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Research Article

Investigation of Sensitivity and Specificity of Biochemical Markers for Cardiac Contusion due to Blunt Chest Trauma

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ABSTRACT

Objective: There remains a significant need for precise biomarkers to diagnose cardiac contusion. The objective of this study is to examine the diagnostic significance of cardiac myosin-binding protein-C (cMyBP-C) in thoracic injuries, including cardiac contusion.

Methods: The inclusion criteria were patients who consented to take part, were over 18 years of age, had sustained severe blunt chest injuries, were admitted within 24 hours post-injury, had their blood samples taken within the first 2 hours of admission, and had at least one of the following injuries: sternal, rib, or left scapular fractures; hemothorax, pneumothorax, pulmonary contusion, traumatic asphyxia, or pneumomediastinum. Troponin I levels ≥ 0.04 ng/mL, measured within 2 hours of admission, are considered the gold standard for diagnosing cardiac contusion. Serum troponin-I concentrations were assessed using an immunochemical technique. Serum cMyBP-C concentrations were measured using an enzyme-linked immunosorbent assay.

Results: Participants included 85 patients, with 59 men (69.4%) and a mean age of 48.6 ± 20.8 years (range: 18-84 years). Among them, 18 individuals (21.2%) experienced cardiac contusion. No significant difference was observed in cMyBP-C levels between the cardiac contusion group and the other group (17.7 ± 18.6 vs. 14 ± 15.3, $P = 0.328$). Receiver operating characteristic analysis revealed that the discriminative performance of cMyBP-C in distinguishing contusion was not statistically significant. In this analysis, the area under the curve for cMyBP-C was 0.575 ± 0.077 (95% confidence interval, 0.425-0.726; $P = 0.328$), indicating poor discriminative ability and a lack of statistically significant diagnostic value for contusion.

Conclusion: While cMyBP-C holds theoretical promise as a cardiomyocyte-specific biomarker, our findings suggest that its current diagnostic performance may be limited in the context of blunt cardiac contusion. Further studies with larger cohorts and refined detection methods are warranted to clarify its clinical utility.

Keywords: Blunt thoracic trauma, cardiac contusion, cardiac myosin binding protein

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INTRODUCTION

Blunt thoracic trauma and its complications are important causes of trauma-related mortality.[1] Blunt cardiac injuries that develop after blunt thoracic trauma are associated with higher mortality than other organ and system injuries.[2] Forceful impact, rapid deceleration, and crush injuries, when they do not penetrate the chest or heart, are common causes of blunt cardiac trauma. Blunt cardiac injuries can lead to various complications, including myocardial contusion; rupture of the cardiac chambers; dissection, thrombosis, or both of the coronary arteries; and disruption of heart valves.[3] Myocardial contusion is the most common type of blunt cardiac injury (BCI); its incidence is primarily reported in autopsy series because the location and mechanism of injury influence survival.[4] The prevalence of myocardial contusion in individuals with blunt thoracic trauma ranges from 0% to 76%, depending on the diagnostic criteria utilized.[5] Myocardial contusion is an emergency condition requiring prompt recognition and diagnosis. However, the variable nature of the symptoms and the anxiety experienced by the patient after trauma make diagnosis difficult. Although direct chest radiography, multidetector computed tomography (CT), electrocardiography (ECG), cardiac biomarkers, transthoracic or transesophageal echocardiography (TEE), magnetic resonance imaging, focused assessment with sonography in trauma, and nuclear imaging are all used for diagnosis, definitive diagnosis can only be made by direct inspection of the damaged myocardium.[3,6]

ECG is the initial approach for identifying myocardial contusion because it is rapid, simple, inexpensive, and non-invasive. Studies report varying rates of ECG findings such as ST-segment elevation or depression, T-wave inversion, arrhythmias (atrial fibrillation, atrial flutter, supraventricular tachycardia, and premature ventricular complexes), and intraventricular conduction abnormalities, in patients with myocardial contusion.[5] However, its value in diagnosing contusion is controversial. ECG alone has low sensitivity and specificity; both measures improve when findings are supported by cardiac biomarkers.[7,8]

MAIN POINTS

- Electrocardiography (ECG) and a single troponin-I test are insufficient to diagnose blunt cardiac contusion (BCI); neither test alone provides diagnostic accuracy, particularly in the early stages of trauma assessment.
- Serial measurements of cardiac enzymes combined with ECG monitoring improve diagnostic reliability: Repeated troponin-I testing over time, alongside ECG follow-up, enhances diagnostic sensitivity and supports more accurate identification of BCI.
- Troponin-I remains the primary biomarker for BCI, but benefits from serial protocolized testing: Although troponin-I is essential, its diagnostic power increases when it is incorporated into structured testing intervals.
- There is a need for more sensitive biomarkers like cardiac myosin-binding protein-C, until such markers are validated, a multimodal approach is preferable. Emerging markers show promise, but current best practice involves using multiple diagnostic tools over time for optimal management.

