Journal of Cardiovascular Disease Research
ISSN: 0975-3583,0976-2833 VOL 15, ISSUE 11, 2024

Original Research

To determine the LVEF and their relationship to Troponin-I levels in acute STEMI patients

[1]Dr. Kamalpreet Singh, [2]Dr. Harsh Gupta, [3]Dr. Ajay Chhabra, [4]Dr. Inderjit Singh, [5]Dr. Navjot Singh

1,4-Assistant professor, Department of Medicine, GMC Amritsar, Punjab India
3-Professor and Head Department of Medicine, GMC Amritsar, Punjab India
2,5-Junior Resident, Department of Medicine, GMC Amritsar, Punjab India

Corresponding Author:
Dr. Harsh Gupta
Junior Resident, Department of Medicine, GMC Amritsar, Punjab India

Received Date:16 September 2024 Accepted Date: 25 October 2024

Abstract:
Background: ST-elevation myocardial infarction (STEMI) is a severe form of acute coronary syndrome associated with significant morbidity and mortality. Troponin I levels and left ventricular ejection fraction (LVEF) are important markers in STEMI, but their relationship is not fully elucidated.
Aims and Objectives:
1. To determine Troponin-I levels in acute STEMI patients.
2. To determine the LVEF in acute STEMI patients and its association with Troponin-I levels.
Materials and Methods: This cross-sectional study included 50 patients with first-attack STEMI admitted to Guru Nanak Dev Hospital, Amritsar. Patients underwent clinical examination, ECG, and measurement of Troponin-I levels. LVEF was assessed using 2D echocardiography. Patients were categorized based on LVEF: normal (≥55%), mildly reduced (45-54%), moderately reduced (35-44%), and severely reduced (<35%).
Results: The mean age of patients was 56.68 ± 11.11 years, with a male predominance (66%). Hypertension (56%) and diabetes mellitus (22%) were common comorbidities. The mean Troponin-I level was 13.55 ± 9.60 ng/ml, and the mean LVEF was 44.04 ± 6.76%. A strong negative correlation was observed between Troponin-I levels and LVEF ($r = -0.829$, $p < 0.001$). Troponin-I levels progressively increased as LVEF decreased across subgroups, with statistically significant differences between most LVEF categories. This relationship supports the use of Troponin-I as a potential predictor of left ventricular function in acute STEMI.
Conclusion: The findings suggest that Troponin-I levels could be a valuable tool for early risk stratification and management decisions in STEMI patients.
KEY WORDS: Left ventricular ejection fraction (LVEF), ST-elevation myocardial infarction (STEMI), Troponin-I.

INTRODUCTION:

ST-Elevation Myocardial Infarction (STEMI) is a severe form of acute coronary syndrome characterized by complete or near-complete coronary artery occlusion, which prolongs myocardial ischemia and eventually causes the damaged heart muscle to necrotize.[1] STEMI is commonly identified by electrocardiographic alterations, particularly ST-segment elevation, in combination with clinical manifestations and elevated cardiac biomarkers, including troponin. Due to its major contribution to cardiovascular-related morbidity and mortality, STEMI is a global public health concern. The prevalence of STEMI varies among populations and geographical areas and is impacted by a number of variables, such as lifestyle, sex, age, and access to healthcare.[2]

The development of atherosclerotic plaque, plaque rupture, and subsequent thrombus formation are all involved in the pathophysiology of STEMI, resulting in acute coronary artery blockage and myocardial ischemia.[3-5] When an ischemic insult occurs, the body responds by releasing cytokines and chemokines, which attracts immune cells like macrophages and neutrophils. These cells cause more plaque instability and worsen tissue damage. Restoring blood flow in a timely manner—for example, by using reperfusion therapy—is essential to save the ischemic myocardium.[6,7]

Diagnosis of STEMI requires a combination of clinical assessment, electrocardiogram, and advanced imaging modalities. Cardiac biomarkers such as troponin and creatine kinase-MB are essential for confirming myocardial damage.[8,9] Transthoracic echocardiography (TTE) is an important investigation to assess regional wall motion abnormality, left ventricular function, and possible complications.[10] Left ventricular ejection fraction (LVEF) is a critical measure for assessing cardiac function and prognosis following STEMI.[11,12]

