Archives of Current Medical Research
Arch Curr Med Res 2025; 6(3): 200-208

Received: 17 March 2025
Revised: 22 March 2025
Accepted: 27 March 2025
DOI: 10.47482/acmr.1647937

ORIGINAL ARTICLE

The Value of Serum Cardiac Myosin Binding Protein C (cMyBP-C) Levels in Detecting Cardiac Contusion in an Experimental Blunt Thoracic Trauma Model

Ayşegül Bayır[1] Hasan Kara[1] Bahadır Öztürk[2] Nejat Ünlükal[3] Muhammed Eseroğlu[4] Abdullah Akkuş[5] Fatih Akbuğa[6] Fatih İkiz[7]

1 Selçuk University, Faculty of Medicine, Department of Emergency Medicine, Konya, Türkiye
2 Selçuk University, Faculty of Medicine, Department of Medical Biochemistry, Konya, Türkiye
3 Selçuk University, Faculty of Medicine, Department of Histology and Embryology, Konya, Türkiye
4 Bünyan State Hospital, Department of Emergency Medicine, Kayseri, Türkiye
5 Cihanbeyli State Hospital, Department of Emergency Medicine, Konya, Türkiye
6 Şuhut State Hospital, Department of Emergency Medicine, Afyon, Türkiye
7 Beyhekim State Hospital Department of Emergency Medicine, Konya, Türkiye

Abstract

Background: The aim of this study is to investigate the usability of Cardiac Myosin Binding Protein C (cMyBP-C) as a marker in demonstrating cardiac damage in the blunt thoracic trauma model.

Methods: It was provided in the trauma groups bilateral blunt thoracic trauma (3.31 joules to the low-energy trauma group, 6.62 joules to the medium-energy trauma group, 9.93 jouls to the high-energy trauma group). Blood samples were taken 0th, 12th and 24th hours to examine cMyBP-C levels from the subjects. At the end of the experiment (24th hour), lung tissues were taken from the subjects for histopathological examination.

Results: The mean cMyBP-C levels of the trauma groups at the 24th hour were statistically significantly higher than the control group (p=0.02). The mean cMyBP-C levels at the 24th hour of the low-energy trauma group, the 0th hour of the medium-energy trauma group, and the 12th and 24th hours of the high-energy trauma group were significantly higher than the control group (p=0.004, 0.007, 0.02, 0.03 respectively). A significant positive correlation was found in the bilateral correlation analysis between the contusion levels at 0th and 24th hours and cMyBP-C levels of the trauma groups (Spearman's ϱ= 0.396, p=0.04, spearman's ϱ= 0.473, p=0.01, respectively) . It was determined that the 24th hour cMyBP-C values (ng/ml) the sensitivity and specificity was 83.3% at the cut-off value of 11,150.

Conclusion: cMyBP-C levels increase in cardiac contusion due to blunt thoracic trauma. However, more comprehensive experimental and clinical studies are required for the use of cMyBP-C as a biomarker in the diagnosis of cardiac contusion due to blunt thoracic trauma.

Keywords: Cardiac Myosin Binding Protein C, myocardial injury, biomarkers, chest trauma

Corresponding Author:
Prof. Dr. Ayşegül Bayır, Selçuk University Faculty of Medicine, Department of Emergency Medicine, Yeni İstanbul Street, Alaeddin Keykubad Campus, +90 42150, Selçuklu/Konya, Türkiye
E-mail: aysegulbayir@hotmail.com

INTRODUCTION

Cardiac contusion is a cardiac injury due to blunt thoracic trauma. Mild cardiac contusions usually heal without fatal or prolonged complications. Severe cardiac contusion is a serious medical problem that may result in death (1-4).

Cardiac myosin-binding protein C (cMyBP-C) is a cardiac muscle-specific thick filament protein associated with the thick filament, a signaling node in cardiac myocytes that contributes to maintaining sarcomeric structure and regulating contraction and relaxation. Serum cMyBP-C levels have been determined to be elevated in the early stages of myocardial infarction (MI) as an indicator of myocardial damage. Moreover, some studies have reported that circulating cMyBP-C levels increase cardiovascular stress and cardiovascular diseases other than MI and that it is a sensitive indicator of cardiovascular diseases (5).

