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Blunt cardiac injury in critically ill trauma patients: A single centre experience

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ABSTRACT

Purpose: This study describes the incidence and outcomes of blunt cardiac injury (BCI) in a single trauma intensive care unit (TICU), together with the spectrum of thoracic injuries and cardiac abnormalities seen in BCI.
Methods: We performed a retrospective observational study of 169 patients with blunt thoracic trauma admitted from January 2010 to April 2013. BCI was diagnosed using an elevated serum troponin in the presence of either clinical, ECG or transthoracic echocardiography (TTE) abnormalities in keeping with BCI. The mechanism of injury, associated thoracic injuries and TTE findings in these patients are reported.
Results: The incidence of BCI among patients with blunt thoracic trauma was 50% ($n = 84$). BCI patients had higher injury severity scores (ISS) (median 37 [IQR 29–47]; $p = 0.001$) and higher admission serum lactate levels (median 3.55 [IQR 2.4–6.2], $p = 0.008$). In patients with BCI, the median serum TnI level was 2823 ng/L (IQR 1353–6833), with the highest measurement of 64950 ng/L. TTEs were performed on 38 (45%) patients with BCI, of whom 30 (79%) had abnormalities. Patients with BCI had a higher mortality (32% vs. 16%; $p = 0.028$) and trended towards a longer length of stay (17.0 days [standard deviation (SD) 13.5] vs. 13.6 days [SD 12.0]; $p = 0.084$).
Conclusions: BCI was associated with an increased mortality and a trend towards a longer length of stay in this study. It is a clinically relevant diagnosis which requires a high index of suspicion. Screening of high risk patients with significant blunt thoracic trauma for BCI with serum troponins should be routine practise. Patients diagnosed with BCI should undergo more advanced imaging such as TTE or TOE to exclude significant cardiac structural injury.

Introduction

Blunt cardiac injury (BCI) describes the myriad of cardiac lesions that can occur following blunt chest trauma. These range from minor arrhythmias to cardiac rupture [1]. The diagnosis of blunt cardiac injury in polytrauma can be difficult due to multiple distracting injuries that occur with high velocity chest trauma. The lack of definitive diagnostic criteria for blunt cardiac injury has also hampered efforts to identify the incidence of this condition [2,3].

The modalities commonly used to diagnose BCI include clinical or surgical findings, the use of electrocardiograms (ECG), serum cardiac troponin I (TnI) or T (TnT) and cardiac imaging (either transthoracic echocardiography (TTE) or transoesophageal echocardiography (TOE). A combination of these modalities may be required for diagnosis [2]. Serum cardiac troponin levels (either I or T) are increasingly being used as a diagnostic tool for patients who have sustained blunt thoracic trauma [4].

In this study, we describe our experience of BCI in a single trauma intensive care unit (TICU) with regard to the incidence, outcomes and the spectrum of cardiac abnormalities seen in patients diagnosed with blunt cardiac injury.

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Methods

We conducted a retrospective observational study of patients admitted to the TICU at Inkosi Albert Luthuli Central Hospital (IALCH) in Durban, South Africa, from January 2010 to April 2013. This 10 bedded trauma unit is dedicated for critically injured patients and serves a drainage area of approximately eleven million people. Admissions are either directly from the scene of the incident, or as inter-hospital transfers from facilities lacking the necessary surgical or intensive care expertise to manage the patient. All patients presenting to the unit with blunt thoracic trauma were included in the study irrespective of age. This included patients with either isolated thoracic trauma, or multisystem trauma that included thoracic trauma. Patients with penetrating thoracic injuries, those with blunt non-thoracic injuries, and those declared dead on arrival to the resuscitation area were excluded from the analysis.

The trauma unit database and hospital information systems (Medicom® and Soarian®) were used to retrieve patient data for this analysis. We extracted:

1. Patient demographics: age, sex, mechanism of injury, Injury Severity Score (ISS) and site of referral (either scene or inter-hospital transfer).
2. Initial lactate and serum TnI or TnT measurement if performed.
3. Presence of pulmonary contusion, rib fractures, flail segments, sternal fracture or blunt aortic injury.

Study outcomes

The primary study outcomes are the incidence of BCI, length of ICU stay and mortality in patients presenting with blunt thoracic trauma.

