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The Role of Relative Troponin Change in Predicting Clinical Outcome and Critical Stenosis in Patients with Chest Pain

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Abstract

Aim: Dynamic changes in troponin levels have been shown to be effective indicators of acute injury and useful for distinguishing acute injuries from chronic injuries. This study investigates the role of the change in troponin I (TnI) values in the prediction of acute myocardial infarction (AMI) and its relationship with the findings of percutaneous coronary angiographies.

Materials and Methods: The patients included here were divided into two groups: a group of patients with AMI and a group of patients without AMI. The patients diagnosed with AMI were subsequently divided into two subgroups as those with and without critical stenosis. The relative troponin change in these patients was calculated as the percentage of the difference between the first and second troponin measurements; the second measurement was taken two hours later after admission.

Results: The receiver operating curve analysis revealed that increases of more than 83.18% in ΔTnI were significant predictors of AMI and critical stenosis [sensitivity 45.24%, specificity 89.67%, area under the curve (AUC)=0.698 (95% confidence interval (CI): 0.639-0.752, p=0.001), sensitivity 56.00%, specificity 87.92%, AUC value of 0.681 (95% CI: 0.620-0.738, p<0.001), respectively].

Conclusion: The ΔTnI value is a useful marker with high negative and positive predictive values for detecting AMI in patients admitted with chest pain. It can be beneficial as an adjunctive tool to predict the critical lesion, in conjunction with percutaneous angiography.

Keywords: Troponin, acute myocardial infarction, chest pain, coronary stenosis, outcome

Introduction

Chest pain suggestive of acute myocardial infarction (AMI) is one of the main reasons for admission to emergency departments (ED) worldwide (1). Serial electrocardiography (ECG) and troponin values must monitor these patients (2). Identification of low-risk cardiac patients might be quite useful in alleviating the patient burden and the length and cost of hospital stay (3).

Troponin I (TnI) is a cardiac-specific protein released after myocardial injury (4). TnI levels measured are used to diagnose AMI, exclude other diagnoses as well as demonstrate the clinical outcome of the disease (5,6). Despite being an indicator of AMI, elevated troponin values are not specific for the diagnosis of AMI (7). Dynamic changes in cardiac troponin are effective in demonstrating acute injuries and distinguishing it from chronic injuries (1,8).

This study to investigated the role of the change in TnI values in the exclusion of patients with myocardial infarction admitted with chest pain and its relation with the findings of percutaneous coronary angiographies (CAG).

Materials and Methods

Study Design

This study was designed as a cross-sectional and retrospective study, which included patients admitted with chest pain, over a period of 6 months (between April-September 2021) in a third-

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level ED. The patients were divided into two subgroups according to the outcome, those without AMI (non-MI) and those who were hospitalized with suspected AMI. Based on the results from the percutaneous CAG performed on the AMI group, the patients therein were then divided into another set of subgroups as those with and without critical stenosis, where critical stenosis was defined as ≥50% stenosis in the left main coronary artery or ≥70% stenosis in a vessel other than the left main coronary artery (9). The study was approved by the Karabük University Non-Interventional Clinical Trials Ethics Committee (approval no: 2021/706, date: 18.11.2021). The Local Ethics Committee waived the requirement for informed consent, also all data were anonymized for statistical analysis. The study was conducted in accordance with the Declaration of Helsinki.

The Selection of Patients

Patients over the age 18, who were admitted to the emergency department for chest pain were included in the study. The data of the patients were analyzed by two emergency physicians and then, independently, by a cardiologist who also re-examined the patients' data according to the Fourth Universal Definition of Myocardial Infarction (4). For the final diagnosis, the patients were examined based on the troponin values measured in the ED and their ECGs and any newly developed wall motion defects observed. Patients who were found to have ST elevation or its equivalents in their ECGs during the examinations performed based on these criteria, those who were under the age of 18, those for whom follow-up troponin measurements had not been taken, and those who had concomitant complaints or non-AMI diagnosis after hospitalization, were excluded from the study.