The study aimed to measure cardiac cMyBP-C levels in serum and investigate the diagnostic value of this biomarker in diagnosing cardiac contusion in mild, moderate, and severe blunt thoracic trauma models experimentally induced in rabbits.

MATERIALS AND METHODS

The study was conducted with the approval of the ethics committee of the Selçuk University Experimental Medicine Research and Practice Center with the date of 26.03.2021 and decision number 2021-28.

A total of 27 New Zealand rabbits between one and two years of age were used in the study. The subjects were divided into four groups: control (n=7), low-energy trauma (n=7), moderate-energy trauma (n=7), and high-energy trauma groups (n=7).

Rabbits in the control group were not traumatized. After anesthesia (intramuscular 40 mg/kg Ketamine HCl and 10 mg/kg Xylazine HCl), 5 ml of blood was collected from the catheterized dorsal auricular artery to evaluate cMyBP-C levels. Then, 5 ml isotonic NaCl was given from the catheterized dorsal auricular vein for replacement. Subjects in this group were sacrificed 24 hours later with high-dose ketamine (60-80 mg/kg). Lung tissues were sampled for histopathologic examination.

Rabbits in the low-energy trauma group underwent low-energy (3.3 joules) blunt thoracic trauma after anesthesia. Rabbits in the moderate-energy trauma group received moderate-energy blunt thoracic trauma (6.62 joules) after anesthesia. Rabbits in the high-energy trauma group were subjected to high-energy blunt thoracic trauma (9.93 joules) after anesthesia. Blood samples were taken from the subjects in the trauma groups to evaluate cMyBP-C levels at 0^{th}, 12^{th}, and 24^{th} hours of the experiment. Subjects who completed the observation period and were alive at the end of 24^{th} hours were sacrificed with high-dose anesthesia. Thoracotomy was performed on the sacrificed subjects, and lung tissues were obtained for histopathologic examination.

With the bilateral blunt thoracic trauma apparatus, 250 g, 500 g, and 750 g weights for low, moderate, and high-energy trauma, respectively, were dropped on the anterior thoracic wall from a height of 0.62 meters after anesthesia. The resulting energy was calculated with the formula E= mgh (E: energy, g: gravity; 10 m/s2 was taken, h: height; 62 cm and m: weight dropped; 0.25 kg, 0.50 kg, and 0.75 kg). Since the weights used were 250 g, 500 g, and 750 g and the height was 0.62 meters, the energy transferred to the thoracic wall was calculated as 3.31 joules (low-energy), 6.62 joules (moderate-energy), and 9.93 joules (high-energy), respectively.

Serum samples obtained from the subjects bloods were studied using with rabbit cMyBP-C Elisa kit and the ELISA method. Blood samples could not be taken at the 24^{th} hour because one subject in the high-energy trauma group died at the 14^{th} hour, one subject died at the 20th hour, and one subject died at the 21^{st} hour.

Immunohistopathologic investigation was made with Tunel Andy FluorTM 488 Apoptosis Detection Kit (ABP Biosciences, Cat No. a050, Lot No. AB2150A2). TUNEL-labelled cells and DAPI-labelled nuclei were counted for evaluation of TUNEL labelling by Image J program (National Institutes of Health, Bethesda, MD, USA), and Apoptotic Index [(number of TUNEL positive cells /DAPI positive nuclei) x 100] was used to evaluate TUNEL labeling.

Statistical analyses were performed using SPSS 21.0 (IBM Inc, Chicago, IL, USA). Descriptive statistics of numerical and categorical data obtained in the study were analyzed, numerical parameters were expressed as IQR (median, minimum, and maximum), and categorical variables were expressed as frequency. Shapiro-Wilk test, histogram analysis, and Q-Q plot graphs were used to conform numerical variables to normal distribution. Furthermore, Levene's test was used to analyze the ho-

mogeneity of the numerical parameters. Since the numerical data did not show normal distribution characteristics and the assumptions of homogeneity between groups were not met, non-parametric tests were preferred as statistical methods. The correlation relations between the numerical data were analyzed using Spearman's correlation coefficient. Friedman's test was used to compare the means of multiple dependent groups. Kruskal Wallis-H test was used to compare multiple means of independent groups, and the Mann-Whitney U test was used for pairwise comparisons. Binary logistic regression was used to determine the prognostic factors, and the regression fit of the models was tested using the Box-Tidwell test. The accuracy of the binary relationships and the analyses in the models were confirmed by the Hosmer-Lemeshow Test. Significant parameters affecting prognosis were subjected to ROC analysis, and diagnostic data were presented. The relationships between categorical groups and numerical parameters were summarized with boxplot graphs. The type-I error rate was taken as 5% in the whole study, and $p<0.05$ was accepted as significant.

