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Differential diagnostic factors of type 1 and type 2 myocardial infarction in patients with elevated cardiac troponin levels

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Objective Emergency physicians experience difficulty in determining the disposition of patients with elevated troponin I levels using emergency room tests. In this study, we aimed to investigate factors that could discriminate between the occurrence of type 1 myocardial infarction (T1MI) and type 2 myocardial infarction (T2MI) in patients with elevated troponin I levels.

Methods Patients admitted to the emergency department between January 1, 2017 and June 30, 2017 with elevated troponin I levels who underwent subsequent cardiac biomarker testing were included. Samples for baseline blood tests, such as cardiac biomarker levels, were collected within approximately 10 minutes of admission. Electrocardiogram, transthoracic echocardiography, and percutaneous coronary intervention results were retrospectively examined via patient report and chart reviews.

Results During the study period, 169 of 234 (72%) patients were diagnosed with T2MI and 65 (28%) were diagnosed with T1MI. Among various factors, typical chest pain (odds ratio [OR], 4.40; 95% confidence interval [CI], 1.46 to 13.24; P=0.008), high troponin I levels (OR, 1.50; 95% CI, 1.19 to 1.90; P<0.001), high cholesterol (OR, 1.01; 95% CI, 1.00 to 1.02; P=0.008), and low D-dimer levels (OR, 0.87; 95% CI, 0.77 to 0.98; P=0.027) were significantly associated with T1MI incidence.

Conclusion Our findings in this study indicate that typical chest pain, high levels of troponin I and cholesterol, and low levels of D-dimer were associated with the diagnosis of T1MI. Further studies are suggested to determine the cut-off values for accurate diagnosis of T1MI in the ED.

Keywords Troponin I; Myocardial infarction; Acute coronary syndrome

Capsule Summary

What is already known

Myocardial infarction (MI) in the emergency department is mainly type 1 MI (T1MI) or type 2 MI (T2MI) with different treatment priorities.

What is new in the current study

This study evaluated factors that distinguish T1M1 from T2MI in patients with elevated troponin I visiting the emergency department. Typical chest pain, high troponin I levels, high cholesterol levels, and low D-dimer levels were significantly associated with the diagnosis of T1MI.

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INTRODUCTION

The early diagnosis of myocardial infarction (MI) in patients admitted to the emergency department is important for their effective treatment and a good prognosis. According to data reported by the Korea National Statistical Office in 2017, heart disease is the second leading cause of death after malignant neoplasms, and its incidence is on the rise.[1] MI refers to myocardial cell damage caused by impaired blood flow to the heart due to coronary artery spasm or obstructive thrombosis and the resultant reduced myocardial oxygen and nutrient supply.[2] In general, MI is diagnosed according to clinical symptoms, and the results of electrocardiogram (ECG) and cardiac biomarker tests. However, because atypical or asymptomatic MI can occur, most emergency centers routinely and selectively perform cardiac biomarker testing in high-risk patients with heart disease. According to the third universal definition of MI updated in 2012, MI is classified into five types based on pathology.[3] Type 1 MI (T1MI) is classified as spontaneous MI related to coronary artery disease, caused by atherosclerotic plaque rupture and thrombosis. Type 2 MI (T2MI) is classified as MI related to an imbalance between myocardial oxygen supply and demand. Type 3 MI (T3MI) is classified as MI resulting in sudden death, which cannot be diagnosed without autopsy. Type 4 and 5 MI (T4MI, T5MI) are classified as MI related to percutaneous coronary intervention (PCI) and coronary artery bypass grafting performed by physicians, respectively. The differential diagnosis of T1MI and non-type 1 MI (T2MI-T5MI) is important when choosing a treatment option for MI among PCI or drug therapy.[4,5] It is difficult for emergency physicians to differentiate between T1MI and T2MI-T5MI when cardiac biomarker test results reveal elevated myocardial biomarker levels. In this study, we investigated clinical features and risk factors that could be used for the differential diagnosis of T1MI and T2MI in patients with elevated troponin I levels who were admitted to the emergency department.[6]

