The Outcome of Cardiac Dysfunction in Critically Ill Trauma Patients: Myocardial Contusion Complicated by Refractory Hypotension

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Abstract

Background: This work attempted to define the care and course of those most severely affected patients in the setting of blunt chest trauma, who had hypotension refractory to routine fluid resuscitation.

Methods: Twenty-three critically ill blunt trauma ICU patients were resuscitated and enrolled with ongoing hypotension required placement of a pulmonary artery catheter. The REF®Explorer (Baxter, Edwards, Anaheim, CA) catheter was placed in the right heart measuring pressure, volume and oxygen utilization information, as well as recording Injury Severity Score, EKG, CXR, CPK/MB and echocardiography over the initial 72-h time period.

Results: There were an approximately 2,300 Level I trauma patients admitted annually over a 4-year period with an overall mortality rate of 4.3% (100) patients with 3.4% (79) patients ''ruling in'' with elevated cardiac enzymes, associated with an increased mortality rate of 6.7% ($p < 0.05$). The 23 patients were male (17, 74%), mean age 41.2 years, with no past medical history (19, 83%), in a motor vehicle accident (21, 91%), with pulmonary injury (9, 39%), undergoing celiotomy in (10, 44%). They presented with moderate to severe trauma acuity defined as mean GCS of 8.6, TS of 11.3, and ISS of 34 with an increased mean hospital stay of 15 days versus 6 days in the ICU; and a 26 days versus 10 days overall stay for those with myocardial contusion ($p < 0.05$). Analysis of diagnostic variables found an abnormal EKG in (21, 91%), CXR in (20, 87%) and echocardiogram in (8, 37%). The total CPK was found to be elevated, mean 2,219 (204–8,278 U/l), while the MB fraction was normal 2.3 ± 1.3%. Invasive cardiac monitoring found an increase in CO of 1.6 l/min from 5.9 to 7.8 l/min during the first 24 h of recovery. Survival was worsened with increased ISS (29 vs. 43) $p < 0.02$, but improved with longer ICU (17 vs. 8) $p < 0.03$ and hospital (39 vs. 7) $p < 0.05$ stay in days. The analysis of commonly used diagnostic modalities – EKG, CXR, ECHO, or CO, did not correlate with survival, but the total CPK was increased in survivors (2,715 vs. 1,432 U/l) $p < 0.009$.

Conclusion: There is worsened morbidity with a 2-fold increase in ICU LOS and hospital stay, and a 1.5-fold increase in mortality in the severe myocardial contusion group. The diagnostic dilemma posed by lack of definitive testing continues unresolved after analysis of routine modalities – EKG, CXR, ECHO, CPK or CO – failing to yield a ''best test''.

Key Words

Blunt chest trauma · Chest trauma · Trauma · Myocardial contusion · Cardiac contusion · EKG · CXR · Echocardiogram · CPK · Cardiac output

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Introduction

Early analysis of myocardial contusion by Kahn in 1929 suggested that prognosis in thoracic contusions depends on the character of the injury – direct due to contusion or concussion of the thorax; or penetrating and indirect due to fall from height or ''heart strain'' [1]. The outlook was found to be grave in those cases associated with marked shock.

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Claude Beck reported his experience with a group of 175 patients presenting with nonpenetrating wounds of the heart describing three clinical syndromes [2]. He found that 87% died from rupture, 6% succumbed to myocardial failure, while 7% recovered. This series vastly underestimated survivorship with over 93% recovery, when all cases were considered. He also described a classic syndrome involving tachycardia, electrocardiographic changes – Q wave and T wave abnormalities, auscultatory findings and hemorrhagic effusion.

Arenberg published a more representative series of 241 cases of chest wall contusion with an incidence of myocardial injury of 7% [3]. They found that the patients older than 50 years seemed to be at greatest risk based on preexisting structural heart disease.

Approximately 20% of patients sustaining blunt chest trauma have some degree of cardiac damage, including cardiac rupture, valvular, coronary artery, pericardial, aortic and great vessel injury [4]. The range of involvement is even more variable with 10–75% presenting with myocardial contusion on admission [5]. However, a range this extensive raises questions of diagnostic accuracy and reliability.

Sternal fractures indicated roentgenographically by mediastinal widening are commonly associated with aortic injuries, and biventricular depression documented by first-pass radionuclide angiography [6]. However, the incidence of displaced or unstable fracture is very low. Buckman's retrospective review of 60 patients admitted with sternal fracture found chest pain in 59 (99%) or almost all presenting patients with an abnormal chest radiograph as well in 100 and 62% with abnormal EKG [7]. Those who presented with a normal EKG in 20 cases (48%) subsequently developed significant changes in 3 (15%) of 20 cases. It is important to note that elevated creatine phosphokinase (CPK) levels were only found in 5% (3 of 60) patients, while 18% (11 of 60) had abnormal echocardiograms. Therefore, routine diagnostic modalities for myocardial contusion are sensitive, but not specific for disease clarification.