Data Collection

The patient data recorded here included demographic (age, gender) and clinical data on presentation, first ECG (normal findings, atrial fibrillation, ST depression, T negativity, left bundle branch block, and right bundle branch block), comorbidities, laboratory results [white blood cell (WBC), glucose, urea, creatinine, sodium, potassium, hemoglobin, and troponin levels], as well as findings of the coronary angiography (if any), performed after the serial TnI measurements. Serum high-sensitive TnI was analyzed using the TnI reagent for ADVIA Centaur XP analyzer (Siemens Healthcare Diagnostics, Germany), and the threshold value was taken as the "99th percentile upper reference limit (47 ng/mL)" that was provided by the manufacturer for this kit.

The relative troponin change (ΔTnI) mentioned in the study was calculated as the percentage of the difference between the first and second troponin measurements, the latter taken at the second hour following admission.

Primary and Secondary Outcomes

The primary outcome was to determine the role of DTnI in identifying or excluding AMI in patients admitted with chest pain, whereas the secondary outcome was to investigate how ΔTnI levels relate to the findings in CAG.

Statistical Analysis

Descriptive statistics were given as mean±standard deviation and median with range for continuous variables depending on their distribution. Numbers and percentages were used as categorical variables. The normal distribution of the numerical variables was analyzed using the Shapiro-Wilk, Kolmogorov-Smirnov, and Anderson-Darling tests.

The Independent Samples t-test was used to compare two independent groups where numerical variables had a normal distribution. The Mann-Whitney U test was applied for variables without normal distribution. Pearson chi-square and Fisher's Exact tests were used to compare the differences between categorical variables in 2x2 tables. The Fisher's Freeman-Halton test is used in RxC tables.

The receiver operating characteristic (ROC) analysis using the DeLong method with the Youden index was used to determine the cut-off values of the percentage changes in troponin levels that predict hospitalization and critical stenosis. The area under the curve (AUC) and the corresponding 95% confidence interval (CI) was calculated.

For statistical analysis, "Jamovi project (2021), Jamovi (version 2.2.3.0) (Computer Software) (Retrieved from https://www.jamovi.org), JASP (version 0.16) (Retrieved from https://jasp-stats.org), and MedCalc Statistical Software Trial version (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2015) were used. The significance level (p value) was set at 0.05 in all statistical analyses. Jamovi project (2021), Jamovi (version 2.2.3.0) (Computer Software)] (Retrieved from https://www.jamovi.org), JASP (version 0.16) (Retrieved from https://jasp-stats.org) programs are free access programs for statistical analysis.

Results

Three hundred patients who applied to the emergency department with chest pain were included in the study. Nineteen were excluded due to the lack of CAG data and six were excluded as some of their laboratory data were not available. Of the patients with chest pain examined in the ED, 188 were treated as non-MI, and 87 were treated as suspicious AMI. The demographic and clinical characteristics of the non-MI and AMI groups are given in Table 1. There were significantly more female patients in the non-MI group than in the AMI group (44.9% vs.

Table 1. Demographic and clinical characteristics of the patients			
	Non-MI group (n=188)	**AMI group (n=87)**	**p**
Age (year)†	58.0±16.7	58.7±13.2	0.701**
Sex‡			
Female	84 (44.9)	19 (21.8)	**<0.001***
Male	103 (55.1)	68 (78.2)	
Comorbidities‡			
Hypertension	57 (30.6)	61 (70.9)	**<0.001***
Ischemic heart diseases	52 (28.0)	84 (97.7)	**<0.001***
Diabetes mellitus	37 (19.9)	15 (17.4)	0.755*
Chronic renal failure	4 (2.2)	4 (4.7)	0.267*
Chronic obstructive pulmonary disease	2 (1.1)	2 (2.3)	0.593*
Asthma	2 (1.1)	0 (0.0)	0.999*
Others	6 (3.2)	6 (7.0)	0.204*
ECG findings‡			
Normal ECG	121 (64.4)	56 (64.4)	0.051*
ST depression	15 (8.0)	17 (19.5)	
Negative T waves	26 (13.8)	10 (11.5)	
Left bundle branch block	9 (4.8)	2 (2.3)	
Atrial fibrillation	9 (4.8)	1 (1.1)	
Right bundle branch block	8 (4.3)	1 (1.1)	