Management of STEMI has evolved significantly over time, with an emphasis on early reperfusion therapy and adjunctive medical interventions. Primary percutaneous coronary intervention (PCI) is the recommended reperfusion method when it can be performed quickly by a qualified team.[13] Thrombolytic treatment may be

considered as an alternative reperfusion method in situations where primary PCI is not easily accessible.[14] Adjunctive therapies include dual antiplatelet medications, anticoagulants, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), and statins.[15]
Complications such as heart failure, ventricular arrhythmias, cardiogenic shock, and mechanical problems can significantly impact prognosis after STEMI. Early identification and management of these complications are crucial for improving patient outcomes.[16]
Although prior research has examined the relationship between troponin levels and clinical outcomes in patients with STEMI, there are still few thorough studies that concentrate solely on the relationship between troponin I levels and LVEF. The purpose of this study is to determine the LVEF in acute STEMI patients and their relationship to Troponin-I levels in order to fill a significant gap in the literature and potentially improve prognostic assessments and treatment strategies.

MATERIALS AND METHODS

This cross-sectional study was conducted in 50 patients admitted or attending the Medicine Emergency or Outpatient Department (OPD) of Medicine in Guru Nanak Dev Hospital attached to Government Medical College, Amritsar. Study population comprised all the patients admitted Medicine department with chest pain. Sample population was selected on the basis of brief history, targeted physical examination, ECG, troponin-I level and on the basis of inclusion and exclusion criteria. The study was carried out after seeking permission from Institutional Ethics Committee, Government Medical College, Amritsar. Written informed consent was obtained from the patients.

INCLUSION CRITERIA:

- Patients with first attack of STEMI.
- Cardiac chest pain.
- ST segment elevation of at least 2 mm in chest leads or 1 mm in limb leads.

EXCLUSION CRITERIA:

- Patients with previous history of myocardial infarction and history of heart failure (due to any cause).
- Patients with valvular heart disease, congenital heart disease and cardiomyopathy.
- Patients with major non-cardiovascular disorder causing elevation of Troponin-I such as severe renal impairment, prolonged immobilization, major surgery, chest trauma, myocarditis, pericarditis, acute pulmonary embolism, prolonged tachyarrhythmia.
- Any systemic infection.
- Patients under chemotherapy on discovery of malignancy.
- Patient not willing to get themselves enrolled in study. Considering inclusion and exclusion criteria; study population was divided into two groups.
- Group-I: Patients with first attack of STEMI LVEF: ≥55%.
- Group-II: Patients with first attack of STEMI with LVEF<55%.
- Group-II was again subdivided into 03 groups:
- Mild left ventricular systolic dysfunction defined as LVEF: 45-54%.
- Moderate left ventricular systolic dysfunction defined as LVEF: 35-44%.
- Severe left ventricular systolic dysfunction defined as LVEF: <35%.
- METHODOLOGY USED:
- After admission detailed history and clinical examination was carried out.
- Following investigations were done in all patients: Hemogram, urine examination, random blood sugar, blood urea, serum creatinine, ECG, S.CPK-MB and S. troponin I.
- Serum troponin I concentration was measured between the 12-48 hours after the onset of chest pain by the microparticle enzyme immunoassay (MEIA) on AXSYM System.
- At present there is no WHO standardization for troponin I due to the use of different troponin antibodies in the test. In our study, the test kit was calibrated with Abott's AXSYM troponin ADV. The diagnostic cut off for the AMi patient was determined to be 0.4 ng/ml.
- Echocardiography and tissue Doppler imaging patients was imaged in the left lateral decubitus position using a commercially available system (high resolution color Doppler vivid 7 ultrasound machine). Images were obtained using a 3.5-MHz transducer, at a depth of 16 cm in the parasternal and apical views (standard long-axis and two-and four-chamber images).
- Left ventricular volumes (end-systolic, end-diastolic) and LVEF was calculated from the conventional apical two and four-chamber images, using the biplane Simpson's technique.

- Ethical approval was required for the study along with a signed patient consent form in local language, for performing 2D ECHO and blood reports. Detailed clinical examination of each and every patient was recorded in proforma.

STATISTICAL ANALYSIS:

All data was initially entered into Microsoft Excel 2013, and these spreadsheets were later used for analysis. Statistical analysis was performed using SPSS version 20.0. Descriptive data were represented using various tables, graphs and diagrams. Categorical variables, were reported as frequencies and percentages, and chi-square test was employed to examine relationships between these variables. Continuous variables, were reported as the mean (M), standard deviation (SD), and range. The Pearson correlation was used to measures the strength of the linear relationship between two variables. All statistical analysis was carried out at 5% level of significance and *P* value below 0.05 was considered as significant.