METHODS

Study setting and population

This study was conducted in the emergency center of a tertiary training hospital with 925 beds, where approximately 60,000 patient visits each year. Subjects in this study included patients ≥ 16 years of age who visited this hospital between January 1, 2017 and June 30, 2017, and underwent troponin I assays. Patients with ST segment elevation MI on their initial ECG or those who experienced cardiac arrest at the time of ED arrival were excluded from the study. Patients who failed to undergo follow-up tests, either because they were voluntarily discharged from the emergency department or transferred to another hospital, or died before tests could be carried out (except for myocardial biomarker tests), were also excluded. The institutional review board of the Inha University Hospital approved this study (2019-05-023). Informed consent was waived for this study due to retrospective design.

Data collection

Demographic, clinical, and laboratory data of the subjects were collected from the related electronic medical records. Based on the basic information of the admitted patients, age and sex were identified, and height and weight were measured. Other demographic data were collected by questionnaire. History of smoking, cardiac symptoms, familial history of coronary artery disease, comorbidities (diabetes mellitus, hypertension, or hypercholesterolemia), and a history of coronary revascularization, MI, stroke, and peripheral arterial occlusive disease were determined by physicians involved in the initial diagnosis and the nurses in charge. ECG findings were divided into three categories following the history, electrocardiogram, age, risk factors, and troponin (HEART) score: normal, non-specific repolarization (ST-T wave changes; abnormal but non-ischemic changes), and significant ST-depression. Transthoracic echocardiography (TTE) and coronary angiography findings were referred to in the official reports.

Chest pain in patients was defined as either typical pain or atypical pain. Typical chest pain referred to pressure or squeezing pain on the center or left-sided chest, radiating to the jaw or throat or arm, with sweating or clamminess. Atypical chest pain referred to pain on the right-sided chest, radiating to the back or worsened on inspiration or palpitation. Patients were categorized as experiencing typical pain, both typical and atypical pain, or atypical pain only.[8]

Blood measurement and troponin assay

Venous blood samples were collected from patients within approximately 10 minutes of arrival at the emergency department. Laboratory test results, including white blood cell count, platelet count, neutrophil count, and levels of hemoglobin, blood urea nitrogen, creatinine, total bilirubin, aspartate aminotransferase, alanine aminotransferase, cholesterol, potassium, chloride, N-terminal proB-type natriuretic peptide (NT pro-BNP), D-dimer, and C-reactive protein (CRP), were retrospectively studied by reviewing laboratory reports. Levels of troponin I, a cardiac biomarker, were tested in blood samples collected in EDTA (ethylenediaminetetraacetic acid) tubes (containing heparin) from the patient's vein within an average of 10 minutes. Each blood tube was sent

to the clinical hematology laboratory of Inha University Hospital for centrifugation before analysis. Measurements were conducted according to the manufacturer's instructions by using the Elecsys cobas e 411 system (Roche Diagnostic, Basel, Switzerland).

Outcome variables

Patients who showed changes from previous findings following PCI performed after admission, those with suspected ischemic heart disease based on TTE findings, and those with suspected ischemic heart disease during the one-month outpatient follow-up were defined as the T1MI group. --> In contrast, patients who demonstrated no changes from previous findings following PCI, those not suspected of having ischemic heart disease on TTE findings, and those not suspected of having ischemic heart disease during the one-month outpatient follow-up were defined as the T2MI group.

Statistical analysis

Continuous variables that are normally distributed, as determined by normality testing, are expressed as mean ± standard deviation. Continuous variables that are not normally distributed are expressed as median (interquartile range, IQR) after analysis using the Mann-Whitney U-test. Categorical variables were analyzed using the chi-squared test. Multivariate logistic regression analysis was performed after adjusting for confounding variables in the univariate analysis. All data were analyzed using IBM SPSS Statistics ver. 19.0 (IBM Corp., Armonk, NY, USA).