RESULTS

The 24th hour cMyBP-C level of the trauma group was statistically significantly higher than the 24th hour cMyBP-C level of the control group ($p<0.05$, Table 1). When the cMyBP-C levels (ng/ml) of the high-energy trauma group at 0th, 12th, and 24th hours were compared with the control group, it was determined that the cMyBP-C levels at 12th (*49.09 ng/ml, p=0.02*) and 24th hours (*38.54 ng/ml, p=0.03*) of the high-energy trauma group were statistically and significantly higher than the control group. No statistically significant difference was observed between these two groups for 0th hour cMyBP-C levels (Table 2).

Table 1. Comparison of mean cMyBP-C levels of trauma and control groups at 0th, 12th, and 24th hours.

	Sample Group		p
	Control Group (n=7, %22.3)	**Trauma Group (n=21, %77.7)**	
Parameter *(ng/ml)*			
Median (min-max)			
0th hour cMyBP-C	18.44 (9.19-60.75)	25.31 (6.25-168.26)	0.69
12th hour cMyBP-C	16.72 (13.24-51.79)	24.52 (7.15-83.04)	0.28
24 hours cMyBP-C	8.20 (6.29-16.04)	16.31 (8.39-199.98)	0.02

Table 2. Comparison of Trauma and Control Groups cMyBP-C (ng/ml) Levels at 0th, 12th and 24th hours.

		Control (n=6)	Low Energy (n=7)	p	Moderate Energy (n=7)	p	High Energy (n=4)	p
cMyBp-C (ng/ml)		**Median (min-max)**	**Median (min-max)**		**Median (min-max)**		**Median (min-max)**	
	0th hour	18.44 (9.19-60.75)	18.79 (6.25-50.38)	0.62	107.67 (30.40-168.26)	0.007	30.37 (10.08-130.18)	0.47
	12th hour	16.72 (13.24-51.79)	20.19 (7.15-25.67)	0.99	33.82 (18.51-46.34)	0.06	49.09 (19.02-82.04)	0.02
	24th hour	8.20 (6.29-16.04)	46.98 (14.96-88.70)	0.004	11.37 (8.39-15.52)	0.20	38.54 (14.42-199.98)	0.03

In the Spearman's correlation analysis performed to determine the correlation between cMyBP-C levels at 0th, 12th, and 24th hours of trauma and the contusion grades in all trauma groups, a significant positive correlation was determined between both parameters at the 0th hour (*Spearman's ρ= 0.396, p=0.04*) (Table 3). Moreover, a significant positive correlation was determined between cMyBP-C levels and contusion grades of the trauma groups at the 12th hour (*Spearman's ρ= 0.473, p=0.01*) (Table 3). There was no significant correlation between the cMyBP-C levels at the 24th hour and the contusion grades in the trauma groups (Table 3).

Table 3. Correlation between cMyBP-C levels at 0th, 12th, and 24th hours and contusion grades of trauma groups.

Correlation Analysis Model*					
		0th hour			
		Contusion grade		**cMyBP-C (ng/ml)**	
		ρ	*P*	*ρ*	*P*
0th hour	**Contusion grade**	-	-	0.396	0.04
	cMyBP-C (ng/ml)	0.396	0.04	-	-
		12th hour			
		Contusion grade		**cMyBP-C (ng/ml)**	
		ρ	*P*	*ρ*	*P*
12th hour	**Contusion grade**	-	-	0.473	0.01
	cMyBP-C (ng/ml)	0.473	0.01	-	-
		24th hour			
		Contusion grade		**cMyBP-C (ng/ml)**	
24th hour	**Contusion grade**	*ρ*	*P*	*ρ*	*P*
	cMyBP-C (ng/ml)	-	-	0.176	0.41
		0.176	0.41	-	-

For the cMyBP-C levels of the traumatized subjects, ROC analysis was performed regarding the control group, and diagnostic significance values were determined in terms of trauma prediction. The cut-off and predictive values of cMyBP-C (ng/ml) levels were determined at the 0th, 12th, and 24th hours. As a result of the evaluation, the sensitivity and specificity of 24th h cMyBP-C levels (ng/ml) were determined to be 83.3% at a diagnostic cut-off value of 11,150. Besides, cMyBP-C was determined to have significant marker properties in terms of prediction of trauma (AUC=0.861, p=0.009) (Table 4, Figure 1).