Results:

TABLE 1: Demographic data

Parameter		Mean ±S.D / N (%)
Mean age		56.68±11.11
Gender distribution	Male	33(66%)
	Female	17(34%)
Diabetes mellitus		11(22%)
Hypertension		28(56%)
Family history of CAD		16(32%)
Chest pain		50(100%)
Dyspnea / Orthopnea		42(84%)

Table 2: Mean Troponin I and Left Ventricular Ejection Fraction (LVEF)

	Mean	Std. Deviation	Minimum	Maximum
Troponin I (ng/ml)	13.55	9.60	2.30	45.40
LVEF (%)	44.04%	6.76	30%	60.00%

Table 3: Correlation between Troponin I and Left Ventricular Ejection Fraction (LVEF)

		Troponin I (ng/ml)
LVEF (%)	Pearson Correlation	-0.829
	P VALUE	<0.001

Figure 1: Troponin I Levels Across LVEF Subgroups

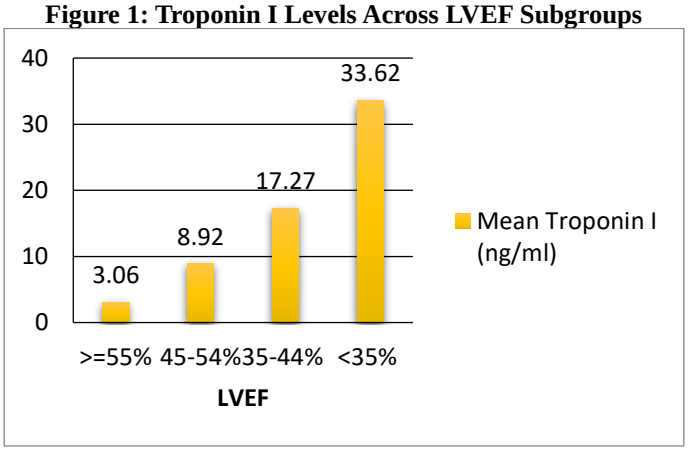
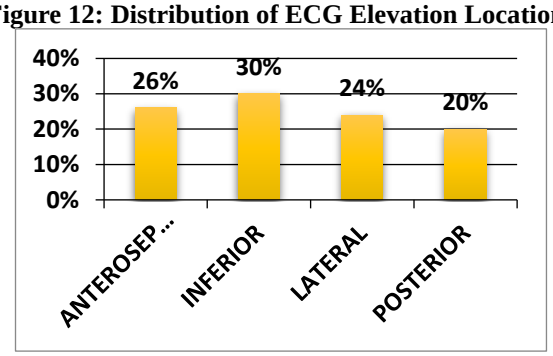


Figure 12: Distribution of ECG Elevation Locations



Discussion:

This study examined Troponin-I levels and Left Ventricular Ejection Fraction (LVEF) in 50 patients with acute ST-elevation myocardial infarction (STEMI). The degree of myocardial damage may be correlated with cTnI, and the size of the infarct and left ventricular ejection fraction are negatively correlated with cTnI release. However, there are very few studies to substantiate this claim. In patients with ST segment elevation and in which ejection fraction analysis was delayed, observed that troponin was a good indicator of depressed ejection fraction.[17]

The study population comprised 50 patients with acute STEMI, with a mean age of 56.68 ± 11.11 years. The age distribution showed that the majority of patients (40%) were in the 51-60 year age group, followed by 26% in the 61-70 year group. This distribution underscores the increasing risk of STEMI with age, which is a well-established risk factor for coronary artery disease.[18] In the study done by Azad MAet al[19]the Mean age of the patients was 49.15 ± 8.28. Most of the patients in their study were in age group 41-60 years (82.5%). The findings of our study are in concordance with HORIZONS-AMI trial reported a median age of 59.9 years for STEMI patients.[56]

The predominance of male patients (66%) in our study is consistent with the general trend in acute coronary syndrome (ACS) epidemiology, where men tend to have a higher incidence of STEMI compared to women. In the study done by Azad MA et al[19]92.5% of patients were male and only 7.5% were female. This observation was consistent with the fact that men are more likely than women to experience myocardial infarction, and there are certain risk factors which are more commonly seen in males (smoking, alcohol intake).[17]

Comorbidities were prevalent in the study population, with 56% of patients having hypertension and 22% having diabetes mellitus. According to this study among modifiable risk factors Hypertension poses more risk than diabetes for STEMI. The GRACE registry reported hypertension in 65.8% and diabetes in 25.1% of STEMI patients.[20]Zangana SN et al[21] in their study reported that Hypertension was present in 24.32% of the patients and diabetes mellitus in 27.5%.