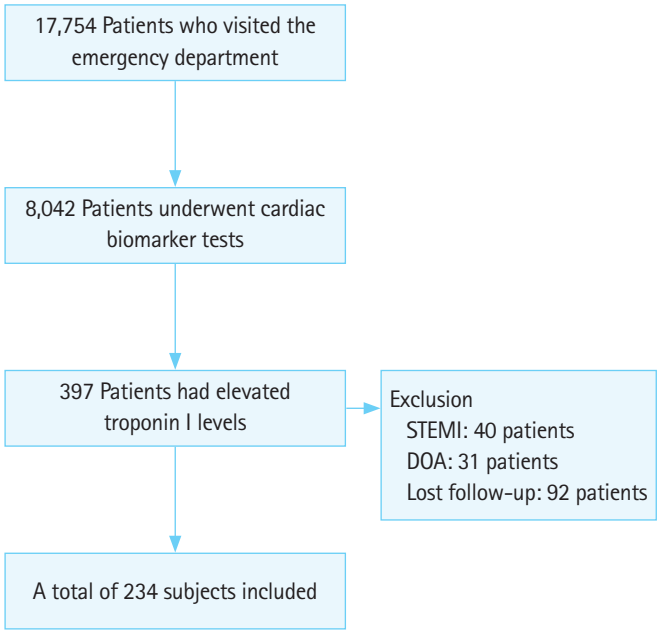

Fig. 1. Flow chart of patients included in this study. STEMI, ST segment elevation myocardial infarction; DOA, dead on arrival.

RESULTS

Characteristics of the patients

Of the 17,754 patients who visited the emergency department during the study period, 8,042 (45.3%) underwent cardiac biomarker tests due to chest pain and suspected acute heart disease or old age. Of these 8,042 patients, 397 had elevated troponin I levels (normal range, 0.000–0.160 ng/mL). Of these 397 patients, 40 were diagnosed with ST segment elevation MI on their initial ECG, 31 were in cardiac arrest at the time of ED arrival, and 92 were not followed up as they were either voluntarily discharged from the emergency department, transferred to another hospital, or died before testing. These patients were excluded from the study. Therefore, a total of 234 subjects were ultimately included in the analysis (Fig. 1).

Univariate analysis: clinical and electrocardiogram findings

Baseline patient characteristics are shown in Table 1. Of the 234 subjects included, 169 (72%) were diagnosed with T2MI and 65 (28%) with T1MI. The median age of those with T2MI was 69 years (IQR, 53 to 79 years), whereas the median age of those with T1MI was 73 years (IQR, 58 to 78 years), with no significant difference in age between the two groups (P = 0.216). The proportion of men with T2MI was 51.5% (87 of 169) and the proportion of men with T1MI was 69.2% (45 of 65), whereas the proportion of women with T2MI was 48.5% (82 of 169) and the proportion

Table 1. Comparison of patient baseline characteristics

Variable	All (n = 234)	T2MI (n = 169)	T1MI (n = 65)	P-value
Age (yr)	69 (54–78)	69 (53–79)	73 (58–78)	0.216
Sex, male	132 (56.4)	87 (51.5)	45 (69.2)	0.018*
Medical history				
Diabetes mellitus	70 (29.9)	51 (30.2)	19 (29.2)	1.000
Hypertension	114 (48.7)	77 (45.6)	37 (56.9)	0.144
Smoking	67 (28.6)	43 (25.4)	24 (36.9)	0.106
Hyperlipidemia	26 (11.1)	18 (10.7)	8 (12.3)	0.817
FHx of CAD	2 (0.9)	1 (0.6)	1 (1.5%)	1.000
Hx of CAD	26 (11.1)	16 (9.5)	10 (15.4)	0.245
Hx of MI	29 (12.4)	16 (9.5)	13 (20.0)	0.044*
Hx of stroke	32 (13.7)	20 (11.8)	12 (18.5)	0.205
Hx of PAOD	6 (2.6)	5 (3.0)	1 (1.5)	0.681
Obesity (BMI > 25 kg/m^2)	16 (6.8)	11 (6.5)	5 (7.7)	0.775