Monitoring cardiac biomarkers—creatine kinase (CK), CK-myocardial band (MB) (CK-MB), troponin I (Tn-I), and troponin T is the most widely used method for diagnosing cardiac contusion in emergency services.[9,10] Following trauma, overall CK levels are often high, and CK-MB levels may also be increased. However, these markers are also elevated in severe damage to skeletal muscle, the diaphragm, the liver, or the intestine, in addition to cardiac injury, limiting their utility and reliability. Troponin-I, which is more specific to the myocardium, is a better indicator of myocardial damage[11] than CK-MB and troponin-T. Additionally, high-sensitivity cardiac troponin (hs-cTn) tests are used for the early diagnosis of acute myocardial injury.[11,12] However, its application in the identification of BCI is not definitive, and it provides no additional information compared with the ECG.[3] The usability of methods such as TEE, nuclear imaging, and dual CT is limited due to difficult access, operator dependence, and trauma-related complications. Additionally, insufficient data exist regarding the safety of their use in cardiac contusions.[13,14]

Following cardiac contusion, pathological findings such as intramyocardial hemorrhage, edema, and necrosis of myocardial cells are observed. Because cardiomyocyte damage can be examined histopathologically only in postmortem cases, the need for biomarkers with high sensitivity and specificity persists. While the degradation of thin filament proteins found in cardiac sarcomeres, including cardiac Tn-I and cardiac troponin T, has been used as a marker of cardiac damage, the degradation patterns of thick filament proteins such as titin, myosin, and cardiac myosin-binding protein C (cMyBP-C) and the consequent contractile dysfunction have not been thoroughly examined.[15] The cardiac-specific sarcomere-binding protein cMyBP-C regulates heart structure and function, and has been shown to increase after tissue damage due to myocardial infarction.[16] Additionally, cMyBP-C has been suggested as a cardiomyocyte-specific biomarker and may measure cardiomyocyte damage more accurately than hs-cTn.[17,18] It is recognized for its potential diagnostic and prognostic significance for the treatment of acute coronary syndromes.[16,19] This protein may be a biochemical marker of damage in myocardial contusion.

This study was designed to examine the use of cMyBP-C as an adjunct or alternative to Tn-I in the detection of cardiac contusion.

MATERIAL AND METHODS

Population of the Study

This study was conducted prospectively in the Emergency Department of Atatürk University Faculty of Medicine between September 2015 and June 2016. Eighty-five consecutive patients aged 18 years or older who presented to the emergency department with blunt chest trauma were included in the study. Patients with blunt chest trauma and their relatives who met the study criteria were included in the study group after obtaining their consent. This study was deemed ethically

appropriate by the Atatürk University Non-Interventional Clinical Research Ethics Committee (session number 6, dated 17.09.2015; decision number 03). It was deemed compliant with the ethical rules by the Atatürk University.

Patients were deemed eligible to participate in the study if they met all of the following criteria: were over the age of 18; were admitted to our emergency department within the first 24 hours after the trauma; had high-energy blunt chest trauma (e.g., assault, falls from heights, motor vehicle accidents in- or outside the vehicle, animal attacks, crushing, or being struck by objects); had at least one of the following findings: rib fracture, sternal fracture, hemothorax, pneumothorax, traumatic asphyxia, pulmonary contusion, pneumomediastinum, or left scapular fracture; and had blood drawn for examination within the first 2 hours after presenting to our emergency department.

The study exclusion criteria were as follows: patients who did not agree to participate in the study (n=13); who were under the age of 18; who reported chest pain that started at least 6 hours before the trauma (n=5); who were suspected of having acute coronary syndrome (n=4); who had a history of rhythm disturbance, congestive heart failure, cardiac surgery, chronic renal failure, or cancer (n=7); and who did not have any of the following findings after thoracic trauma: rib fracture, sternum fracture, hemothorax, pneumothorax, traumatic asphyxia, pulmonary contusion, pneumomediastinum, or left scapula fracture (n=92).

