

Cardiac injury in severe head trauma: a review of literature

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ABSTRACT

Neurogenic stress cardiomyopathy (NSC) is a stress induced cardiomyopathy reported in various neurological disorders. The most widely accepted theory for the mechanism of NSC is the "catecholamine hypothesis". The available evidence suggests the presence of NSC in patients with severe TBI. The presence of cardiac injury could be a poor prognostic finding in patients with TBI. The possible cardiac injury in TBI patients would make the critical care physicians more cautious with hemodynamic management of these patients. Larger studies with more sophisticated assessment would help to confirm the presence of cardiac injury in these patients.

Introduction

Neurogenic stress cardiomyopathy (NSC) is a stress-related cardiomyopathy that was reported in different neurological conditions. NSC was described as a brain-heart interaction; it was frequently reported in subarachnoid hemorrhage (SAH)^{1,2}; NSC was also reported in cerebrovascular strokes^{3,4}, status epilepticus⁵, brain tumors⁶, central nervous system infections⁷, and even with emotional stress⁸.

The incidence of NSC in patients with traumatic brain injury (TBI) is not clear; NSC was documented in TBI in few case series⁹⁻¹¹. Four observational studies investigated the incidence of cardiac injury in patients with TBI (table 1); however, the results of these studies were not consistent. Although the four studies reported ECG changes and elevated serum troponin suggesting the presence of cardiac injury, the presence of functional echocardiographic abnormalities was variable among the four studies.

The current evidence for NSC

The first study to report NSC in patients with TBI was a retrospective study conducted by Hüttemann et al¹² on 51 patients with severe TBI. In the aforementioned study, cardiac dysfunction was reported using trans-esophageal echocardiography in 15% of patients. In another retrospective study, Prathep and colleagues¹³ investigated the presence of cardiac injury among a larger number of patients (139 patients) with variable grades of TBI. Prathep et al reported abnormal echocardiography in 22.3% and elevated serum troponin in 24% of patients; the third study was conducted Serri et al¹⁴ on 49 patients with severe TBI; Serri et al did not report significant cardiac injury among their cohort. The fourth study was conducted by our group¹⁵ on 50 patients with severe TBI; we reported the presence of cardiac injury among 50% of our patients.

First author, year	Patients	Study type	Evaluation	Main results
Hüttemann, 2002 ⁽¹²⁾	51 patients with severe TBI (50% brain dead)	Retrospective	TEE	LV dysfunction in 8% of patients
Prathep, 2014 ⁽¹³⁾	150 patients with mild, moderate, and severe TBI	Retrospective	TTE, troponin and BNP	RWMA in 16% of patients, LV dysfunction in 12% of patients
Serri, 2016 ⁽¹⁴⁾	49 patients with severe TBI	Prospective	TTE, ECG, and troponin	No LV dysfunction
Hasanin, 2016 ⁽¹⁵⁾	50 patients with severe TBI	Prospective	TTE, ECG, and troponin	LV dysfunction in 28% of patients

BNP: brain natriuretic peptide, ECG: Electrocardiograph, LV: left ventricular, RWMA: Regional wall motion abnormalities, TBI: Traumatic brain injury, TEE: Trans-esophageal echocardiography, TTE: Transthoracic echocardiography

Table 1: Summary for studies reporting cardiac injury in TBI.

Many reasons could explain the higher incidence of cardiac injury in Hüttemann study¹² and Hasanin et al study¹⁵ when compared to Prathep et al study¹³; although the later study included more number of patients, only 56% of these patients had severe TBI; moreover, the retrospective design of the later study would question its ability to find the incidence of NSC because the echocardiographic examination was conducted according to the clinical need and not according to the study design; thus, some patients with possible cardiac injury might be missed.

Although Serri et al¹⁴ reported abnormal ECG and troponin elevation in a considerable number of their patients, they did not report any significant echocardiographic abnormalities; thus, their findings differed from Hasanin et al¹⁵ findings; this diversity could not be explained by any difference in the methodology; both studies included nearly the same number, age, and type of pathology. In our study, we included severely ill patients with mean APACHE II score of 21, Serri et al did not report the APACHE scores of their patients; however, they reported injury severity score of 32 which is a severe grade of injury. The main difference between the two studies is in the timing of echocardiographic examination which is delayed (49 hour) in Serri et al patients.