We attempted in this evaluation to better define diagnostic parameters and predictors of survival in those patients with severe blunt chest and presumed myocardial contusion complicated by refractory hypotension.

Materials and Methods

Protocol

This study was approved by the University of Pittsburgh Investigational Review Board. The human subjects included 23 critically ill blunt chest trauma patients admitted to the trauma intensive care unit in a convenience sample all to receive a protocol of standardized ICU care attempting to maintain adequate, perfusion, ph and tonicity from a group of 2,305 average trauma admissions annually over a 4-year period. Patients were monitored for up to a 96-h period, as warranted by patient condition.

Patients were enrolled, who presented with (1) Refractory hypotension defined a systolic BP < 90 mmHg after 2 l of crystalloid resuscitation fluid; and (2) conventional myocardial contusion criteria including (a) EKG abnormalities; (b) cardiac enzyme elevations (total CPK ≥ 278 mg/dl and/or CPKMB $\geq 5.0\%$); or (c) echocardiographic evidence of cardiac wall motion abnormalities) [5].

Emergency department resuscitation: patients were resuscitated and stabilized according to standard Advanced Trauma Life Support guidelines. Adequate oxygenation was ensured while intubation and mechanical ventilation were instituted for altered level of consciousness, hypoxemia manifested as $PaO_2 < 60$ mmHg or oxygen saturation $< 90\%$ or hemodynamic instability. Circulatory support was instituted with large bore intravenous access – femoral 8.5 FR introducer and hemorrhage control was provided – including operative intervention if necessary. Fluid administration consisted of crystalloid-Lactated Ringer's solution to a total of 2000 cc followed by blood administration, as necessary to achieve a SBP ≥ 90 mmHg, followed by transport to the operating room or ICU.

Intervention

Patient was admitted to the trauma intensive care unit to reevaluate cardiopulmonary stability – ensuring adequate oxygenation ($pO_2 > 60$ mmHg) ventilation ($pCO_2 < 40$ mmHg). Patients were administered high flow oxygen at 12–15 LPM to ensure saturations were $> 90\%$. If unable to maintain this saturation then mechanical ventilation was begun with FiO_2 0.5–1.0 and Positive End Expiratory Pressure (PEEP) of 5 cm H_2O. Volume control ventilation in an assist control mode was provided at 10 ml/kg to maintain peak pressure < 40 cm H_2O.

Adequate systemic perfusion was maintained by administration of additional crystalloid in 10 ml/kg boluses. If systolic blood pressure was inadequate then a 8.5 FR introducer and REF® Explorer pulmonary arterial catheter (Baxter-Edwards Critical Care Division, Anaheim, California) were inserted using an internal jugular or subclavian vein approach. The oximetric device was calibrated in an *in vivo* or *in vitro* fashion dependent on patient condition.

Table 1. Presentation of blunt chest trauma and myocardial contusion.

Year	Total trauma admissions	Mortality	Total chest trauma admissions	Myocardial contusion	Mortality	AVG ICU LOS (days)	AVG LOS (days)	Myocardial contusion rule out	AVG LOS rule out
1	1,827	71 (3.9%)	84 (4.6%)	49 (58.3%)	8 (16.3%)	11.0	14.8	35 (41.7%)	6.1
2	2,205	118 (5.4%)	102 (8.7%)	4 (52.8%)	1 (3.9%)	4.6	4.4	91 (47.1%)	4.1
3	2,515	103 (4.1%)	114 (8.4%)	5 (54.3%)	7 (4.3%)	9.4	13.2	96 (45.7%)	4.3
4	2,673	108 (4.0%)	50 (3.4%)	4 (54.9%)	6 (8.0%)	1.3	10.4	41 (45.0%)	3.7
Total	9,220	400 (4.3%)	350 (3.8%)	62 (0.7%)	21 (0.2%)	26.3	42.8	263	18.2
Average	2,305	100 (4.3%)	144 (6.3%)	79 (3.4%)	5 (0.3%)	6.6	10.7	65 (2.8%)	4.5 (0.19%)
Myocardial contusion			57%	54.9%	6.3%	13.4	26.5	45.8%	9.2%

Fisher's exact; chi square with Pearson correlation ($p < 0.05$)