Definitions

The diagnosis of BCI was made in all patients with a serum TnT or TnI above the 99th centile for the diagnosis of acute myocardial infarction, measured at or within 12 h of presentation following blunt thoracic trauma and who fulfilled any one of the following criteria:

1. Clinical findings of pericardial rupture, cardiac herniation or central tendon rupture.
2. Hypotension requiring inotropic support not explained by haemorrhagic, septic or neurogenic shock.
3. ECG abnormalities in keeping with blunt cardiac injury (i.e., atrial or ventricular arrhythmias, ST or T wave abnormalities or conduction abnormalities).
4. TTE findings in keeping with blunt cardiac injury.

A flail chest was defined as two or more ribs broken in two or more places or sternal dissociation with costochondral dysjunction. Pulmonary contusions were defined as typical parenchymal changes noted on X-ray or CT scan with the presence of hypoxaemia.

Statistical analysis

Means and standard deviations are reported for normally distributed data; median and inter-quartile range for data not normally distributed. The $\chi 2$ or Fisher's exact test were used for categorical data, and Student's *t*-test and Mann–Whitney *U* test for continuous data where appropriate. A p value of <0.05 was considered significant.

Results

Two hundred and thirty nine patients were identified as having sustained blunt thoracic trauma over the 3 year study period. Seventy patients were excluded from the analysis due to serum troponin measurement being not performed, or performed outside of the 12 h time limit. Of the remaining 169 patients, the majority were male (73%) with a median age of 34 years (interquartile range [IQR] 25–43). The median ISS was 34 (IQR 25–43). Admission serum lactate measurements were significantly higher in the BCI cohort (3.55 [IQR 2.4–6.2] vs. 2.4 [1.4–4.3]; $p = 0.008$). Eight patients with BCI were under the age of 18, of whom six were under the age of 10 (Table 1).

Eighty four patients were diagnosed with BCI, of whom two were diagnosed on clinical grounds. The incidence of BCI in the entire cohort was 50%. Patients diagnosed with BCI were of similar age (median 34 [25–42] vs. 35 (25–41); $p = 0.514$) but had a higher ISS (37 [29–47] vs. 31 [21–38]; $p = 0.001$) than those without. Two patients had serum TnT measurements performed, with the remainder diagnosed from serum TnI measurements. The median serum TnI level was 2823 ng/L (IQR 1353–6833), with the highest measured at 64950 ng/L. 59 patients with BCI presented with hypotension requiring inotropic support. The average time spent on inotropes was 3.1 days.

ECG findings were documented in only 29 patients in whom BCI had initially been confirmed by troponin estimation. The abnormalities ranged from bradycardia, ventricular and supra-ventricular tachycardia, non-specific ST segment elevation or depression, T wave inversion and one patient developed a third degree heart block requiring pacing. The dysrhythmias were treated according to standard Advanced Cardiovascular Life Support (ACLS) algorithms either pharmacologically or electrically.

TTEs were performed on 38 (45%) patients with BCI, of whom 30 (79%) had abnormal findings. Twenty-two (73%) patients with abnormal TTEs presented with hypotension. Some patients had more than one abnormality documented. Despite 6 patients being diagnosed with posterior or anterior mitral leaflet chordal rupture, none had clinically significant mitral regurgitation. In 10 (26%) patients there was one or more regional wall motion abnormality (Table 2). The majority of echocardiograms were performed within the first 3 days of admission to the TICU.

Table 1
Demographics of patients admitted to the TICU at IALCH with blunt thoracic trauma January 2010–April 2013.

	All thoracic trauma	No BCI	BCI	*p*-Value
n (%)	169	85 (50)	84 (50)	
Age median (IQR)	34 (25–41)	35 (25–41)	34 (25–42)	0.514
Male *n* (%)	123 (73)	61(72)	62 (74)	0.900
Injury severity score (median, IQR)	34 (25–43)	31 (21–38)	37 (29–47)	0.001
Admission lactate (median, IQR)	2.85 (1.7–5.1)	2.4 (1.4–4.3)	3.55 (2.4–6.2)	0.008
Referral from scene *n* (%)	44 (26)	20 (24)	22 (26)	0.824

TICU, trauma intensive care unit; IALCH, Inkosi Albert Luthuli Central Hospital; BCI, Blunt Cardiac Injury; SD, standard deviation; ISS, injury severity score; IQR, interquartile range.

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Table 2
Echocardiogram findings ($n = 38$)[a].