Marker Accepted in the Study for Cardiac Contusion Diagnosis

A Tn-I level of 0.04 ngmL or higher at initial presentation to the emergency department (within the first 2 hours) was considered indicative of cardiac contusion. Serum Tn-I levels were measured by the immunochemical method (Beckman Coulter UniCel DxI 600, USA). The measurement (reference) range of the Tn-I kit used was 0.01-40 ng/mL.

Data Collection

After clinical evaluation and stabilization, patients who met the above criteria underwent assessment for cardiac contusion using ECG and troponin measurement. In addition to the ECG and troponin I levels measured in patients with suspected cardiac contusion as part of the routine trauma protocol in the emergency department, and after obtaining consent from the patients or their relatives, blood was drawn from these patients into biochemistry tubes to measure their cMyBP-C levels. The samples were centrifuged at 4000 rpm for 15 minutes at +4°C within 1 hour of collection and stored at -80°C in a deep freezer. The storage periods in the deep freezer varied between 10 days and 24 months.

Data on patients' demographic characteristics (such as age and gender), cause and time of trauma, time of hospital admission, known diseases, vital signs, ECG findings on arrival, cardiac troponin levels, and accompanying traumas were recorded. Serum cMyBP-C levels were measured using the enzyme-linked immune sorbent assay (ELISA) method using the ''Human CMyBP-C Binding Protein (MYCBP) ELISA Kit'' (Bioassay Technology Laboratory, lot no:20151123, China) according to the manufacturer's instructions. Concentrations were calculated using the ELISA reader's KC Junior software (Bio-Tek Inc.). The intra-assay coefficient of variation (CV) of the MYCBP kit is below 8% and the inter-assay CV is below 10%.

Statistical Analysis

Statistical analyses were performed using the SPSS software package (Version 22.0; IBM Corp., Armonk, NY, USA). Continuous variables were presented as median (min-max) and/or mean ± standard deviation (SD), whereas categorical variables were expressed as frequencies and percentages. The normality of the data distribution was evaluated using the Shapiro-Wilk test, and the homogeneity of variances was assessed using Levene's test.

For comparisons between two independent groups, the Student's t-test was applied to normally distributed variables, whereas the Mann-Whitney U test was used for non-normally distributed variables. Categorical variables were compared using the chi-square (χ^2) test.

A receiver operating characteristic (ROC) curve analysis was performed to assess the diagnostic performance of cMyBP-C for predicting the presence of a contusion. The area under the ROC curve (AUC) was calculated, together with its standard error and 95% confidence interval (CI). A P value < 0.05 was considered statistically significant.

RESULTS

A total of 85 patients, 59 (69.4%) of whom were male, were included in the study. As the gold standard, a troponin I level of 0.04 ng/mL or higher at initial admission to the emergency department (within the first 2 hours) was considered diagnostic of cardiac contusion. According to this gold standard, 18 patients (21.2%) had cardiac contusion. Patient characteristics, vital signs, and Glasgow Coma Scale (GCS) are summarized in Table 1.

The mean age was 48.1 ± 21.4 years in the cardiac contusion (-) group and 50.6 ± 18.7 years in the cardiac contusion (+) group. However, this difference was not statistically significant (P = 0.649). The proportion of males was 71.6% in the cardiac contusion (-) group and 61.1% in the cardiac contusion (+) group. There was also no significant difference in gender distribution (P = 0.389). The median (min-max) of systolic blood pressure in the contusion (+) group was 117 (64-180), and the mean (Mean ± SD) was 116.3 ± 25.4, which was lower than the mean in the contusion (-) group. However, this difference was not statistically significant (P =0.070). Nevertheless, a tendency toward decreased blood pressure was observed in the presence of cardiac contusion.