Intensive Care

Additional crystalloid was administered to ensure adequate perfusion. Dopamine was used as a vasoconstrictor agent at a dose of 2.5–20 μg/kg/min if refractory hypotension existed, dobutamine at a 5–20 μg/kg/min dose for inotropic support, and norepinephrine or epinephrine at a 0.1–1.0 μg/kg/min dose was used less commonly for refractory cardiovascular failure. Mechanical support including extracorporeal membrane oxygenation (ECMO) and intraaortic balloon pump (IABP) was provided for refractory hypotension, < 90 mmHg SBP or MAP < 60 mmHg. Fentanyl at 1–2 μg/kg/h was used for analgesia and midazolam at 0.01–0.05 mg/kg/h for sedation and ventilation, while vecuronium 0.01 mg/kg/h for muscle relaxation to assist in patient control for ventilation.

Temperature control was targeted to maintain patients in a 36–38°C range. Acid base status was also maintained with base deficit correction to ≤ –5 by the administration of sodium bicarbonate 0.1 mEq/kg. Calcium chloride (0.01 mg/kg) was also used to correct hemodynamically significant hypocalcemia, measured as an ionized Ca of < 0.90 mEq/l.

Assessment

Demographic variables were assessed including age, sex, past medical history, history of present illness, diagnosis and surgical intervention. Diagnostic testing used to identify myocardial contusion included electrocardiogram (EKG), chest radiography (CXR), creatine phosphokinase (CPK) with myocardial fraction-muscle-brain (MB), and transthoracic (TTE) or transesophageal echocardiogram (TEE).

Outcome variables measured were survival, length of ICU stay (LOS ICU), length of hospital stay (LOS HOSP), Glasgow Coma Score (GCS 3–15), Trauma Score (TS 4–16), and Injury Severity Score (ISS 0–75) were measured on admission and daily thereafter.

Cardiopulmonary variables were obtained in a static daily fashion for 72 h and in a dynamic fashion after a 500 cc fluid challenge. The information obtained included hemodynamic variables or work indices such as cardiac output (CO), ejection fraction (EF), or stroke volume (SV), as well as cardiac length indices such as central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), end diastolic volume (EDV) and end systolic diastolic volume (ESV) (Table 1). Additional respiratory variables measured included oxygen delivery (DO_2), oxygen consumption (VO_2), as well as arterial and venous oxygen saturation and content. Data was then compared for survivors and non-survivors for trends.

Data Analysis

Demographic data was reported as frequency and proportions of survivors compared to non-survivors were analyzed using Fisher's exact test where appropriate for differences. The descriptive variables were reported as mean, standard deviation and range as well as objective numerical data where continuous variables were analyzed using Student's *t* test or Mann-Whitney *U*-Wilcoxon Rank Sum test for parametric and non-parametric data, respectively. The cardiopulmonary data was analyzed with regression and analysis of variance (ANOVA) comparisons where significance was indicated by conventional criteria ($p < 0.05$).

Results

There were an average of 2,305 Level I Trauma admissions annually over a 4-year period with an overall mortality of 4.3% (100) patients (Table 1). Blunt chest trauma was the admission diagnosis in 6.3% (144) of patients with slightly over half 55% (79) or 3.4% of total patients ''ruling in'' by conventional criteria for myocardial contusion.

Table 2. Assessment and prognostic data.

Patients (23)	Mean ± SD (range)	Survivors (9)	Non-survivors (14)	Significance
Age (years)	41.2 ± 19.0 (15–77)	39.8 ± 19.2	43.8 ± 18.4	NS
GCS	8.6 ± 5.8 (3–15)	8.1 ± 5.9	9.6 ± 5.7	NS
TS	11.3 ± 3.9 (5–16)	11.4 ± 3.9	11.3 ± 4.2	NS
ISS	34.6 ± 12.7 (17–75)	29.4 ± 6.6	42.7 ± 15.9	$p < 0.023$[a] $p < 0.019$[b]
ICU LOS (days)	13.4 ± 10.7 (1–40)	17.2 ± 10.1	7.6 ± 9.1	$p < 0.031$
Hosp LOS (days)	26.5 ± 38.1 (1–188)	38.7 ± 44.7	7.5 ± 9.1	$p < 0.053$

[a]Student's *t* test, Levine test for equality of variance ($p < 0.05$)
[b]Mann-Whitney *U*–Wilcoxon Rank Sum *W* test ($p < 0.05$)

Those with myocardial contusion had an average length of ICU stay of 13.4 days and hospital stay of 26.5 days compared to 6.6 and 10.7 days overall for trauma patients ($p < 0.05$). The mortality in those with myocardial contusion was increased slightly (6.7 vs. 4.3%; $p < 0.05$) compared to general trauma mortality. The remainder (4.5%) ruled out for myocardial contusion and included 66 patients or 2.8% of total trauma population. Their average length of hospital stay was decreased (4.5 days) compared to those with myocardial contusion (26.5 days, $p < 0.05$), as well as the overall trauma population (10.7 days, $p < 0.05$).