Table 4. ROC curve data and diagnostic values of cMyBP-C prediction of cardiac trauma, cardiac contusion and contusion grade.

			AUC (95% CI)	Cut-off	p	Sensitivity (%)	Specificity (%)
Trauma Group*	cMyBP-C (ng/ml)	0th hour	0.630 (0.399-0.861)	17.00	0.35	72.2	50.0
		12th hour †	0.704 (0.443-0.964)	21.74	0.14	66.7	83.3
		24th hour †	0.861	11.150	0.009	83.3	83.3
Contusion Grade			0.873 (0.729-1.000)	2.05	0.006	90.5	83.3

AUC: Area under curve, ROC: Receiver operating characteristic, CI: Confidence Interval
**It refers to all subject groups that were subjected to low, moderate, and high trauma energy and for which contusion is predicted to occur. Reference Category: Control Group.*
† Based on Youden's index.

**Reference Category: Control Group. AUC: Area under curve, ROC: Receiver operating characteristic, CI: Confidence Interval*
¥ Cut-off value prediction was based on Youden's Index.

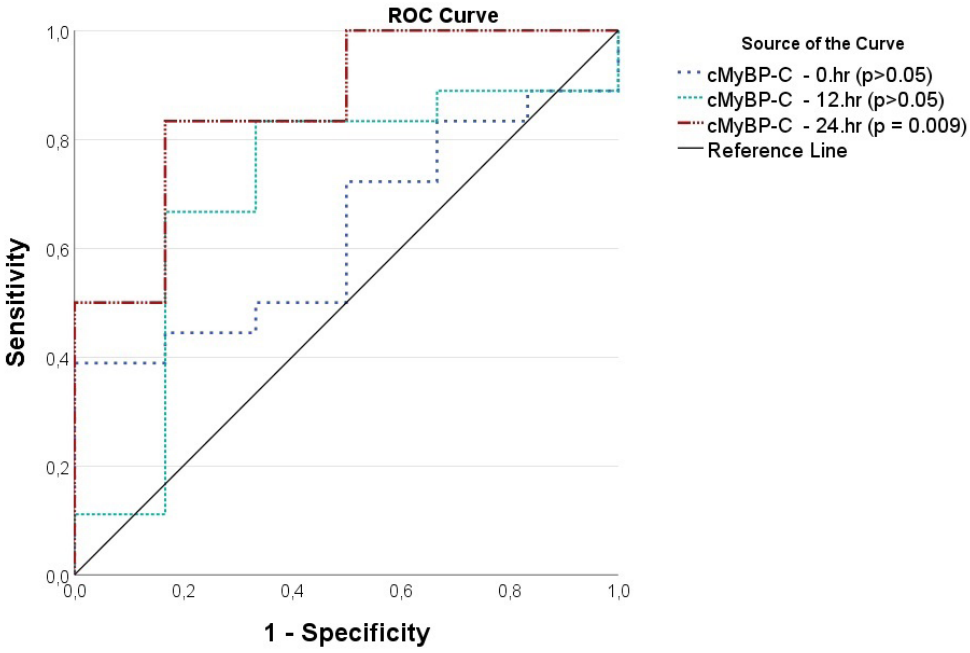

Figure 1: cMyBP-C ROC Curve (0th, 12th, 24th hours)

Taking the control group as reference, the contusion grade for the trauma group was subjected to ROC analysis, and the diagnostic value of the contusion grade was investigated. According to the statistical results, the contusion grade was determined to be diagnostically significant (p=0.006 AUC=0.87 Sensitivity=90.5% Specificity=83.3%). Furthermore, the cut-off value for the contusion grade was determined as 2.05 (Table 4, Figure 2).