Regional wall motion abnormality *n* (%)	10 (26)
All measurements and observations within normal ranges *n* (%)	8 (21)
Paradoxical septal wall motion *n* (%)	8 (21)
Dilated RA/RV *n* (%)	7 (18)
Chordal rupture to AML/PML *n* (%)	6 (16)
Pericardial effusion *n* (%)	2 (5)
Aortic root dissection *n* (%)	1 (3)
Ventricular septal defect *n* (%)	1 (3)
Pneumopericardium *n* (%)	1 (3)

RA, right atrium; RV, right ventricle; AML, anterior mitral leaflet; PML, posterior mitral leaflet.
[a] Findings add up to more than 38 due to more than one finding per patient.

Motor vehicle collisions (MVCs) accounted for 92% of injury mechanism and the majority of patients were pedestrians (40%). Very few patients were injured during motorcycle collisions (4%), with the remainder of injuries sustained either as MVC drivers, passengers or from other mechanisms (e.g. falls, structural collapse or assault). There was no significant difference in the causes of injury between the two BCI and non-BCI groups ($p = 0.21$).

Only 8 (5%) patients sustained isolated blunt thoracic trauma with multiple system injuries diagnosed in the remaining 161 (95%). Eighty-two percent of the cohort sustained pulmonary contusions. The incidence of this injury was highest in the BCI group (96% vs. 66%; $p < 0.001$). There were very few sternal fractures diagnosed (5%) with no significant difference between the two groups. Rib fractures, flail segments and blunt aortic injuries were also similar across both groups (Table 3).

The primary outcomes of the study were length of stay and mortality. There was a tendency towards a longer length of stay in patients diagnosed with BCI (17.0 days [SD 13.5] vs. 13.6 days [SD12]; $p = 0.084$). The overall mortality was 24%. There was a significantly higher mortality in the BCI cohort (32% vs. 16%; $p = 0.028$) (Table 4). None of the patients described required acute surgical intervention during their admission in the TICU but those patients with valvular, septal or chordal injuries were referred for follow up with the cardiothoracic service.

Two patients were diagnosed with BCI clinically. One was a 25 year old female who presented in asystole and underwent emergency laparotomy for haemorrhage control and was found to have a diaphragmatic central tendon rupture with cardiac displacement into the abdomen. She subsequently demised eight days later with severe hypoxic brain injury. The other patient was a 66 year old male that presented following a MVC as a driver. His computed tomography (CT) images revealed an acute tear of the diaphragmatic central tendon with colonic herniation into the pericardium and displacement of the heart. This hernia was subsequently repaired via laparotomy on day 4 following physiological stabilisation. He demised 42 days after admission secondary to a massive pulmonary embolism.

No patients required acute cardiothoracic intervention specifically for BCI. Patients that sustained valvular or chordal injury were referred to for further cardiothoracic follow up. No long term outcome data are available for those patients referred to the cardiothoracic surgery service.

Table 3
Spectrum of injuries sustained from blunt thoracic trauma.

	All thoracic trauma $n = 169$	No BCI $n = 85$	BCI $n = 84$	*p*-Value
Pulmonary contusion *n* (%)	138 (82)	56 (66)	82 (96)	<0.001
Rib fractures *n* (%)	72 (43)	35 (41)	37 (44)	0.824
Sternal fracture *n* (%)	9 (5)	2 (2)	7 (8)	0.165
Blunt aortic injury *n* (%)	8 (5)	3 (6)	5 (6)	0.704
Flail segment *n* (%)	41 (24)	17 (20)	24 (29)	0.263

BCI, blunt cardiac injury.

Table 4
Primary outcomes of patients with blunt thoracic trauma admitted to TICU in IALCH from 2010 to 2013.

	All trauma $n = 169$	No BCI $n = 85$	BCI $n = 84$	*p*-Value
Length of stay mean (SD)	15.3 (12.9)	13.6 (12.0)	17.0 (13.5)	0.084
Mortality *n* (%)	41 (24)	14 (16)	27 (32)	0.028

BCI, blunt cardiac injury; SD, standard deviation.

Discussion

The definition of BCI has been problematic. Previously the terms "myocardial contusion" and "myocardial concussion" were used, however these are often inappropriate for the pathology and conditions being described [1,5]. There is concern that the diagnosis of "myocardial contusion" may lead to unnecessary monitoring and waste of resources. Therefore, the recommendation has been made that these terms no longer be used for its diagnosis and that documentation rather describe the specific pathology which is present [1,2,5].

