters.

Statistical analysis

Descriptive statistics were used to examine the demographic and clinical characteristics of the study cohort, stratified by the presence of myocardial injury. Group comparisons used Fisher's exact tests for categorical variables and Wilcoxon Rank Sum tests for continuous variables. Baseline hsTn values were summarized with median and interquartile range (IQR), and compared across TBI severity groups (mild, moderate, severe) using the Kruskal Wallis test. Lastly, we compared clinical and functional outcomes, including hospital discharge status (alive vs. dead) and GOSE (1–4 vs. 5–8) at six months post-injury between the groups with and without myocardial injury among the moderate-severe TBI (GCS<13) cohort. Statistical software R (version 3.6.1) was used for the analysis (<http://www.r-project.org>).

Results

Demographic and clinical characteristics of the 133 participants included in the study cohort, stratified by the presence of myocardial injury, are shown in Table 1. The mean(SD) age of the participant cohort was 44 (17) years; among these, 87 (65%) were male and 109 (82%) were of white race. The primary mechanism of injury was road traffic accidents (65%), followed by falls (20%), and violence/assault (10%). Sixty participants (56%) had evidence of blood on initial head CT. Twenty six (20%) participants developed myocardial injury following TBI. Compared with participants that did not develop myocardial injury, those who developed myocardial injury had a higher mean (SD) injury severity score (20.8 (8.8) vs. 14.7 (9.4), respectively; $p=0.003$) and higher Rotterdam score on initial head CT scan (3.4 (1.4) vs. 2.5 (0.9), respectively; $p=0.004$).

When stratified by TBI severity, myocardial injury was present in 15% of participants with mild TBI and 29% of participants with moderate-severe TBI ($p=0.13$). The distribution of baseline hsTn, stratified by TBI severity, is shown in Figure 1. Median (IQR) hsTn value was 3.8 ng/L (2.1, 9.0) in mild TBI participants, 5.8 ng/L (4.5, 34.6) in moderate TBI participants, and 10.2 ng/L (3.0, 34.0) in severe TBI participants ($p=0.04$). Among the 35 participants with moderate-severe TBI, 34 had hospital disposition data. Four of 10 (40%) participants in the group with myocardial injury experienced in-hospital mortality, compared with 1 of 24 (4.2%) who did not experience myocardial injury following moderate-severe TBI ($p=0.02$). Among the 35 participants with moderate-severe TBI, 29 had GOS-E assessed at 6-months post-injury. One of 9 (11%) in the group with myocardial injury experienced a good outcome (GOS-E 5) at six months, compared with 13 out of 20 (65%)

with good outcome who did not experience myocardial injury following moderate-severe TBI ($p=0.01$).

Discussion

In this pilot study examining the incidence of myocardial injury across TBI severities using hsTn we found that: 1) myocardial injury is relatively common following TBI and occurs across all TBI severities; 2) there is a possible dose-response relationship of TBI severity with the risk and degree of myocardial injury; and 3) among patients with moderate-severe TBI, the development of myocardial injury was associated with poor clinical outcomes at six months post-injury. Thus, myocardial injury following TBI is a common and possibly clinically significant problem.

Prior retrospective studies of troponin following TBI have reported acute myocardial injury in approximately 30% of severe TBI patients^{9,10,17}. However, these findings are susceptible to selection bias since they are derived from studies in which troponin measurements were performed at the discretion of treating clinicians. Furthermore, these studies focused primarily on severe TBI patients and used conventional troponin assays which have limited sensitivity for detection of subclinical myocardial injury. Our data suggests that myocardial injury occurs across the spectrum of TBI severities (mild, moderate, and severe), with a possible dose-response relationship between TBI severity and degree of myocardial injury. Furthermore, our data suggest that the development of early myocardial injury following TBI is clinically relevant, as it is associated with clinical and functional outcomes at six months after injury.

Myocardial injury has been observed to result in reduced cardiac function in critical illness, but this relationship has not been studied in TBI patients. Cerebral autoregulation can be impaired after TBI^{18–27}; therefore, cerebral ischemia can occur when cerebral perfusion is not maintained during hypotension. In this situation, early myocardial injury may contribute to the development of cardiac dysfunction, shock, cerebral hypoperfusion and secondary brain injuries following TBI. Therefore, preventing post-TBI myocardial injury might mitigate hypotension, improve cerebral perfusion and CBF in the setting of TBI-related impaired autoregulation; this is an intriguing hypothesis to test in future studies.

We recognize several limitations to our study. First, the data are from a pilot sample of participants in the TRACK-TBI study and are thus limited by a small sample size, specifically among participants with moderate and severe TBI. Furthermore, this may contribute to selection bias in the population ascertained for this study. Second, our sample was primarily of white race and male gender TBI patients, limiting the generalizability of our findings of myocardial injury to non-white populations and females. Third, the clinical value of an isolated organ injury biomarker may be limited when other organ parameters are not evaluated concurrently; for example, the presence of myocardial injury defined by elevated hsTn may not fully capture the extent of cardiac dysfunction and should ideally be coupled with an echocardiogram to examine cardiac function. Unfortunately, this approach was not possible given the retrospective nature of this study, and future studies should take a multidimensional perspective into account when ascertaining organ injury.

Lastly, given the pilot nature of our study, the results must be considered exploratory and hypothesis-generating, with confirmation only possible in future studies examining larger and more heterogeneous TBI populations.