The patient demographic profile finds a threefold predominance of males (17, 74%) compared to females (6, 26%). The mean age was 41.2 years with a range of 15–77 years. Patients were healthy with no past medical history in (19, 83%), followed by a cardiac history in (2, 8.7%). The mechanism of injury was predominately motor vehicle accident (MVA) in (21, 91%). Those who were injured were found to have a pulmonary diagnosis in (9, 39%), orthopedic injury in (5, 22%) and other cardiovascular diagnosis in (4, 17%) of cases. Likewise, most patients underwent some surgical intervention – celiotomy (10, 44%), or open reduction and internal fixation in (5, 22%).

Patient profile defined by outcome assessment variables found a mean Glasgow Coma Score (GCS) of 8.6 (3–15), Trauma Score (TS) of 11.3 (6–16) and Injury Severity Score (ISS) of 34.6 (17–75) (Table 2). The mean length of ICU stay was 13 days (1–40) and hospital stay of 26 days (1–188) with mortality of 39% in the 23 most severely affected cases with 9 survivors and 14 non-survivors.

The noninvasive diagnostic variables analyzed demonstrated an abnormal EKG in (21, 91%) of cases, presenting mainly with sinus tachycardia or T wave/ST changes (Table 3). The chest radiography (CXR) was abnormal in (20, 87%) of cases usually indicating a pulmonary contusion or pneumothorax (Table 3). Echocardiogram was abnormal in (8, 37%) of cases where both the LV and RV exhibited dysfunction (Table 4).

Table 3. Diagnostic variables.

	Abnormal	Normal	Survival (p)
EKG	91.3% (21)	8.7% (2)	NS
CXR	86.9% (21)	13.1%	NS
Echo	37.5% (6)	62.5% (16)	NS

Chi square with Pearson correlation ($p < 0.05$)

Analysis of total CPK finds a mean value on presentation of 2,218 with a range of 204–8728 U/l (Table 4). There was little specificity offered by examining for abnormal total CPK (> 210 U/l) with all (100%) cases fulfilling this criteria. However, the CPK myocardial specific fraction was 2.3% with range of 0.8–5.9% with only 8.7% of cases positive. Lastly, invasive hemodynamic monitoring was compared with an initial cardiac output of 5.9 l/min was initially followed by increase to 7.8 l/min, which with mean change of 1.6 l/min (Table 4).

Demographic characteristics including scoring systems and testing variables were analyzed as predictors of survivorship. There was no significant decrease in survival based on risk or other demographic variables. Comparison of various scoring systems found no appreciable survival predictive ability using GCS or TS in the myocardial contusion population, while a decreased ISS (29 vs. 43) was associated with improved outcome ($p = 0.023$).

There was an association between survival and an increased ICU (17 vs. 8 days, $p = 0.031$) and hospital stay (39 vs. 7 days, $p = 0.023$) where longer stays where

Table 4. Diagnostic variables – cardiac enzymes – invasive.

	Mean/SD/range	Survival	Non-survival	Significance
CPK				
Total (iu)	2217.9 ± 2197.1 (204–8278)	2714.6 ± 2567.6	1432.5 ± 1198.6	$p < 0.009$
MB %	2.34 ± 1.3 (0.8–5.9)	2.3 ± 1.41	2.5 ± 1.4	NS
Variables – invasive Hemodynamic monitoring				
Co (l/min)				
0 h	5.9 ± 2.2 (2.3–9.2)	5.9 ± 1.9	6.2 ± 2.2	NS
	11	9	2	
24 h	7.8 ± 2.9 (2.5–13.5)	7.9 ± 3.4	8.3 ± 2.8	NS
	16	9	8	
CO_{24}–CO_o	1.6 ± 3.8 (0.43–8.6)	2.1 ± 4.7	1.8 ± 1.4	NS

Student's *t* test; Fisher's exact; Mann-Whitney *U*–Wilcoxon Rank Sum *W* test

found in survivors. The analysis of diagnostic techniques found no prognostication of survival using EKG CXR or echocardiogram analysis (Tables 4, 5). Although, there was no difference in MB fraction, the total CPK was increased in the group that survived (2,715 vs. 1,432 IU/l, $p < 0.009$). Lastly, assessment of cardiac output found no change in absolute measurements at either the initial (0 h) or final (24 h) time points during the analysis.