In the present study 34 people (68%) reported no family history of CAD, while 16 individuals (32%) indicated a positive family history. This is broadly consistent with findings from other STEMI studies, a study by Panduranga et al. found that 31.7% of STEMI patients had a family history of CAD.[22] However, the INTERHEART study reported a higher prevalence, with 45.2% of myocardial infarction patients having a family history of CAD.[23]

In the present study all the patients reported experiencing chest pain as it was the inclusion criteria, Additionally, 42 individuals (84%) reported having dyspnea or orthopnea. The 100% incidence of chest pain aligns with the symptom's status as a hallmark of STEMI, though some studies report lower rates (e.g., Canto et al found 33% of MI patients presented without chest pain).[24] As atypical presentation (i.e. Diaphoresis, dizziness, fatigue) without chest pain are common in diabetes. In the study done by Patel et al dyspnea was present in about 49% of patients, and the GRACE registry reported 40-50%.[25]

Left Ventricular Ejection Fraction: The mean LVEF in our study was 44.04 ± 6.76%, ranging from 30% to 60%. Half of the patients (50%) had mildly reduced LVEF (45-54%), while 36% had moderately reduced LVEF (35-44%). These results align with a study by Planer et al[26], which reported a mean LVEF of 46 ± 11% in STEMI patients. Similarly, a study by Antoni et al[27] found a mean LVEF of 47 ± 9% in STEMI patients at baseline. The relatively preserved LVEF in many of our patients (50% with LVEF 45-54%) could be indicative of successful early reperfusion strategies, as timely restoration of blood flow can limit infarct size and preserve left ventricular function.

The mean Troponin-I level observed was 13.55 ± 9.60 ng/ml, ranging from 2.30 to 45.40 ng/ml. This wide range indicates significant variability in myocardial damage among STEMI patients. It's important to note that Troponin-I levels can be influenced by various factors, including the timing of measurement relative to symptom onset, the specific assay used, and individual patient characteristics. In our study, the mean duration of chest pain was 20.5 ± 6.746 hours, which suggests that blood samples for Troponin-I were likely taken

relatively early in the course of the infarction. This timing is crucial, as Troponin levels typically peak at 12-24 hours after symptom onset in STEMI patients.[28]

The observed Troponin-I levels are consistent with those reported in other studies of STEMI patients. For instance, Nguyen et al[29]. reported a median peak Troponin-I of 14.3 ng/ml, which is very close to our mean value. However, it's worth noting that some studies have reported higher peak values. For example, Hallén et al[30] found a median peak Troponin-I of 78 μg/L (equivalent to 78 ng/ml) in their STEMI cohort.

A strong negative correlation ($r = -0.829$, $p < 0.001$) was observed between Troponin-I levels and LVEF. This indicates that higher Troponin-I levels are associated with lower LVEF, suggesting more extensive myocardial damage leads to poorer left ventricular function. This inverse relationship can be explained by the underlying pathophysiology of STEMI. Higher Troponin-I levels indicate more extensive myocardial necrosis, which in turn leads to greater impairment of left ventricular contractile function and thus, lower LVEF.[31] This relationship is further supported by the analysis of Troponin-I levels across LVEF subgroups, which showed progressively higher Troponin-I levels with decreasing LVEF ($p < 0.001$).

Our findings are consistent with those of Hallen et al., who reported an inverse correlation between peak Troponin-T levels and LVEF in STEMI patients ($r = -0.45$, $p < 0.001$).[68] Similarly, Mayr et al[32] demonstrated that higher Troponin-T levels were associated with lower LVEF in acute myocardial infarction patients. The results of our study are also consistent with other previous studies done by Xue et al[33] and You et al[34] where high levels of admission cTnI was associated with more severe heart failure, including worse left ventricular function. Also higher value of troponins were associated with more severe symptoms, more need for aggressive supportive measures, including inotropic therapy, and worse outcomes.