Values are presented as median (interquartile range) or number (%).
T2MI, type 2 myocardial infarction; T1MI, type 1 myocardial infarction; FHx, family history; CAD, coronary artery disease; Hx, history; MI, myocardial infarction; PAOD, peripheral arterial occlusive disease; BMI, body mass index.
*P < 0.05, significant change from baseline values.

of women with T1MI was 30.8% (20 of 65), resulting in a significantly higher proportion of men with T1MI (P = 0.018). The proportion of T2MI patients with a history of MI was 9.5% (16 of 169 subjects). History of MI was a significant factor predictive of T1MI (P = 0.044); 20.0% of patients with T1MI had a history of MI (13 of 65 subjects). Regarding other risk factors, there was no significant association found between these conditions and the presence of diabetes mellitus (P = 1.000), hypertension (P = 0.144), or hyperlipidemia (P = 0.817) among underlying conditions; history of smoking (P = 0.106) among social history; cardiovascular disease (P = 1.000) and body mass index (BMI, obesity defined as a BMI ≥ 25 kg/m^2) (P = 0.775) among family history; and history of previous PCI (P = 0.245), cerebrovascular disease (P = 0.205) or peripheral arterial occlusive disease (P = 0.681) among medical history (Table 1). T1MI was more likely in patients with typical symptoms, and T2MI was more likely in patients with atypical symptoms (T1MI n (%) vs. T2MI n (%): typical symptoms = 21 (72.4%) vs. 8 (27.6%), both typical and atypical symptoms = 11 (42.3%) vs. 15 (57.7%), and atypical symptoms = 33 (18.4%) vs. 146 (81.6%), P < 0.001). There was no significant difference in ECG findings between the T1MI and T2MI groups (T1MI n [%] vs. T2MI n [%]: normal or nonspecific ST-T wave change = 13 [18.8%] vs. 56 [81.2%], abnormal but not diagnostic ischemia = 37 [31.4%] vs. 81 [68.6%], finding of infarction or ischemia not old = 15 [31.9%] vs. 32 [68.1%], P = 0.090).

Univariate analysis: laboratory findings

Laboratory findings are shown in Table 2. In blood tests involving cardiac biomarkers, there were significant differences between the T2MI and T1MI groups, specifically in troponin I levels (median 0.34 [IQR 0.22–0.61] vs. 0.94 [IQR 0.26–2.69], P < 0.001), creatine kinase muscle and brain (CK-MB) levels (median 5.10 [IQR 2.80–11.00] vs. 8.00 [IQR 4.2–27.30], P = 0.001), CK/CK-MB ratio (median 2.96 [IQR 1.39–5.53] vs. 4.65 [IQR 2.66–8.04], P = 0.001), and cholesterol levels (median 141.00 [IQR 111–165.50] vs. 174.00 [IQR 138–201], P ≤ 0.001) (Table 2). These results show these markers were significantly higher in the T1MI group. In addition, D-dimer levels (median 2.70 [IQR 0.93–6.36] vs 1.14 [IQR 0.50–