†: Mean±standard deviation, ‡: n (%) *: Independent samples t-test, **: Pearson chi-square, Fisher's Exact, or Fisher Freeman Halton test.
ECG: Electrocardiogram, AMI: Acute myocardial infarction, MI: Myocardial infarction

21.8%, p<0.001). There was a significant difference between the groups also in terms of comorbidities. Accordingly, hypertension and ischemic heart disease was more frequent in the AMI group (p<0.001 and p<0.001). The number of patients with normal ECG findings at admission was 64.4% in each group. Accordingly, there was no significant difference between the groups in terms of ECG findings (p=0.051) (Table 1).

The distribution of the laboratory test results by the group is given in Table 2. Accordingly, significant differences were found between the groups in hemoglobin, WBC count, and serum sodium levels. The median WBC count was significantly lower in the non-MI group than in the AMI group (8.2 cells/10^9 L vs. 10.5 cells/10^9 L, p<0.001).

Variations in the levels of troponin via the serial measurements in the ED were also analyzed. The median troponin levels at the admission (1.648 vs. 3.9 ng/mL) and the 2nd hour troponin levels (4.250 vs. 4.9 ng/mL) during the follow-up were significantly higher in the AMI group (p<0.001 and p<0.001). The difference in ΔTnI levels between the groups was also significant (p<0.001) (Table 2).

The demographic and clinical characteristics of the hospitalized patients with and without critical stenosis are presented in Table 3. The mean age of the group of patients with critical stenosis was significantly higher than that of the group of patients without critical stenosis (60.5 vs. 55.8 years, p=0.107). Additionally, a significantly higher number of patients with critical stenosis was found to have hypertension compared with patients without critical stenosis (79.6% vs. 56.2%, p=0.021) (Table 3).

The comparative analysis of the laboratory test results of the with and without critical stenosis did not reveal any significant difference except for the blood glucose levels (p=0.023), the 2nd hour troponin levels (p=0.012), and ΔTnI levels (p=0.034) (Table 4). The median 2nd hour troponin level of the critical stenosis group was significantly higher than that of the without the critical stenosis group (7.512 vs. 2.029 ng/mL). The median ΔTnI levels of critical stenosis were also significantly higher compared with those without critical stenosis (132.5% vs. 15.1%).

The ROC analysis revealed that increases in ΔTnI more than 83.18% significantly predicted the requirement for AMI with sensitivity of 45.24% specificity of 89.67%, a positive predictive

value (PPV) 66.7%, and a negative predictive value (NPV) 78.1% (AUC=0.698, 95% CI: 0.639-0.752, p=0.001) (Table 5) (Figure 1A).

The optimal cut-off value of ΔTnI for the diagnostic yield of critical stenosis was 83.18%. The sensitivity and specificity of the highest percentages in the serial determination of the troponin levels were determined as 56.00% and 87.92%, respectively, with an AUC value of 0.681 (95% CI: 0.620-0.738, p<0.001) (Table 5) (Figure 1B). Accordingly, ΔTnI levels of patients with critical stenosis were significantly higher than those of patients without critical stenosis (p<0.001 for all cases) (Figure 2).

Table 2. Comparison of the laboratory investigations between the groups

	Non-MI group (n=188)	AMI group (n=87)	p
Hemoglobin (g/dL)†	13.3±2.0	14.1±1.9	**0.001****
White blood cell count (cells/10^9/L)§	8.2 (2.4-22.0)	10.5 (5.1-22.4)	**<0.001***
Sodium (mEq/L)§	137.8 (114.2-145.1)	138.9 (133.5-144.4)	**<0.001***
Potassium (mEq/L)§	4.3 (0.6-6.4)	4.2 (3.2-6.5)	0.518*
Blood glucose (mg/dL)§	117.0 (70.0-653.0)	117.0 (81.0-415.0)	0.996*
Urea (mg/dL)§	34.2 (12.8-145.5)	34.2 (14.3-184.0)	0.865*
Creatinine (mg/dL)§	0.9 (0.1-3.6)	0.8 (0.5-7.1)	0.477*
Troponin-admission (ng/mL)§	3.9 (2.4-235.3)	1,648.1 (2.5-383,347.8)	**<0.001***
2nd hour troponin (ng/mL)§	4.9 (2.5-14,206.0)	4,250.9 (2.5-174,777.9)	**<0.001***
Δ Troponin (%)	0.0 (-73.4-18115.1)	44.1 (-94.6-10683.6)	**<0.001***