Diastolic blood pressure was also lower in the contusion (+) group (69.1 ± 20.4), but this difference was not statistically significant (P = 0.138). The mean pulse in the contusion (+) group was 93.6 ± 24.6, and in the (-) group was 86.9 ± 15.1. However, this difference was also not statistically significant (P = 0.285). No significant differences were observed between the two groups for respiratory rate, fever, and oxygen saturation (P = 0.833, 0.396, and 0.935, respectively). The mean ± SD and

median (min-max) of GCS in the contusion (+) group were 11.7 ± 5.5 and 15 (3-15), respectively; in the contusion (-) group, they were 14.2 ± 2.7 and 15 (3-15), respectively. This indicates that cardiac contusion reduces GCS, although this reduction was not statistically significant (P = 0.058).

ECG Findings

When the ECGs collected in our study were examined, normal sinus rhythm was detected in 50 of the 85 patients (58.8%). The most common pathological finding was sinus tachycardia, observed in 18 patients (21.2%). Table 2 lists the ECG changes observed in the patients, ordered by frequency.

Analysis of cMyBP-C According to Troponin-I

The median (min–max) and mean cMyBP-C values in cardiac contusion (+) patients are higher than those in cardiac contusion (-) patients. However, no statistically significant difference was detected (Table 3).

Diagnostic Value of cMyBP-C in Detecting Cardiac Contusion

According to the ROC analysis, the discriminatory power of CMyBP-C levels for the diagnosis of contusion was not statistically significant (AUC = 0.575; SE = 0.077; 95% CI: 0.425-0.726; P = 0.328). The obtained AUC was close to 0.5, indicating that the CMyBP-C had limited diagnostic value for detecting contusion (Figure 1).

Table 1. The Characteristics of the Population

Characteristics	Total n=85	Cardiac contusion (-) n=67	Cardiac contusion (+) n=18	*P*
Age*	48.6 ± 20.8	48.1 ± 21.4	50.6 ± 18.7	0.649
Male*	59 (69.4%)	48 (71,6%)	11 (61.1%)	0.389
Systolic blood pressure**				
Median (min-max)	122 (64-191)	123 (80-191)	117 (64-180)	0.070
Mean ± SD	126.1 ± 23.7	128.7 ± 22.7	116,3 ± 25,4	
Diastolic blood pressure*	75.2 ± 14.6	76.9 ± 12.3	69.1 ± 20.4	0.138
Pulse*	88.4 ± 17.6	86.9 ± 15.1	93.6 ± 24.6	0.285
Respiratory rate**				
Median (min-max)	17 (10-38)	17 (10-34)	17 (12-38)	0.833
Mean ± SD	16.9 ± 3.8	16.8 ± 3.2	17.4 ± 5.8	
Fever*	36.2 ± 0.52	36.2 ± 0.53	36.1 ± 0.52	0.396
Oxygen saturation**				
Median (min-max)	93 (68-100)	93 (70-99)	93.5 (68-100)	0.935
Mean ± SD	92 ± 5.5	92.1 ± 5.2	91.5 ± 6.9	
GCS**				
Median (min-max)	15 (3-15)	15 (3-15)	15 (3-15)	0.058
Mean ± SD	13.6 ± 3.6	14.2 ± 2.7	11.7 ± 5.5	

P value of < 0.05 was considered statistically significant. *According to the Student's *t*-test, **according to the Mann-Whitney U test was used.
GSC, Glasgow Coma Scale; SD, standard deviation.

Table 2. ECG Changes of the Patients

ECG findings	n (85)	%
Normal sinus rhythm	50	58.8
Sinus tachycardia	17	20
T (-) in the anterior	5	5.9
Early repolarization	3	3.5
Ventricular extra beat	3	3.5
Sinus bradycardia	2	2.4
Sinus arrhythmia	2	2.4
Right bundle branch block	2	2.4
Atrial fibrillation	1	1.2

ECG, electrocardiography.

Table 3. Mean Levels of cMyBP-C in Cardiac Contusion Negative and Positive Patients

Feature	Cardiac contusion (-) n=(67)	Cardiac contusion (+) n=(18)	*P**	Total
cMyBP-C Median (min-max) Mean ± SD	7.2 (1.6-56.1) 14 ± 15.3	9 (3.6-55.9) 17.7 ± 18.6	0,328	7.3 (1.6-56.1) 14.8 ± 16

*P value of < 0.05 was considered statistically significant according to Mann-Whitney U test.
cMyBP-C, cardiac myosin-binding protein-C; SD, standard deviation.