Table 2. Comparison of laboratory findings between T2MI and T1MI

	All (n = 234)		T2MI (n = 169)		T1MI (n = 65)		P-value
	Median	IQR	Median	IQR	Median	IQR	
Cardiac enzyme							
TnI (ng/mL)	0.38	0.23–1.03	0.34	0.22–0.61	0.94	0.26–2.69	< 0.001***
CK (IU/L)	172.50	88–342.75	163.00	79.50–325.50	194.00	117–915	0.099
CK-MB (ng/mL)	5.50	2.98–12.88	5.10	2.80–11.00	8.00	4.2–27.30	0.001**
CK/CK-MB	3.72	1.62–6.25	2.96	1.39–5.53	4.65	2.66–8.04	0.001**
NT-pro BNP (pg/mL)	7,093	1,736.25–25,361.75	5,962 (n = 93)[a]	1,694.50–25,506.50	8,502 (n = 24)[a]	1,808–27,931.50	0.593
D-dimer (μg/mL)	2.28	0.70–5.36	2.70	0.93–6.36	1.14	0.50–2.74	< 0.001***
Complete blood cell count							
WBC (1,000/μL)	9.78	7.10–13.64	9.92	7.12–14.11	9.24	7.05–12.62	0.200
Hb (g/dL)	12.40	10.30–14.10	12.15	10.40–13.60	13.35	10.03–14.85	0.099
Plt (1,000/μL)	206.50	149–265	196.50	143.50–261.50	220.00	172–266.75	0.194
Neutrophil (%)	78.70	68.85–88	82.15	72.13–88.38	71.70	60.45–81.55	< 0.001***
CRP (mg/dL)	1.98	0.34–7.80	2.37	0.47–8.08	0.72	0.16–11.82	0.025*
BUN (mg/dL)	21.55	13.68–38.93	21.80	13.65–41.90	19.60	13.60–28.50	0.431
Cr (mg/dL)	1.11	0.81–2.23	1.09	0.78–2.25	1.15	0.86–1.79	0.716
Tb (mg/dL)	0.60	0.40–0.90	0.60	0.40–0.90	0.50	0.35–0.80	0.304
AST (IU/L)	37	24–63	38.00	24–67.50	36.00	25.50–53.50	0.682
ALT (IU/L)	22	14–42.75	25.00	14–50	18.00	13.50–34	0.072
Na (mEq/L)	137	133–139	136.00	132–139	137.00	134.50–139.50	0.104
K (mEq/L)	4	3.70–4.63	4.10	3.60–4.70	4.00	3.80–4.40	0.730
Cl (mEq/L)	99	95–102	99.00	95–102	100.00	97–103	0.063
Cholesterol (mg/dL)	145.50	114.75–181.25	141.00	111–165.50	174.00	138–201	< 0.001***

T2MI, type 2 myocardial infarction; T1MI, type 1 myocardial infarction; IQR, interquartile range; TnI, troponin I; CK, creatine kinase; MB, muscle and brain; NT-pro BNP, brain natriuretic peptide; WBC, white blood cell count; Hb, hemoglobin; Plt, platelet; CRP, C-reactive protein; BUN, blood urea nitrogen; Cr, creatine; Tb, total bilirubin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; Na, sodium; K, potassium; Cl, chloride.
*P < 0.05, **P < 0.01, ***P < 0.001, significant change from baseline values. [a]Missing value (n): T2MI 76 of 169 (45%), T1MI 24 of 65 (36.9%).

Table 3. Risk factors for type 1 myocardial infarction

Characteristics	Univariate analysis		Multivariate analysis	
	Unadjusted OR (95% CI)	P-value	Adjusted OR (95% CI)	P-value
Sex, male	2.12 (1.16–3.89)	0.018*	1.86 (0.82–4.21)	0.136
Medical history				
MI	2.39 (1.08–5.30)	0.044*	2.32 (0.85–6.36)	0.101
TnI (ng/mL)	1.41 (1.18–1.69)	<0.001***	1.50 (1.19–1.90)	<0.001***
CK/CK-MB	1.07 (1.01–1.15)	0.036*	1.04 (0.95–1.14)	0.369
D-dimer (μg/mL)	0.85 (0.76–0.95)	0.003**	0.87 (0.77–0.98)	0.027*
CRP (mg/dL)	0.96 (0.92–1.01)	0.084	1.01 (0.96–1.07)	0.624
Cholesterol (mg/dL)	1.01 (1.01–1.02)	<0.001***	1.01 (1.00–1.02)	0.008**
Neutrophil (%)	0.97 (0.95–0.99)	0.001***	0.99 (0.96–1.02)	0.655

OR, odds ratio; CI, confidence interval; MI, myocardial infarction; TnI, troponin I; CK, creatine kinase; MB, muscle and brain; CRP, C-reactive protein.
*P<0.05, **P<0.01, ***P<0.001, significant change from baseline values.