†: Mean±standard deviation, §: Median (min-max), *. Mann-Whitney U test, **. Independent samples t-test.
AMI: Acute myocardial infarction, MI: Myocardial infarction, min-max: Minimum-maximum

Table 3. Demographic and clinical characteristics of the hospitalized patients with and without critical stenosis

	Inpatient group (n=87)		
	Critical stenosis (-) (n=33)	Critical stenosis (+) (n=54)	p
Age (year)†	55.8±12.4	60.5±13.4	0.107**
Sex‡			
Female	7 (21.2)	12 (22.2)	0.912*
Male	26 (78.8)	42 (77.8)	
Comorbidities‡			
Hypertension	18 (56.2)	43 (79.6)	**0.021***
Ischemic heart diseases	32 (100.0)	52 (96.3)	0.527*
Diabetes mellitus	4 (12.5)	11 (20.4)	0.353*
Chronic renal failure	3 (9.4)	1 (1.9)	0.143*
Chronic obstructive pulmonary disease	1 (3.1)	1 (1.9)	0.999*
Asthma	0 (0.0)	0 (0.0)	-
Others	2 (6.2)	4 (7.4)	0.999*
ECG findings‡			
Normal ECG	22 (66.7)	34 (63.0)	0.999*
ST depression	6 (18.2)	11 (20.4)	
Negative T waves	4 (12.1)	6 (11.1)	
Atrial fibrillation	0 (0.0)	1 (1.9)	
Left bundle branch block	1 (3.0)	1 (1.9)	
Right bundle branch block	0 (0.0)	1 (1.9)	

†: Mean±standard deviation, ‡: n (%), *: Pearson chi-square, Fisher's exact, or Fisher Freeman Halton test, **: Independent Samples t-test.
ECG: Electrocardiogram

Table 4. The comparison of the laboratory investigations between hospitalized patients with and without critical stenosis			
	Inpatient group (n=87)		
	Critical stenosis (-) (n=33)	Critical stenosis (+) (n=54)	p
Hemoglobin (g/dL)†	13.9±1.8	14.2±1.9	0.389**
White blood cell count (cells/10^9/L)§	10.9 (5.2-19.6)	10.1 (5.1-22.4)	0.333*
Sodium (mEq/L)§	138.8 (134.3-144.0)	139.0 (133.5-144.4)	0.600*
Potassium (mEq/L)§	4.3 (3.2-5.3)	4.2 (3.2-6.5)	0.305*
Blood glucose (mg/dL)§	112.0 (86.0-229.0)	125.0 (81.0-415.0)	**0.023***
Urea (mg/dL)§	34.2 (19.3-184.0)	36.4 (14.3-66.1)	0.666*
Creatinine (mg/dL)§	0.8 (0.5-7.1)	0.8 (0.5-2.1)	0.807*
Troponin-admission (ng/mL)§	1,007.6 (2.5-92,429.4)	2,527.3 (20.6-383,347.8)	0.287*
2nd Troponin (ng/mL)§	2,029.0 (2.5-111,930.4)	7,512.1 (26.4-174,777.9)	**0.012***
Δ Troponin (%)	15.1 (-73.1-3544.3)	132.5 (-94.6-10683.6)	**0.034***

†: Mean±standard deviation, §: Median (minimum-maximum), *: Mann-Whitney U test, **: Independent Samples t-test