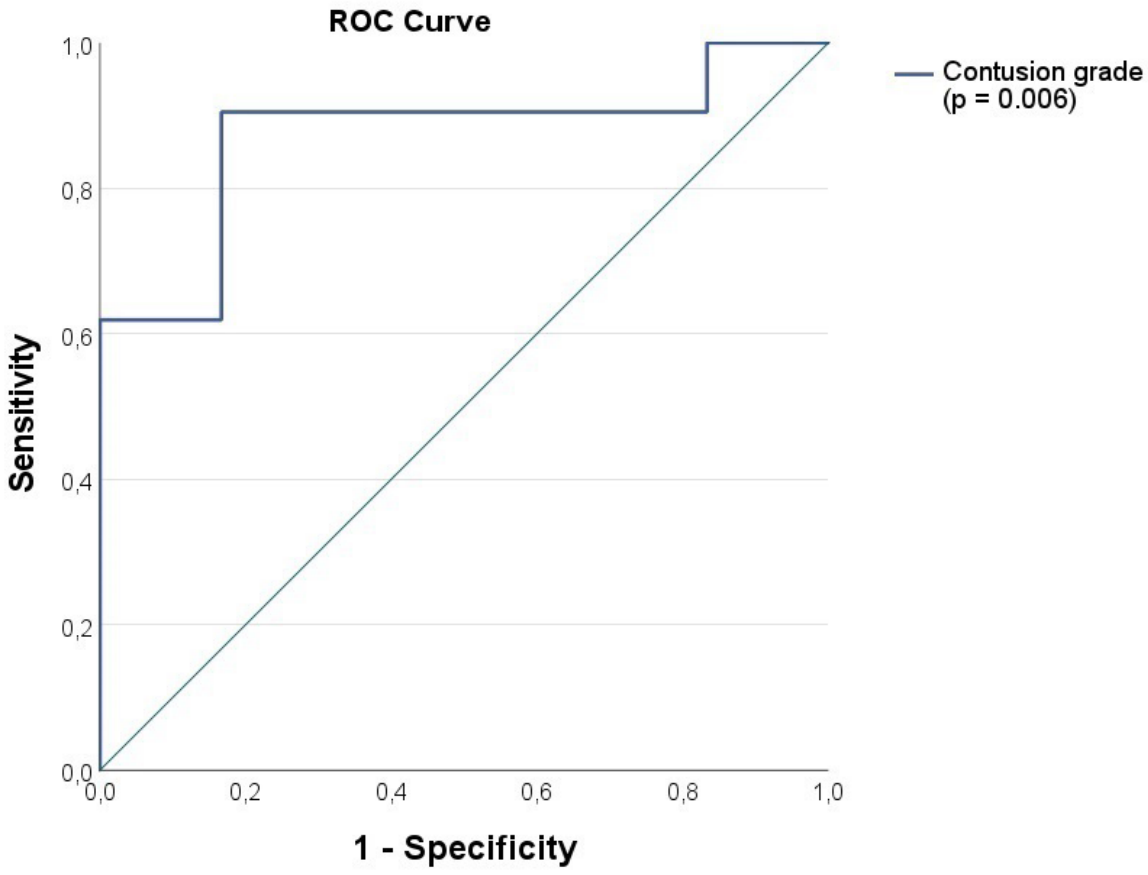

Figure 2: ROC Curve and AUC for contusion grade

DISCUSSION

The most common form of cardiac injury due to blunt thoracic trauma is cardiac contusion. Mild cardiac contusion heals spontaneously without any long-term complications. Severe cardiac contusion is a serious problem that can result in long-term complications and death. Cardiac contusion and other cardiac injuries due to blunt thoracic trauma are reported to be rare. However, the reason for this is thought to be that most cardiac contusions are missed or misdiagnosed. Diagnosis of cardiac contusion is difficult because there is no standardized diagnostic approach. Clinically, dyspnea, chest pain, and dysrhythmias are observed in patients with cardiac contusion (1-4).

CK-MB and troponin serum levels, which are markers of myocardial damage, are used in diagnosing cardiac contusion. Abnormal ECG findings are present in 40-83% of patients. Since the right ventricle is closer to the sternum, the right bundle branch block is the most common ECG finding. However, the left bundle branch block is less common. Diffuse ST-T wave changes and pathologic Q waves may be seen in left ventricular contusion.