2.74], P<0.001), CRP levels (median 2.37 [IQR 0.47–8.08] vs. 0.72 [IQR 0.16–11.82], P=0.025), and neutrophil counts (median 82.15 [IQR 72.13–88.38] vs. 71.70 [IQR 60.45–81.55], P<0.001) were significantly higher in the T2MI group. There were no significant differences in the other blood test findings.

Multivariate analyses

The results of multivariate logistic regression analyses after controlling for confounding variables revealed statistically significant associations between the incidence of T1MI and typical chest pain (odds ratio [OR], 4.40; confidence interval [CI], 1.46–13.24; P=0.008), troponin I (OR, 1.50; CI, 1.19–1.90; P<0.001), cholesterol levels (OR, 1.01; CI, 1.00–1.02; P=0.008), and D-dimer levels (OR, 0.87; CI, 0.77–0.98; P=0.027) (Table 3).

DISCUSSION

We excluded patients with T3MI, T4MI, and T5MI as we were unable to review the charts of these patients, either because they were dead on arrival to the emergency department or had already undergone an intervention such as PCI or bypass. T2MI is typified by an imbalance between myocardial oxygen demand and oxygen supply, caused by coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension. However, many patients with pulmonary disease, anemia, septicemia, renal failure, stroke, tachycardia, or hypotension may have elevated troponin levels.[7] To the best of our knowledge, this is one of the first studies examining the use of clinical risk factors as a tool for the differential diagnosis of T1MI and T2MI in patients with high troponin levels.

It has always been challenging for emergency room physicians to differentiate T1MI and T2MI in patients with elevated troponin I levels. This study selected factors to determine the best treatment strategy for patients with elevated troponin I levels, and investigated possible correlations among the selected factors.[4] Most previous studies investigating MI involved patients presenting to the clinic with chest pain. However, this study involved patients with elevated troponin I levels who presented with atypical symptoms or even asymptomatic MI. Therefore, we believe that this study will be highly useful for managing the patients with MI in a clinical ED setting.

In this study, there was no significant difference in traditional risk factors such as age, medical history (excluding MI history) and BMI between T1M1 and T2MI patients. 69.2% of patients with T1MI were male, and there was a significant difference in the proportion of patients with MI history between the two groups, with 20% in the T1MI group and 9.5% in the T2MI group. However, results of a multivariate analysis after controlling for confounding variables revealed no significant correlation between sex or MI history with either type of MI, suggesting that it would be difficult to use these factors as tools for differential diagnosis.[9]

In blood test findings, levels of the cardiac biomarkers troponin I and CK-MB, and the CK/CK-MB ratio were significantly higher in the T1MI group (all P<0.001). Previous studies have demonstrated that use of CK as a prognostic marker has a low specificity; the results of this study also revealed no significant difference in CK.[4] Our data are consistent with the results of previous studies demonstrating that cardiac biomarkers (especially troponin I) could improve the early diagnosis of MI.[10] Moreover, this study also demonstrated that the cardiac biomarker test results were highest in the T1MI group.[5]

The relationship between inflammatory markers and non-ST elevation-acute coronary syndrome has been studied over the past several decades; however, the reasons for the elevated levels of inflammatory markers in non-ST elevation-acute coronary syndrome remain unclear.[11] Myocardial injury acts as a major inflammatory stimulus and, in turn, acute inflammation can cause myocardial injury, leading to an elevation in CRP levels.[4,11] However, CRP elevation is a nonspecific phenomenon in the acute phase, and thus, is difficult to use for diagnosis. In our study, CRP levels were found to be elevated in both the T1MI and T2MI groups, in agreement with previous studies.[12] We also looked at the neutrophil counts in each group. Neutrophils are inflammatory mediators, and the inflammatory responses of neutrophils in vulnerable plaques are thought to cause acute coronary syndrome (ACS). Activated leukocytes, including neutrophils, are also found in unstable angina; however, it is not clear whether activated leukocytes are a risk factor for stenosis.[11] Multivariate analysis correct-

ed for confounding variables revealed that CRP and neutrophil counts were not significantly associated with either T1MI or T2MI, further suggesting that they are not suitable tools for differential diagnosis.