In conclusion, we found that myocardial injury is common across TBI severities, with a likely dose-response relationship with TBI severity. Furthermore, the presence of myocardial injury was associated with poor six-month clinical outcomes following moderate-severe TBI. Our data support the observation that myocardial injury is a common and clinically significant problem following TBI. Further studies are necessary to confirm our findings, examine mechanisms, and improve clinical outcomes in this population.

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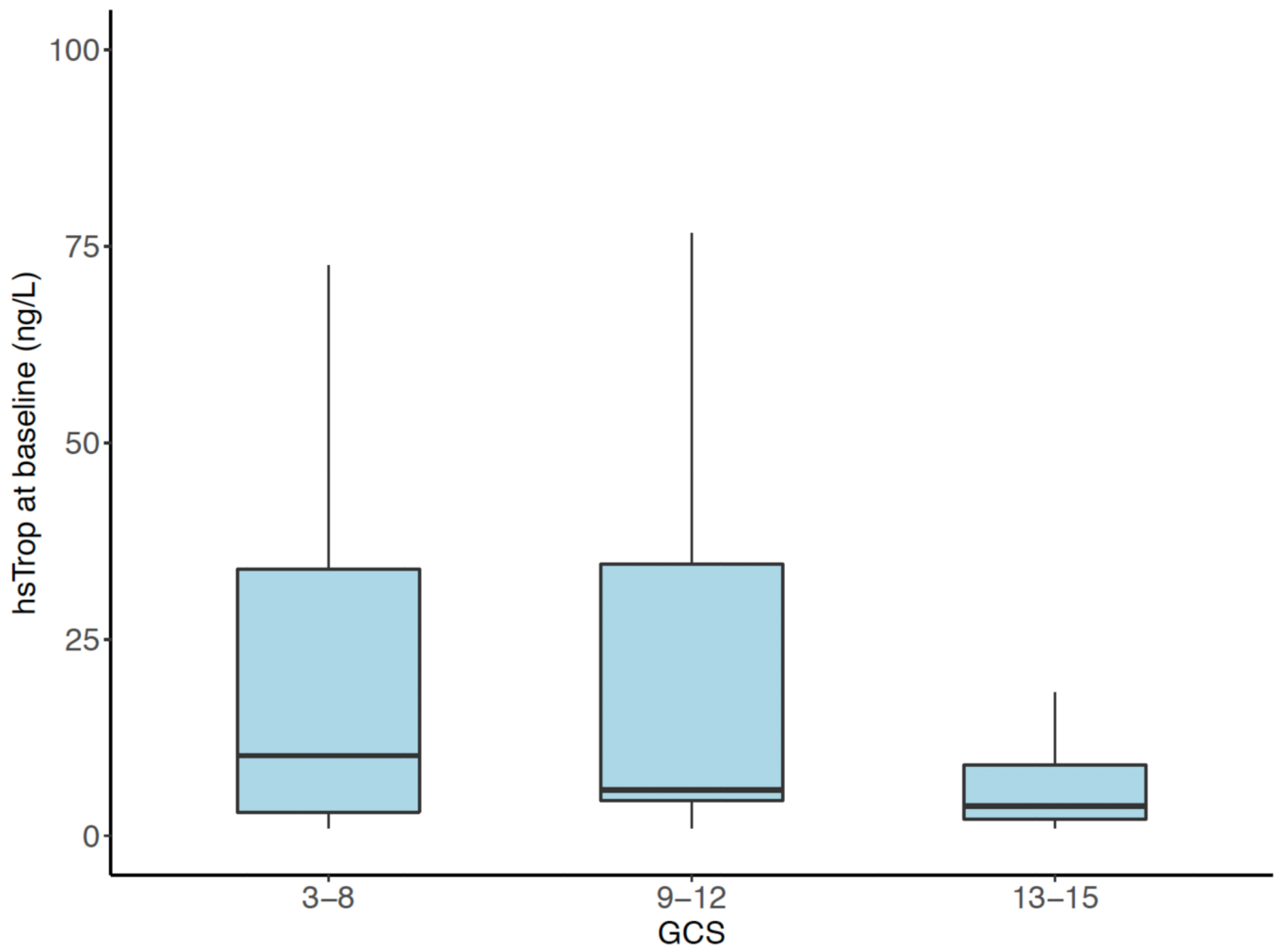


Figure 1. Distribution of baseline high sensitivity troponin, stratified by traumatic brain injury severity

Table 1

Demographic and clinical characteristics

	No myocardial Injury (n=107)	Myocardial Injury (n=26)	p-value
Age (years) ¹	42.5 (17.1)	49.0 (18.0)	0.10
Male gender ²	70 (65.4%)	17 (65.4%)	>0.99
Race ^{2,3}			0.73
White	89 (83.2%)	20 (80.0%)	
Black	13 (12.2%)	3 (12.0%)	
Other	5 (4.7%)	2 (8.0%)	
Injury mechanism ²			>0.99
Road traffic incident	68 (63.6%)	18 (69.2%)	
Fall	21 (19.6%)	5 (19.2%)	
Violence/Assault	11 (10.3%)	2 (7.7%)	
Other	7 (6.5%)	1 (3.9%)	
Emergency department GCS ⁵	15 (13,15)	14 (3,15)	0.13
Injury severity score ^{1,4}	14.7 (9.4)	20.8 (8.8)	0.003
Presence of blood on initial head CT scan ²	60 (56.1%)	19 (73.1%)	0.13
Rotterdam score ¹	2.5 (0.9)	3.4 (1.4)	0.004

¹ Mean (SD)² Count (percentage)³ Column may not add to total due to missing value⁴ Only available for hospitalized subjects (n=125)⁵ Median (IQR)

CT, computerized tomography; GCS, Glasgow coma scale