Fatal dysrhythmias such as ventricular fibrillation may occur in the first 24-48 hours after trauma. The presence of atrial fibrillation on ECG is an indicator of poor prognosis. However, there is no correlation between the complexity and number of pathologic findings detected on ECG and the contusion grade. Echocardiography is the most important imaging modality used for diagnostic purposes. Echocardiography provides structural and functional evaluation of the heart. It also helps to diagnose or exclude cardiac pathologies such as valvular dysfunction, septal and free wall rupture, and cardiac tamponade. Radionuclide imaging may give false negative results (1,6-12). There is no method or gold standard for the definitive diagnosis of cardiac contusion in the emergency department (7-12).

In our study, we investigated the diagnostic value of cMyBP-C levels in diagnosing cardiac contusion. We investigated the changes in cMyBP-C levels at the 0th, 12th, and 24th hours after trauma in rabbits subjected to different levels of blunt trauma to the thorax. We compared the cMyBP-C levels of the trauma groups with each other and with the control group and evaluated whether there was a change according to the severity of trauma. We determined that cMyBP-C levels of moderate (64.3, 66.3, and 89.3 ng/ml according to hours) and high (81.3, 108.0, and 207.4 ng/ml) energy trauma groups increased gradually at the 12th and 24th hours. These findings support that cMyBP-C is secreted from the damaged heart muscle in myocardial injury due to blunt thoracic trauma and can be used as a diagnostic marker. In the low-energy trauma group, we observed that cMyBP-C levels increased at the 12th hour compared to the 0th hour and decreased slightly at 24 hours (21.6, 219.1, and 149.9 ng/ml, respectively). The fact that the cMyBP-C level in the low-energy trauma group increased at the 12th hour and showed a mild downward trend at the 24th hour suggests that this group was able to compensate for mild trauma, and the healing process started early.

Although there was no statistically significant difference between the 0th hour (18.44 and 28.31 ng/dl) and 12th hour (16.72 and 24.52 ng/dl) cMyBP-C levels of the control and trauma groups, the trauma groups had higher cMyBP-C levels. The cMyBPP-C level of the trauma group at the 24th hour (16.31 ng/dl) was statistically significantly higher than the control group (8.20 ng/dl) ($p=0.02$). These results again show that it can be used as a marker to diagnose cardiac contusion.

As a biomarker, cMyBP-C has been used for diagnosis and follow-up in some cardiac pathologies such as acute heart failure, acute MI, aortic stenosis and aortic fibrosis, hypertrophic cardiomyopathy, and cardiac diastolic dysfunction. However, a study investigating cMyBP-C level in cardiac contusion is not yet available in the literature. As a biomarker, cMyBP-C has mostly been investigated in ischemic cardiac events and heart failure. Apart from these studies, cMyBP-C levels in the postoperative period were investigated in a clinical study conducted by Chen et al. in patients who underwent cardiac surgery (13). The study included 151 patients who underwent cardiac surgery. Blood cMyBP-C levels were determined before surgical intervention, at the 0th hour after cardiac intervention, between the 2nd and 48th hours during intensive care unit follow-up, and before discharge. In the study, it was observed that the cMyBP-C level increased 3.8 times at the 0th hour after surgical intervention compared to the preoperative period and reached a peak level. During the follow-up of the patients, it was reported that cMyBP-C levels decreased rapidly within 24 hours but remained elevated until discharge, and cMyBP-C levels correlated with high sensitive troponin T (hs-cTnT), CK-MB, and myoglobin levels. This study also stated that cMyBP-C was elevated at different levels according to the surgical procedure performed on the patients. The researchers reported that cMyBP-C levels were less elevated in patients undergoing coronary bypass surgery and much higher in patients undergoing valve replacement. The study concluded that cMyBP-C is a promising biomarker for monitoring cardiac surgical injury in patients undergoing cardiac surgery. When we consider cardiac surgery as an iatrogenic trauma to the myocardium, the results obtained in this study are similar to those of our study.