D-dimer is the primary degradation by-product of cross-linked fibrins and is a direct marker of ongoing coagulation coupled with fibrinolysis.[13] D-dimer levels are elevated in unstable angina and acute MI.[14,15] However, D-dimer assays typically have low specificity and a wide variation in sensitivity.[13] The results of this study revealed a valid difference in low D-dimer levels, and we believe that D-dimer levels can be used as a tool to differentiate T1MI from T2MI.

Several studies have investigated the relationship between coronary artery diseases and hypercholesterolemia. Hypercholesterolemia is a well-established causal factor for MI and is an atherogenic factor.[16] In our study, cholesterol levels were found to be significantly higher in the T1MI group than in the T2MI group.

The HEART score is used to improve the stratification of the risk of chest pain causes in the emergency department.[17] However, the HEART score is based on the outcome of major adverse cardiac events, and is helpful in evaluating the risk for ACS and hospitalization in patients with chest pain, but limited in its use as a differential diagnosis tool for determining treatment strategy. When we compared our results with those from the HEART score, we also found that there was no difference in age between T1MI and T2MI patients, although there was a difference in the proportion of each sex between the two groups. In this study, in which patients with ST elevation on ECG were excluded, 65 (28%) patients were diagnosed with T1MI. Meanwhile, there were no significant differences between T1MI and T2MI patients with respect to hypertension, diabetes, history of smoking, BMI, and a family history of vascular-related diseases, which are other known risk factors.

Currently, accurate differential diagnosis of T1MI and T2MI is important for making decisions regarding treatment methods and discharge.[18] Previous retrospective studies involving patients with ACS have identified risk factors for ACS, and the findings have been studied for differential diagnosis. Several previous studies over the past few years have attempted to examine various risk factors and combine blood test results so as to differentiate between T1MI and T2MI; however, it has been difficult to definitively determine which factors are useful for making a differential diagnosis. The results of this study revealed significant differences in typical cardiac symptoms, cholesterol levels, and D-dimer levels between T1MI and T2MI patients. In agreement with other studies, we found that troponin I levels were high in the T1MI group, and there was a significant difference in troponin I levels between T1MI and T2MI patients, indicating the potential for troponin levels as a tool for differential diagnosis.[5] We believe that the findings of this study will help to differentiate between T1MI and T2MI in the clinical setting. However, future studies using accumulated data from larger-scale, multicenter trials are required to determine the cut-off values for these diagnostic markers.

This study had several limitations, among which were its single-center design and relatively small sample size; as such, it is difficult to extrapolate our results to the population as a whole. Moreover, this study used retrospective analysis through reviewing patient medical records, and patients and their caregivers were asked to answer questions regarding their medical history, including their history of hyperlipidemia or peripheral arterial disease. Thus, there may have been some omissions from the finished questionnaires. Another limitation was the possibility of selection bias; this study was conducted within the same population at consecutive time intervals. In our analysis of the NT pro-BNP, several study group patients were not available for initial blood laboratory tests for several reasons. Therefore, the results of this test was not reliable. Finally, in patients with acute coronary syndrome who experience a worsening of the disease, there may be a gradual elevation in cardiac markers and other test findings, whereas in patients with acute coronary syndrome who do not experience any further worsening, there may be a decrease in the levels of cardiac biomarkers. Therefore, there is a possibility of false negative diagnoses depending on when the tests are performed.

In summary, this study analyzed the factors that distinguish T1MI from T2MI. We found that typical chest pain, high troponin I, high cholesterol and low D-dimer were associated with T1MI rather than T2MI. We believe that these findings will help distinguish between T1MI and T2MI and provide appropriate treatment.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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