Blunt thoracic trauma injury can produce a variety of cardiac sequelae, ranging from minor and clinically insignificant bruising, coronary artery injury or even cardiac rupture [1,6–8] and is responsible for up to 25% of trauma-associated mortalities [9–11]. As exhibited by our own data, the commonest mechanism of injury is a motor vehicle collision. The force required to produce a myocardial lesion being as little as 32 km/h [12]. At the moment of impact there is sudden increased pressure within the thoracic cavity which may damage the myocardium through shearing stress forces. The heart may also be forced and compressed between the sternum and vertebrae [10], with the right ventricle being the most susceptible to injury due to its location just beneath the sternum. Our study shows that high energy transfer injuries, such as pulmonary contusions, should alert the clinician to the possibility of a BCI.

While the more serious injuries will often be obvious to the clinician on presentation, some of the conditions may be particularly difficult to diagnose [1,8,9] and therefore appropriate management may be delayed. A definitive diagnosis of myocardial damage can really only be confirmed histologically at post-mortem by the presence of necrotic myocardial cells [10], therefore in order to assess and diagnose myocardial damage, clinicians have to make use of alternative methods, some of which are unreliable. These include history and physical examination, plain chest radiograph, electrocardiography, measurement of cardiac enzymes, echocardiography and nuclear studies [2,10].

Obtaining an accurate and detailed history in the trauma setting may be particularly difficult. Likewise, thorough clinical examination may be challenging especially in the presence of more life-threatening injuries. Precordial pain is the most common complaint in patients having sustained a myocardial injury; however, this may easily be mistaken as stemming from associated thoracic injuries [10]. Sternal fracture may be an important finding but the presence of a sternal fracture was not an association with blunt cardiac injury in our study. It is worth noting that although the overall incidence of sternal fractures was low, 7 out of the 9 (78%) patients with sternal fractures were diagnosed with BCI. No patients sustained sternal fractures in isolated. Other studies have also failed to demonstrate association with isolated sternal fractures and BCI [13–15].

Injury to any skeletal muscle, kidneys, brain or liver causes release of creatine phosphokinase (CPK). Creatine phosphokinase

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myocardial band (CPK-MB) will peak in the first 24 h following skeletal muscle or myocardial cellular injury and returns to normal levels within 72 h, rendering it a useful marker of cellular damage [16]. A CPK-MB fraction greater than 5% of the total CPK level is considered abnormal. However, in the trauma population with multiple injuries, the total CPK may well be elevated thereby decreasing the sensitivity of measuring the CPK-MB fraction. For these reasons CPK-MB is not particularly useful in the assessment of myocardial injuries following trauma [10,17].

Cardiac troponins are only released when there has been disruption of myocardial cell membrane; therefore they are significantly more specific markers of myocardial damage [18]. A number of studies have looked at the role of screening TnI or TnT in the diagnosis of BCI [4,19–23]. The current recommendations from the Eastern Association for the Surgery of Trauma practice management guidelines include routine serum TnI surveillance on all patients that are suspected to have sustained BCI, in combination with a screening ECG [2]. Some authors have described absolute levels of serum TnI in the diagnosis of BCI [17,20], but there is no consensus for the absolute cut-off of serum TnI for the diagnosis. It has been shown in a number of studies that patients with troponin elevation not related to ischaemic cardiac disease have a worse prognosis [24–26]. Due to the numerous types of assays available for serum TnI with varying reference ranges, it is practical to take a value above the 99th centile for the diagnosis of acute myocardial dysfunction as significant in blunt thoracic trauma, as we did in this study.

Between 40% and 80% of patients sustaining myocardial injuries will exhibit ECG abnormalities, although these are neither consistent nor predictable [1,10]. This was reflected in the findings of our study. Left ventricular injuries may present with ST segment abnormalities, but changes in vagal tone, increased release of catecholamines, pre-injury cardiac abnormalities, pulmonary contusions and hypovolaemia may produce similar ischaemic changes and should be excluded. Other entities which can lead to abnormal ECG changes include pain, anxiety, haemorrhage, hypoxia, head trauma and drug or alcohol intoxication [10].