The four main studies reporting cardiac injury in TBI were characterized by a relatively small sample size; thus, we encourage larger studies with more sophisticated cardiac assessment including more echocardiographic details and more biomarkers.

Mechanism of NSC

The most widely accepted theory for NSC is the “catecholamine hypothesis” which is defined as “catecholamine-mediated direct cardiac injury”. Autonomic stimulation caused by direct brain injury is responsible for NSC. Elevated levels of catecholamines were reported in patients with SAH^{16,17} and in cases of cardiac stunning after emotional stress⁸. A similar troponin elevation was also reported in an animal experimental model of SAH¹⁸. In the aforementioned study, there was a positive correlation between cardiac biomarkers (troponin I and CK-MB) and serum catecholamines (adrenaline and noradrenaline)¹⁸.

The role of elevation of serum catecholamines in the pathogenesis of cardiac injury in CNS conditions is supported by pathologic features of catecholamine-induced injury in myocardial biopsy specimens taken from brain-dead patients who were donors for solid organ transplantation¹⁹, and the absence of features of myocardial necrosis in the postmortem specimens taken from patients with SAH who received Propranolol and phentolamine²⁰. This role is also confirmed by a study reporting normal coronary angiogram in patients with SAH and elevated ST segment with reversible RWMA²¹.

The most widely used biomarker in diagnosis of NSC is serum troponin I. Serum troponin elevation was a clue marker in diagnosis in NSC in patients with TBI¹³⁻¹⁵, CVS^{3,4}, and SAH^{16,22}. Serum troponin showed a sensitivity of 100% and specificity of 91% in prediction of cardiac injury in patients with SAH. Brain natriuretic peptide (BNP) was another biomarker used by Prathep et al¹³ in assessment of NSC in patients with TBI. More biomarkers are encouraged for assessment of NCS in different patient populations.

Cardiac injury and patient outcomes

The relation between cardiac injury and outcome was reported in different CNS conditions such as SAH, CVS, and TBI. The severity of cardiac injury was a risk factor of mortality in many reports. In patients with SAH, there was an association between ECG abnormalities and outcomes^{23,24}; there was also an association between left ventricular dysfunction and mortality²⁵. In a large retrospective study on 4695 patients with SAH, Zarof et al²⁶ reported an increased mortality and stroke rates in patients with cardiac injury compared to patients without cardiac injury. In patients with cerebrovascular strokes, there was an association with serum troponin and patients’ outcome^{3,4,27}. In TBI there was an association between cardiac injury and poor outcomes^{13,15}; moreover, a neurogenic cardiac injury score (NCIS) was developed by our group to grade the severity of cardiac injury, the score was based upon the presence of one or more of three components: elevated serum troponin, abnormal echocardiogram, and hypotension. We reported that increased NCIS is an independent risk factor for mortality¹⁵.

Management and clinical implications of NSC

Although cardiac protection using propranolol and phentolamine was suggested by Neil-Dwyer et al²⁰, they only reported the absence of histopathological necrosis in the post-mortem specimens of the patients receiving the drugs without any improvement in the outcomes. It was suggested that the severity of cardiac injury is related to the severity of head injury; thus, management of cardiac injury might not improve the patient outcome; however, this suggestion should be confirmed in future trials. The awareness with cardiac injury in patients with TBI would be made by the anesthetists and critical care physicians more cautious when dealing with these patients especially with induction of anesthesia as well as sedation in the intensive care units. Risk stratification of TBI patients should not ignore the presence of cardiac injury as an independent risk factor for poor outcomes.

In conclusion, the available collective evidence suggests the presence of cardiac injury in patients with severe TBI. The presence of cardiac injury could be a poor prognostic finding. Larger studies with more sophisticated measures would help to confirm the presence of cardiac injury in these patients and would also investigate its mechanism.

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