In a study conducted with 1330 patients admitted to the emergency department with complaints of dyspnea, the value of cMyBP-C in diagnosing acute heart failure and risk stratification was investigated (14). In the study, blood samples were taken from the patients after they were admitted to the emergency department, and cMyBP-C levels were evaluated. A diagnosis of acute heart failure was made in 548 of 1330 patients included in the study. The cMyBP-C levels of patients diagnosed with acute heart failure (72 ng/L) were significantly higher than those of patients diagnosed with other diseases (22 ng/L) (536 patients). Among patients diagnosed with acute heart failure, cMyBP-C levels were reported to be

much higher in patients with acute heart failure associated with acute coronary syndrome and pulmonary edema. A strong correlation was stated between cMyBP-C levels and hs-cTnT and N Terminal pro-brain Natriuretic Peptide (NT-proBNP) levels in patients diagnosed with heart failure. Besides, cMyBP-C had a sensitivity of 95%, specificity of 37%, negative predictive value of 88%, and positive predictive value of 61% (95% confidence interval). In conclusion, it was reported that cMyBP-C plasma concentration may be helpful in the diagnosis of patients admitted to the emergency department with suspected acute heart failure. In our study, the sensitivity and specificity of cMyBP-C in cardiac contusion were 66.7% and 83.3% at the 12th hour, and 83.3% and 83.3% at the 24th hour after trauma (p=0.009). This result shows that cMyBP-C is a marker that can be used to diagnose cardiac contusion.

In a study investigating the relationship between serum cMyBP-C levels and myocardial damage, fibrosis, and mortality due to aortic stenosis, cMyBP-C levels of 161 patients with aortic stenosis were compared with a healthy control group. In the study, it was reported that there was a relationship between cMyBP-C levels and left ventricular mass index, cardiac troponin, and comorbidity in the patient group. No relationship was determined in the healthy control group (15).

In another study by Kaier et al., prehospital cMyBP-C levels and hs-cTnT levels were evaluated in 776 patients with suspected acute MI who complained of chest pain and/or shortness of breath and who had no known pulmonary disease (16). Patients were divided into a group diagnosed with acute MI and a group with other diagnoses. The cMyBP-C levels (98 ng/L) of patients diagnosed with acute MI (173 patients) were significantly higher than those (17 ng/L) of patients without acute MI (603 patients). Furthermore, cMyBP-C had a higher discriminatory power for acute MI compared to hs-cTnT (area under the curve, 0.839 vs. 0.813; P=0.005). In conclusion, it was reported that cMyBP-C levels in blood taken very early from the onset of symptoms in patients with suspected acute MI would significantly improve the early diagnosis and triage.

In our study, unlike the studies mentioned above, we investigated the diagnostic value of cMyBP-C in myocardial damage due to experimental blunt cardiac trauma in rabbits and its relationship with the degree of damage. Levels of cMyBP-C were higher in the blood samples taken at the 0th, 12th, and 24th hours after trauma in the blunt thoracic trauma group than in the control group. In addition, there was a statistically significant difference between the cMyBP-C levels that we measured higher in the trauma group compared to the control group at the 12th and 24th hours. This result shows that cMyBP-C can be used as a marker in the diagnosis of cardiac contusion due to blunt thoracic trauma. We determined a statistically significant and positive correlation between cMyBP-Cd levels measured at the 0th and 12th hours and contusion grade in the trauma group. According to this finding, cMyBP-C level increases as myocardial damage increases.

In our study, cMyBP-C levels were monitored until the 12th hour after trauma. cMyBP-C levels were not evaluated in the subjects' blood samples at the 24th hour after trauma. Because our study was conducted to examine changes in cMyBP-C levels in the very early hours following blunt thoracic trauma.

In conclusion; according to the findings obtained in our study, cMyBP-C levels increase in cardiac contusion due to blunt thoracic trauma in the early hours after the trauma, depending on the severity of the trauma. However, our findings are limited to definitively state that cMyBP-C is a diagnostic marker for cardiac contusion due to blunt thoracic trauma. More comprehensive experimental and human studies are needed to conclude that cMyBP-C is a biomarker that can be used to detect myocardial injury due to blunt thoracic trauma.