The strong correlation observed in our study emphasizes the potential prognostic value of Troponin-I in predicting left ventricular function post-STEMI. This information could be valuable for risk stratification and management decisions in the acute phase of STEMI.

Conclusion: In conclusion, our study demonstrates a strong inverse relationship between Troponin-I levels and LVEF in acute STEMI patients. This finding underscores the potential utility of early Troponin-I measurement in predicting left ventricular function and highlights its possible role in risk stratification and management decisions in the acute phase of STEMI. Further research is needed to fully elucidate the clinical implications of this relationship and to explore its potential in improving patient care and outcomes in STEMI.

Bibliography

1. Choudhury T, West NE, El-Omar M. ST elevation myocardial infarction. Clin Med (Lond). 2016 Jun;16(3):277-82.
2. lendu C, Amaechi DC, Elendu TC, Omeludike EK, Alakwe-Ojimba CE, Obidigbo B, et al. Comprehensive review of ST-segment elevation myocardial infarction: Understanding pathophysiology, diagnostic strategies, and current treatment approaches. Medicine. 2023 Oct 27;102(43):e35687.
3. Bucholz EM, Butala NM, Rathore SS, Dreyer RP, Lansky AJ, Krumholz HM. Sex differences in long-term mortality after myocardial infarction: a systematic review. Circulation. 2014 Aug 26;130(9):757-67.
4. Libby P. Inflammation in atherosclerosis. Nature. 2002;420:868–74.
5. Virmani R, Kolodgie FD, Burke AP, Farb A, Schwartz SM. Lessons from sudden coronary death: a comprehensive morphological classification scheme for atherosclerotic lesions. ArterioscIerThrombVasc Biol. 2000;20:1262–75.
6. Yellon DM, Hausenloy DJ. Myocardial reperfusion injury. N Engl J Med. 2007;357:1121–35.
7. Libby P, Loscalzo J, Ridker PM, Farkouh ME, Hsue PY, Fuster V et al. Inflammation, immunity, and infection in atherothrombosis: JACC review topic of the week. J Am Coll Cardiol. 2018;72:2071–81.
8. Hausenloy DJ, Yellon DM. Myocardial ischemia-reperfusion injury: a neglected therapeutic target. J Clin Invest. 2013;123:92–100.
9. Lakatta EG. Cardiovascular aging in health. Clin Geriatr Med. 2000;16:419–44.
10. Ahmad MI, Yadaw BK, Sharma N, Varshney AK, Sharma L, Singh R, et al. Cardiac Troponin I Level in STEMI and Clinical Correlation with Left Ventricular Dysfunction in Indian Population. J Cardiovasc Dis Diagn. 2013;1(4):116.
11. Kim HW, Farzaneh-Far A, Kim RJ. Cardiovascular magnetic resonance in patients with myocardial infarction: current and emerging applications. J Am Coll Cardiol. 2009;55:1–16.
12. Radesich C, Cappelletto C, Indennidate C, Perotto M, Di Lenarda A. Predicting left ventricular functional recovery in ischaemic cardiomyopathy: needs and challenges. Eur Heart J Suppl. 2023 Apr 21;25 (Suppl B):B69-B74.
13. Partow-Navid R, Prasitlumkum N, Mukherjee A, Varadarajan P, Pai RG. Management of ST Elevation Myocardial Infarction (STEMI) in Different Settings. Int J Angiol. 2021 Mar;30(1):67-75.
14. Bhavsar PK, Dhoot GK, Cumming DV, Butler-Browne GS, Yacoub MH, Barton PJ. Developmental expression of troponin I isoforms in fetal human heart. FEBS Lett. 1991 Nov 4;292(1-2):5-8.
15. Joseph J, Koka M, Aronow WS. Prevalence of use of antiplatelet drugs, beta blockers, statins, and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers in older patients with coronary artery disease in an academic nursing home. J Am Med Dir Assoc. 2008 Feb;9(2):124-7.
16. Bodor GS, Porter S, Landt Y, Ladenson JH. Development of monoclonal antibodies for an assay of cardiac troponin-I and preliminary results in suspected cases of myocardial infarction. Clin Chem. 1992;38:2203-14.