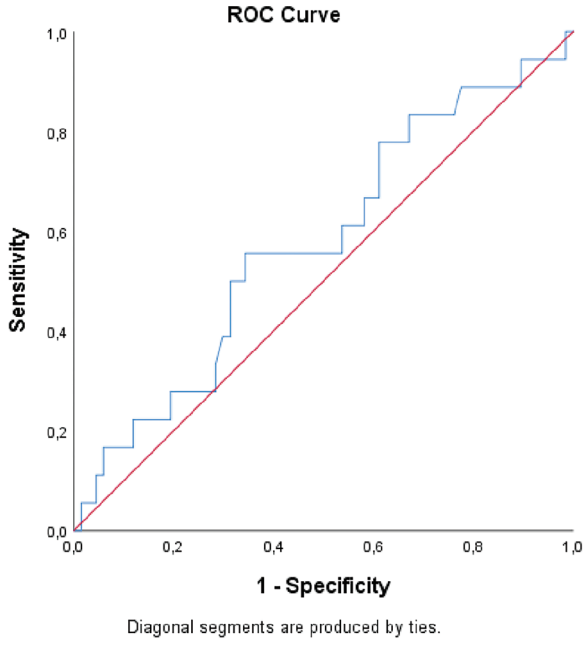

Figure 1. ROC value of cMyBP-C in determining cardiac contusion.

ROC, receiver operating characteristic; cMyBP-C, cardiac myosin-binding protein-C.

DISCUSSION

Cardiac contusion is an important and insidious injury that is often overlooked in trauma patients, is difficult to diagnose, and can affect morbidity and mortality. Despite numerous studies on cardiac contusions, are no universally accepted diagnostic criteria for trauma patients admitted to emergency departments. The cMyBP-C protein is crucial for cardiac contraction. It is thought that the concentration of this protein may increase in the blood early after cardiac contusion due to cellular damage. Therefore, in this study investigating the sensitivity and specificity of biochemical markers in cardiac contusion caused by blunt chest trauma, cMyBP-C levels were examined in patients who had troponin (+) and were considered to have cardiac contusion.

Troponin and CK-MB are initial screening tools used to detect cardiac injury. The increased sensitivity and specificity relative to creatine phosphokinase-MB are due to the presence of serum cardiac troponins, which are regulatory contractile proteins exclusive to heart muscle cells and absent from skeletal muscle. When the cardiac muscle is damaged, membrane integrity is disrupted, leading to the release of cardiac troponins into the bloodstream. Troponin is considered invaluable in the diagnosis of heart damage; positive serum cardiac troponins accurately diagnose cardiac contusion, whereas negative serum troponins strongly indicate the absence of disease.[14] Among the markers used to detect myocardial damage to date, Tn-I is a more specific marker for detecting cardiac contusion.[5]

Nonetheless, it is important to acknowledge that considerable cardiac damage may occur without significant troponin elevation. Studies have also reported that patients with initially normal Tn-I who showed increases after 24 hours were positive for BCI, and it has been suggested that troponin release may reflect secondary ischemic insults—hemorrhage or shock—rather than direct myocardial contusion in severely injured patients.[7] Another study demonstrated that patients receiving pre-hospital norepinephrine treatment had notably higher troponin levels, and that these levels are significantly associated with mortality in patients with BCI.[20] The study, which evaluated the precision of diagnostic assessments for identifying BCI and reviewed the literature over an extended period, reported that Tn-I had a sensitivity of 64.4% and a specificity of 84.1%. Also, ECG demonstrated a sensitivity of 55.1% and a specificity of 84.5% in the same study. However, when both ECG and Tn-I are positive, the diagnostic sensitivity for BCI is reduced. Conversely, when only one test is positive, sensitivity increases while specificity decreases. A normal ECG combined with a normal Tn-I level is highly effective in excluding BCI.[14]

In our study of 85 patients with blunt chest trauma, Tn-I values measured at initial presentation (< 2 hours) were elevated in 18 (21.2%). In light of all this literature, we accepted it as the gold-standard diagnostic test and considered Tn-I (+) patients to have cardiac contusion.