REFERENCES

1. Nair L, Winkle B, Senanayake E. Managing blunt cardiac injury. J Cardiothorac Surg. 2023;18(1):71.
2. Singh S, Heard M, Pester JM, Angus LD. Blunt Cardiac Injury. [Updated 2022 Oct 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532267/
3. Mistry RN, Moore JE. Management of blunt thoracic trauma. BJA Educ. 2022;22(11):432-9.
4. El-Andari R, O'Brien D, Bozso SJ, Nagendran J. Blunt cardiac trauma: a narrative review. Mediastinum. 2021;25;5:28.
5. Kuster DW, Cardenas-Ospina A, Miller L, Liebetrau C, Troidl C, Nef HM, et al. Release kinetics of circulating cardiac myosin binding protein-C following cardiac injury. Am J Physiol Heart Circ Physiol. 2014:15;306(4):H547-56.
6. Bansal MK, Maraj S, Chewaproug D, Amanullah A. Myocardial contusion injury: redefining the diagnostic algorithm. Emerg Med J. 2005;22(7):465-9.
7. Sağlam Gürmen E, Tulay CM. Attention: Cardiac contusion. Ulus Travma Acil Cerrahi Derg. 2022;28(5):634-40.
8. Gautam PL, Luthra N, Kaur M, Singh J, Wander GS, Tandon R, et al. Evaluation of Myocardial Injury using Standard Diagnostic Tools and Tissue Doppler Imaging in Blunt Trauma Chest. J Clin Diagn Res. 2017;11(6):OC33-OC36.
9. Shoar S, Hosseini FS, Naderan M, Khavandi S, Tabibzadeh E, Khavandi S, et al. Cardiac injury following blunt chest trauma: diagnosis, management, and uncertainty. Int J Burns Trauma. 2021;11(2):80-9.
10. Alborzi Z, Zangouri V, Paydar S, Ghahramani Z, Shafa M, Ziaeian B, et al. Diagnosing Myocardial Contusion after Blunt Chest Trauma. J Tehran Heart Cent. 2016;11(2):49-54.
11. Kyriazidis IP, Jakob DA, Vargas JAH, Franco OH, Degiannis E, Dorn P, et al. Accuracy of diagnostic tests in cardiac injury after blunt chest trauma: a systematic review and meta-analysis. World J Emerg Surg. 2023;18(1):36.
12. Scagliola R, Seitun S, Balbi M. Cardiac contusions in the acute care setting: Historical background, evaluation and management. Am J Emerg Med. 2022;61:152-7.
13. Chen XJ, Zhang W, Bian ZP, Wang ZM, Zhang J, Wu HF, et al. Cardiac Myosin-Binding Protein C Release Profile After Cardiac Surgery in Intensive Care Unit. Ann Thorac Surg. 2019;108(4):1195-1201.
14. Kozhuharov N, Wussler D, Kaier T, et al. Cardiac myosin-binding protein C in the diagnosis and risk stratification of acute heart failure. Eur J Heart Fail. 2021;23(5):716-25.
15. Anand A, Chin C, Shah ASV, Kwiecinski J, Vesey A, Cowell J, et al. Cardiac myosin-binding protein C is a novel marker of myocardial injury and fibrosis in aortic stenosis. Heart. 2018;104(13):1101-8.
16. Kaier TE, Stengaard C, Marjot J, Sørensen JT, Alaour B, Stavropoulou-Tatla S, et al. Cardiac Myosin-Binding Protein C to Diagnose Acute Myocardial Infarction in the Pre-Hospital Setting. J Am Heart Assoc. 2019 Aug 6;8(15):e013152.

Abbreviations list

Cardiac myosin-binding protein C: cMyBP-C
Myocardial infarction: MI
N Terminal pro-brain Natriuretic Peptide: NT-proBNP

Ethics approval and consent to participate

The study was conducted with the approval of the ethics committee of the Selçuk University Experimental Medicine Research and Practice Center with the date of 26.03.2021 and decision number 2021-28.

Consent for publication

Two patients whose data were presented in our study provided written and verbal consent for the presentation of their clinical data in the case report.

Availability of data and materials

The data supporting the findings of this study are available from the corresponding author upon request.

Competing interests

The authors declare that they have no conflict of interest.

Funding

This study was supported by university scientific reaserch projects coordination center.

Authors' contributions

All authors contributed at all stages.

Acknowledgements

There are no individuals or institutions for acknowledgements.