TTE may be difficult to perform and interpret in trauma patients due to pain and other injuries, which may have caused subcutaneous emphysema or necessitated the placement of intercostal drains. It also is non-contributory in the assessment of arrhythmias which are the most common manifestation of significant BCI. That said, valuable information may be obtained regarding wall motion abnormalities, changes in the size of the atria or ventricles, valve structure and function as well as the presence of thrombus, shunting or cardiac tamponade [10,27]. In our study only half of the patients diagnosed with BCI underwent TTE examination but 79% had significant abnormalities some of which necessitated further cardiothoracic review, including an aortic root dissection and ventricular-septal defect. This indicates that TTE is a valuable investigation in BCI although echocardiographic facilities are not easily accessible for the majority of centres in developing countries.

Numerous studies have looked at the incidence of myocardial contusion injury using different methods to make the diagnosis [2,3,16,28]. However, even when BCI is confirmed by regional wall motion abnormalities or increased cardiac enzymes, these patients may not always manifest significant clinical signs and symptoms, and they do not all require intervention [5,28]. Therefore while some of the above tests may be able to determine the presence of a cardiac injury following blunt chest trauma, individually they are poor predictors of whether or not the lesion is likely to be clinically significant. However, the combination of two or more of these investigations, as used in the critically ill trauma patient, significantly increases their diagnostic and prognostic value.

The incidence of BCI in this study is 50%. Other studies describe ranges of between 7% and 56%, depending on the method of diagnosis [11,19,28]. Unsurprisingly, patients diagnosed with BCI tended to be more severely injured with a higher ISS and initial serum lactate. There were a surprising number of children in the BCI cohort. Children that have sustained BCI are poorly described due to their small numbers and require more investigation [21,29].

Limitations

The definition applied to BCI in this study is not universally accepted. However, the combination of an elevated serum TnI or TnT measurement in the presence of: (i) clinical signs of pericardial rupture, cardiac herniation or central tendon rupture, (ii) clinically significant hypotension suggesting cardiac dysfunction, or (iii) ECG changes, or (iv) structural injuries to the heart on TTE; provides good evidence for BCI, rather than using an isolated serum TnI or TnT measurement.

This was a retrospective study in a single centre over 3 years. Practise changed over the course of time in the TICU to include TnI measurement routinely when a patient presents with significant blunt chest trauma. Only 71% of patients identified with blunt thoracic trauma had a screening TnI or TnT performed on admission. As there may already have been clinical suspicion of BCI in the patients in whom early serum troponins were measured, the actual incidence of BCI may be lower than that reported.

There was significant heterogeneity in the assays used for serum troponin measurement, due to changes over time in the TICU. Instead of taking an absolute level as significant, only patients presenting with serum troponin levels above the 99th centile for diagnosis of an acute myocardial infarction were considered significant. We consistently used this level in our study as a cut off as any measurement about this threshold indicates a significant troponin leak, irrespective of the absolute value and would be therefore be clinically relevant.

Although an ECG is often the initial investigation in the diagnosis of BCI, there were only 29 ECG findings that could be found in the notes. This does not mean that they were not performed, but rather that the relevant findings were not documented, or that the findings were non-specific in the context of the severely injured polytrauma patient.

Only 45% of all patients diagnosed with BCI underwent imaging by TTE. This may have led to under-diagnosis of structural or regional wall motion abnormalities. TTEs were performed by a variety of echocardiographers and hence there may be significant variability in interpretation of the TTE findings.

The exclusion of patients with serum troponin elevation measured after 12 h from admission was aimed at eliminating as many reasons as possible, other than direct cardiac muscle damage, for troponin elevation. The time window of 12 h was chosen as to reduce the time for accumulation of serum troponins in those patients with concomitant acute kidney injury. By measuring only the early serum troponin level, it reduces the problem of detecting elevated troponins secondary to reduced renal clearance.

Lastly, patients presenting to the TICU may have had previous undiagnosed underlying cardiac disease and developed an elevated troponin for reasons other than direct cardiac injury. However, the average age of the patients was 34, so the overall likelihood of previous cardiovascular disease is low.

Conclusion

BCI is associated with an increased mortality in our study. Patients also tended towards a longer length of stay. BCI is a clinically relevant diagnosis which requires a high index of

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suspicion, even in the paediatric blunt thoracic trauma patient. Screening of high risk patients with significant blunt thoracic trauma with a TnI or TnT should be routine practise. Patients diagnosed with BCI should undergo more advanced imaging such as TTE or TOE to exclude significant cardiac structural injury.

Conflict of interest

All authors declare that there is no conflict of interest noted for the submitted paper.

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