17. Tucker JF, Collins RA, Anderson AJ, Hauser J, Kalas J, Apple FS. Early diagnostic efficiency of cardiac troponin I and Troponin T for acute myocardial infarction. Acad Emerg Med. 1997 Jan;4(1):13-21.
18. Mehta LS, Beckie TM, DeVon HA, Grines CL, Krumholz HM, Johnson MN, et al. Acute Myocardial Infarction in Women: A Scientific Statement From the American Heart Association. Circulation. 2016;133(9):916-47.
19. Azad MA, Uddin K, Barai LC, Saha BK, Khan MA, Sowdagar MN, et al. Cardiac Troponin I Level in STEMI and Clinical Correlation with Left Ventricular Dysfunction in Bangladeshi Population. Sch J App Med Sci. 2020;8(3):907-13.
20. Steg PG, Goldberg RJ, Gore JM, Fox KA, Eagle KA, Flather MD, et al. Baseline characteristics, management practices, and in-hospital outcomes of patients hospitalized with acute coronary syndromes in the Global Registry of Acute Coronary Events (GRACE). Am J Cardiol. 2002;90(4):358-63.
21. Zangana SN, Al-Othman AA, Hamad AA. Correlation of elevated cardiac troponin T level with severity and in-hospital outcomes in patients with acute ischemic stroke. Med J Babylon. 2018;15(2):174-7.
22. Panduranga P, Sulaiman K, Al-Zakwani I, Abdelrahman S. Acute coronary syndrome in young adults from Oman: results from the Gulf Registry of Acute Coronary Events. Heart Views. 2010;11(3):93-98.
23. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.
24. Canto JG, Shlipak MG, Rogers WJ, Malmgren JA, Frederick PD, Lambrew CT, et al. Prevalence, clinical characteristics, and mortality among patients with myocardial infarction presenting without chest pain. JAMA. 2000;283(24):3223-9.
25. Patel A, Mohanan PP, Prabhakaran D, Huffman MD. Pre-hospital acute coronary syndrome care in Kerala, India: A qualitative analysis. Indian Heart J. 2017;69(1):93-100.
26. Planer D, Mehran R, Ohman EM, White HD, Newman JD, Xu K, et al. Prognosis of patients with non-ST-segment-elevation myocardial infarction and nonobstructive coronary artery disease: propensity-matched analysis from the Acute Catheterization and Urgent Intervention Triage Strategy trial. Circ Cardiovasc Interv. 2014;7(3):285-93.
27. Antoni ML, Hoogslag GE, Boden H, Liem SS, Boersma E, Fox K, et al. Cardiovascular mortality and heart failure risk score for patients after ST-segment elevation acute myocardial infarction treated with primary percutaneous coronary intervention (from the Leiden MISSION! Infarct Registry). Am J Cardiol. 2012;109(2):187-94.
28. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction. J Am Coll Cardiol. 2018; 72(18): 2231-64.
29. Nguyen TL, Phan JA, Hee L, Moses DA, Otton J, Terreblanche OD, et al. High-sensitivity troponin T predicts infarct scar characteristics and adverse left ventricular function by cardiac magnetic resonance imaging early after reperfused acute myocardial infarction. Am Heart J. 2015;170(4):715-25.
30. Hallen J, Jensen JK, Fagerland MW, Jaffe AS, Atar D. Cardiac troponin I for the prediction of functional recovery and left ventricular remodelling following primary percutaneous coronary intervention for ST-elevation myocardial infarction. Heart. 2010;96(23):1892-7.
31. Chia S, Senatore F, Raffel OC, Lee H, Wackers FJ, Jang IK. Utility of cardiac biomarkers in predicting infarct size, left ventricular function, and clinical outcome after primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. JACC Cardiovasc Interv. 2008;1(4):415-23.
32. Mayr A, Mair J, Schocke M, Klug G, Pedarnig K, Haubner BJ, et al. Predictive value of NT-pro BNP after acute myocardial infarction: relation with acute and chronic infarct size and myocardial function. Int J Cardiol. 2011;147(1):118-23.
33. Xue Y, Clopton P, Peacock WF, Maisel AS. Serial changes in high- sensitive troponin I predict outcome in patients with decompensated heart failure. Eur J Heart Fail 2011;13:37–42.
34. You JJ, Austin PC, Alter DA, Ko DT, Tu JV. Relation between cardiac troponin I and mortality in acute decompensated heart failure. Am Heart J 2007;153:462–70.