Table 5. The receiver operating curve analysis of the diagnostic yield of the percent increase in the troponin levels predicts AMI and critical stenosis in patients		
	% Increase in troponin levels	
	AMI	Critical stenosis
AUC	0.698	0.681
Sensitivity	45.24	56.00
Specificity	89.67	87.92
Cut-off	>83.18	>83.18
95% CI	0.639 to 0.752	0.620 to 0.738
p value	**0.003**	**<0.001**
Positive predictive value	66.7%	52.6%
Negative predictive value	78.1%	89.5%
Positive likelihood ratio	4.360	4.590
Negative likelihood ratio	0.611	0.484

AUC: Area under the curve, CI: Confidence interval, AMI: Acute myocardial infarction

Discussion

Troponin values taken at different time periods, which evaluate the differences in troponin markers over time, play an important role in the determination of acute or chronic myocardial injury (3). The ΔTnI obtained via serial measurements was found to be higher in the AMI group (44.1%) than in the non-AMI group in this study. In the subgroups of the AMI group, i.e., those with and without critical stenosis, the ΔTnI values were found to be 15.1% and 132.5%, respectively. Some studies also found the ΔTnI value to be higher in patients admitted with chest pain and diagnosed with AMI (9,10). In a study by Cullen et al. (11) where the researchers considered a cut-off value of ΔTnI ≥100 in relation to the TnI value measured at the time of admission and at the second hour, they found the sensitivity for AMI to be 45.7%, whereas the specificity was 95.6%, PPV was 47.8%, and NPV was 95.3% for AMI. Similarly, in our study, where we considered ΔTnI ≥83.1, the sensitivity, specificity, PPV, and NPV for AMI was 45.2%, 89.6%, 66.7%, and 78.1%, respectively. In a similarly designed study in which the cut-off value was fixed at ΔTnI ≥117 (for the TnI value taken 0-2nd hours later), the sensitivity was 57.0%; specificity, 83.0%; PPV, 32.0%; and NPV, 93.0% for the diagnosis of AMI (12).

The sensitivity of the same cut-off value (ΔTnI ≥83.1) in identifying patients with critical stenosis was 56.0%, whereas it had a specificity of 87.9%, PPV of 52.6%, and NPV of 89.5. In a study conducted to determine the usefulness of high-sensitivity troponin T (TnT) for evaluating patients with significant critical stenosis, the serial changes in TnT were found to be effective in predicting critical stenosis. In the same study, the patient group with critical stenosis was found to have higher blood glucose levels (8). Similarly, in another study, it was shown that an increase in troponin values is useful for predicting acute coronary lesions (13). In this study, a high-ΔTnI value was also found to be effective in predicting critical stenosis. Similarly, in this study, blood glucose levels were found to be high in the subgroup of patients with critical stenosis.

In a study by Tahto et al. (14), WBC values were found to be higher in the AMI group than in the non-AMI group. However, no correlation was found between the localization of myocardial infarction and WBC (15). Similarly, WBC values were found to be higher in the group of patients who were hospitalized in this study.

Study Limitations

This study only included patients who were known to have been admitted to the hospital with chest pain. Patients with the