Discussion

Our study sample consisted of over 2,300 annual trauma admissions. The 23 patients evaluated were included due to the presence of refractory hemodynamic instability and only accounted for 0.1% of the total population. While, overall those admitted with blunt chest trauma accounted for only 3.4% of patients with over 50% ''ruling-in'' for myocardial contusion.

Although not comprising a significant proportion of the trauma patients generally, myocardial contusion assumes significance due to its 1.5-fold increase (9 vs. 14) in mortality in this trauma population. Therefore, an effective diagnosis and treatment strategy can potentially impact the survivability of significant myocardial contusion, as well as decreasing morbidity with a twofold increase in length of ICU (13 vs. 6 days) and 2.5-fold increase in length of hospital stay (26 vs. 10 days) in those with myocardial contusion compared to the general population.

The demographic analysis was not helpful as a survival discriminator based on small sample size. However, the myocardial contusion patient profile – healthy, middle aged adult males involved in motor vehicle accidents may be useful as a general descriptor of those at risk.

Likewise, resource allocation may be improved by defining necessary interventions in those with antecedent pulmonary (9, 39%) and cardiovascular (4, 17%) disease, which may affect the perioperative outcome of those who undergo celiotomy (10, 44%) or orthopedic intervention (5, 22%). This awareness may require operative delay it warranted or careful hemodynamic monitoring by both invasive and noninvasive routes to protect those at risk for further cardiopulmonary compromise.

Qualitative scoring systems predict a moderate to severe injury with mean GCS 9, TS 11, and ISS 34. Although neither triage score – GCS or TS – measured prospectively correlated with outcome, the ISS measured on admission correlated inversely appropriately with survival. However, this linear relationship cannot absolutely predict outcome and limits of a small representative sample fail to delineate a discriminator between survivors and non-survivors limiting clinical utility.

Likewise, very little definitive information can be inferred from the current diagnostic armatarium currently utilized for myocardial contusion. Clearly, the admission EKG (91%) and CXR (81%) were sensitive, but not specific markers for this disease condition, while the echocardiogram offered slightly better specificity (63%) but poor sensitivity.

The ''best'' conventional testing battery would probably be a combination of total CPK offering 100% sensitivity and the CPK/MB fraction offering 91.3% specificity [8, 9]. This acknowledges the cross-reactivity between cardiac and other body regions, such as brain resulting in decreased specificity in head injury cases. However, most recent data offered by Biffl suggest no correlation of CPK measurement to the diagnostic accuracy in myocardial contusion with a 30% myocardial contusion rate and complications in 5% [10]. However,

this and other similar methodology is currently followed since there is no ''gold standard'' diagnostic resulting in incorporation bias. Even in this group of the most severely affected blunt chest trauma patients only 37.5% have an abnormal echocardiogram. Thus, little of the routinely employed diagnostic testing can be recommended as being confirmatory of myocardial contusion, causing it to remain largely a clinical diagnosis.

However, some prognostic information was noted with a significant increase in total CPK activity in survivors. There is little clinical relevance as this finding appears to be a function of the rapid demise of those severely injured who succumb prior to enzymatic degradation product release.

Lastly, even invasive cardiopulmonary monitoring failed to diagnose myocardial contusion with most patients with a normal CO 5.9 l/min at 0 h and > 7.8 l/min at 24 h. Analysis of the ΔCO 24–0 was limited by small sample size. Therefore, further study will evaluate other cardiopulmonary variables more carefully examining markers of cardiac performance and oxygen delivery.

The contention that in fact patients did not have significant myocardial injuries is probably not the correct conclusion. It is important to note that in a relative robust trauma program, with 2,300 patients admitted only a small number actually met criteria for myocardial contusion. So again, these were patients that were deemed to be critically ill by comprehensive trauma and critical care providers as manifested by hypotension refractory to emergency department resuscitation yet did not have cardiac contusion as the driving force behind this dysfunction.

Conclusion

This study was a preliminary evaluation that described the demographic, diagnostic and outcome profile of patients with blunt chest trauma and presumed myocardial contusion, accompanied by severe hemodynamic instability. The routine diagnostic modalities including EKG, CXR and CPK are perhaps overly sensitive; while CPK/MB and echocardiograms are too specific, acknowledging that a true ''gold standard'' for myocardial contusion is lacking.

Some prognostic information was offered by the inverse correlation of ISS and survival, while no further delineation in outcome was offered by invasive cardiopulmonary monitoring and CO assessment. The proper ''diagnosis'' and clinical management of significant myocardial contusion has remained elusive requiring good medical judgment, factoring often conflicting test results, to generate an effective and efficient plan of care.

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