The ECG is a simple, rapid, non-invasive, and crucial diagnostic tool for detecting BCI in patients with recent chest trauma. However, the value of ECG changes in diagnosing cardiac contusion remains highly controversial. The ECG after a BCI may be normal or exhibit abnormalities.[9] ECG abnormalities may result from catecholamine release provoked by traumatic stress. However, lung pathologies, existing coronary artery disease, hypovolemia, abnormal arterial blood gases, serum electrolyte and pH disturbances, and changes in vagal tone may also cause changes on the ECG. The type of disturbance in BCI depends on the injured portion of the heart.[21] Because the left ventricle has greater myocardial mass, ECG findings primarily reflect its electrical activity, making the test relatively insensitive to right ventricular involvement. Mild right ventricular contusions may produce only nonspecific ECG changes, whereas more severe

injury may lead to a transient right bundle branch block.[22] For these reasons, the reliability of the ECG alone for the diagnosis of cardiac contusion is debatable. In this study, ECG abnormalities were detected in 35 (41.2%) of 85 patients. In many studies, the most common ECG abnormalities were ST-segment and T-wave changes, and supraventricular tachycardias.[5,23] In our study, we detected sinus tachycardia as the most common ECG abnormality.

During tachycardia, stimulation of β-adrenergic receptors activates intracellular signaling pathways that enhance myocardial contractility. In this context, phosphorylation of cMyBP-C, a cardiac-specific sarcomeric regulatory protein, enhances contractile efficiency by modulating the structural and functional dynamics of the myocardium. Importantly, cMyBP-C is released into the circulation when cardiomyocytes are injured; studies in both rats and humans with acute myocardial infarction (MI) have demonstrated that plasma cMyBP-C levels rise significantly within the first hour post-infarction—before any detectable increase in Tn-I—supporting its role as a rapid, cardiomyocyte-specific biomarker.[16,18,24] Moreover, cMyBP-C has been shown to correlate closely with infarct size and is highly specific for cardiac injury, potentially outperforming high-sensitivity troponins in early MI detection.[17,19]

In contrast, when we examined patients with blunt myocardial injury, we found that—despite its mechanistic plausibility—plasma cMyBP-C levels did not increase significantly in Tn-I–positive cases. In this context, cMyBP-C phosphorylation under adrenergic stress may facilitate contractility; however, its release kinetics and the sensitivity of current assays limit its diagnostic utility in the setting of blunt trauma. Possible explanations include (1) degradation fragments that are rapidly cleared or present at concentrations below assay detection thresholds, (2) non-infarct-mediated mechanisms (e.g., increased proteolysis without full cardiomyocyte necrosis), and (3) confounding hemodynamic factors that alter cMyBP-C release independently of direct myocyte injury. Although cMyBP-C is a promising early biomarker in ischemic MI—where its plasma rise precedes detection by conventional troponin assays—its role in blunt cardiac contusion remains uncertain. Future research involving larger cohorts, serial time-point sampling, and more sensitive immunoassays may yet reveal a complementary role for cMyBP-C within a multimodal biomarker panel alongside troponin and ECG.

Study Limitations

Limitations of our study include its single-center design, modest sample size, and the exclusion of 12 patients who succumbed acutely to severe trauma, which may have skewed the true incidence and spectrum of BCI. Moreover, the absence of a universally agreed biomarker cut-off for cMyBP-C and the heterogeneity of our ECG and enzyme measurement intervals may have attenuated our ability to detect subtle patterns.

CONCLUSION

Neither an ECG nor a single troponin-I measurement provides a definitive diagnosis of blunt cardiac contusion. Rather, a combination of serial cardiac enzyme sampling and ECG monitoring enhances diagnostic accuracy. Troponin-I remains the cornerstone biomarker for BCI; its diagnostic sensitivity can be improved by protocolized serial testing. The search for additional, more sensitive markers, such as cMyBP-C, continues, but until a consensus emerges, a multimodal, time-dependent diagnostic approach offers the best strategy for identifying and managing cardiac contusions in trauma patients.

Ethics

Ethics Committee Approval: This study was deemed ethically appropriate by the Atatürk University Non-Interventional Clinical Research Ethics Committee (session number 6, dated 17.09.2015; decision number 03).

Informed Consent: Patients with blunt chest trauma and their relatives who met the study criteria were included in the study group after obtaining their consent.

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Footnotes

Author Contributions

Concept Design – Y.B., M.E.; Data Collection or Processing – Y.B., B.E.; Analysis or Interpretation – Y.B., M.E.; Literature Review - Y.B., F.M.S., M.E.; Writing, Reviewing and Editing – Y.B., F.M.S., B.E., M.E., F.Ö.

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