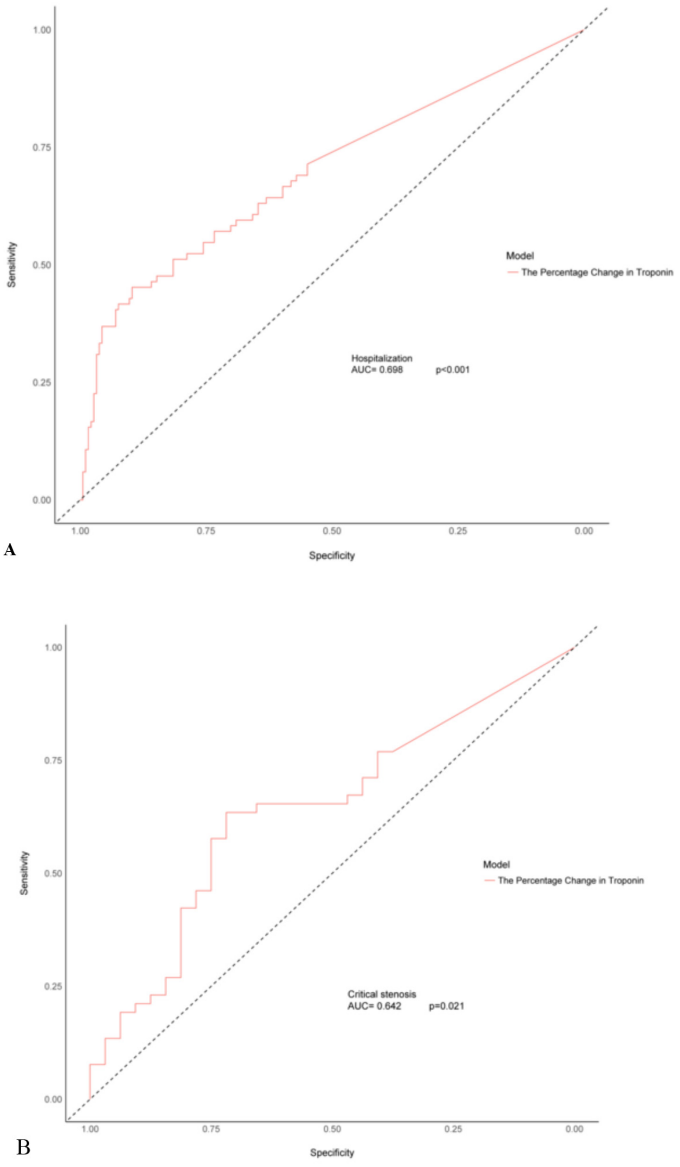

Figure 1. A) The receiver operating curve analysis of the percent change in troponin levels in predicting AMI. B) The receiver operating curve analysis of the percent change in troponin levels predicts critical stenosis in hospitalized patients

AMI: Acute myocardial infarction

equivalent of chest pain were excluded from this study. There is no data or information available regarding the onset times of the pain for the patients included in this study, which counts as a study limitation since it can affect the change in TnI values. The presence of major adverse cardiac events in the short term could not be established in the non-AMI group, which included patients who were not hospitalized during the study period. Also, we only measured the relative troponin changes in the study. Absolute troponin changes may predict different and better diagnoses of critical lesions.

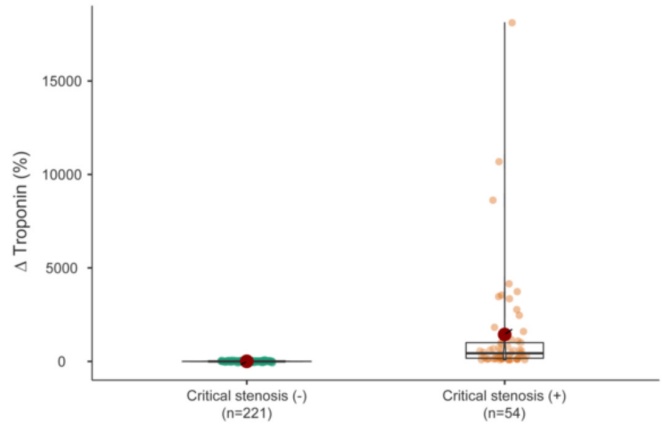

Figure 2. Graphic representation of the percent changes in the troponin levels between the hospitalized patients with critical stenosis and all patients (hospitalized and discharged) without critical stenosis

Conclusion

In conclusion, for patients admitted with chest pain, the ΔTnI value taken 0-2nd hours is a useful marker for detecting AMI and predicting the critical lesion that can only be seen via percutaneous angiographies.

Ethics

Ethics Committee Approval: The study was approved by the Karabük University Non-Interventional Clinical Trials Ethics Committee (approval no: 2021/706, date: 18.11.2021).

Informed Consent: Retrospective study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Z.K., Concept: Ş.E.A., B.Ç., Design: Ş.E.A., B.Ç., Ö.K., Data Collection or Processing: Ş.E.A., Z.K., Analysis or Interpretation: Ş.E.A., B.Ç., Literature Search: Ş.E.A., Ö.B., Ö.K., Writing: Z.K., Ö.B., Ö.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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