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# Predictors of left ventricular systolic dysfunction after primary percutaneous coronary intervention for patients with left anterior descending artery occlusion

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# Predictors of left ventricular systolic dysfunction after primary percutaneous coronary intervention for patients with left anterior descending artery occlusion

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## Abstract

**Background**

Depressed left ventricular (LV) systolic function after acute coronary syndrome is the most important predictor affecting morbidity and mortality. The primary objective of reperfusion therapy is to restore epicardial flow and to reperfuse the myocardial tissue. However, not all the patients show improvement, and a group of patients may experience depression in heart function.

**Aim**

To determine the factors affecting impairment of LV function assessed by echocardiography following successful primary percutaneous coronary intervention (PCI) in patients with acute coronary syndrome involving the left anterior descending (LAD) artery.

**Patients and methods**

A total of 50 patients were included in this study, who underwent primary PCI and stenting to LAD artery, as a culprit artery, and applying the inclusion and exclusion criteria, patients treated with the 1-stent technique were selected.

**Results**

The results of the current study showed that 34 patients had diabetes mellitus, 29 patients had hypertension, 31 patients had dyslipidemia, and 25 patients were smokers. Female percentage was 48% and male 52%, and the mean age was $57.2 \pm 12.7$ years. The factors that showed significant difference to predict LV systolic dysfunction were previous systolic dysfunction, multivessel disease, and thrombolysis in myocardial infarction (TIMI) flow after PCI.

**Conclusion**

We concluded that patient with myocardial infarction, preprocedural depressed LV systolic function, patient with multivessel disease, and those with postprocedural TIMI flow less than III were all predictive of depressed LV systolic function after primary PCI to LAD.

**Keywords:** Left anterior descending, left ventricular systolic function, primary percutaneous coronary intervention, ST-segment elevation myocardial infarction

## Introduction

Depressed left ventricular (LV) systolic function after acute coronary syndromes (ACS) is the most important factor affecting morbidity and mortality. The primary objective of reperfusion therapy is to restore epicardial flow and to reperfuse the myocardial tissue. However, not all the patients show improvement, and a group of patients may experience depression in heart function [1].

Mortality rates because of ACS have decreased markedly over the past 30 years [2]. However, despite numerous medical advances, a substantial degree of patients with ACS experience

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subsequent clinical events, including death and myocardial dysfunction (World Health Organization, 2016).

The clinical presentation in ST-segment elevation myocardial infarction (STEMI) varies depending on the affected coronary artery. Several research contrasted results in anterior and nonanterior infarctions, stratifying patients based on patterns of ECG rather than angiography [3]. A comparison of anterior infarction with an inferior one, using ECG-based stratification, concluded that anterior infarction resulted in greater infarction size, lower left ventricular ejection fraction (LVEF) on admission, more heart failure, more in-hospital deaths, and more cardiac mortality (even after correction for infarction rate). Califf *et al*. [4] also reported tougher results with more proximal left anterior descending (LAD) artery occlusion, as well as with multivessel disease.

## Aim

The present study investigated factors associated with LV dysfunction, and deterioration of LV systolic function at follow-up echocardiography 3 months following successful primary percutaneous coronary intervention (PCI) in patients with ACS involving the LAD artery.

## Patients and methods
### Type of the study
This study was carried out as a one-center prospective, cohort, observational and nonrandomized study.

### Target population
A total of 50 consecutive patients presenting to the National Heart Institute who will undergo emergency PCI and stenting for the LAD artery were included. We selected patients treated with the 1-stent technique or main vessel stenting as the only strategy.

### Inclusion criteria
The inclusion criteria were as follows:
(1) Patients with STEMI and patients with non-ST-segment elevation MI (NSTEMI).
(2) Patients with sinus rhythm or atrial fibrillation will be included.

### Exclusion criteria
Patients were excluded if they had the following:
(1) A previous coronary balloon angioplasty.
(2) Unsuccessful angioplasty.
(3) Previous coronary artery bypass graft surgery.
(4) Evidence of bleeding disease, recent gastrointestinal bleeding, significant 6-week surgery, stroke evidence or other documented anatomical abnormality of the central nervous system, extreme hypertension, pregnancy, and elevation of baseline prothrombin period (>1.2 times control), hematocrits less than 30%, platelets less than 100 000/μl, or irregular renal function.

### Methods
All patients were subjected for the following:

(1) Full history taking, with emphasis on the following:
   (a) Personal data (age and sex).
   (b) History of cardiovascular disease, risk factors (smoking, dyslipidemia, diabetes mellitus, hypertension, and positive family history).
   (c) Previous history of coronary artery disease, PCI, or coronary artery bypass grafting.
(2) Complete clinical examination, with emphasis on the following:
   (a) General examination regarding vital signs (arterial blood pressure, pulse, temperature, and respiratory rate).
   (b) Local cardiac examination (S3 gallop, bilateral basal crepitation, elevated jugular venous pressure, hemodynamic instability, and others).
(3) Type of STEMI was diagnosed from the ECG upon his admission as follows:
   (a) ST-segment elevation and the leads affected.
   (b) Rhythm presentation (sinus, atrial fibrillation, ventricular arrhythmias, or asystole).
   (c) Conduction disturbances:
      (i) Left bundle branch block (LBBB) whether old, new, or unknown.
      (ii) Right bundle branch block (RBBB) whether old, new, or unknown.
      (iii) First-degree, second-degree, or third-degree A-V block.
(4) Laboratory workup:
   (a) Blood sample results (cardiac enzymes = CK-MB and troponin) of all the patients on admission.
   (b) Blood glucose level (random samples) at admission.
   (c) Kidney function tests (serum creatinine level) at admission.
   (d) Profile of lipids like total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides.
(5) In-hospital management:

All patients got aspirin (loading 300 mg then 150 mg per day), unfractionated heparin (70 IU/kg), and clopidogrel (600 mg as a loading dose and 75 mg once daily as a maintenance dose) or ticagrelor (loading 180 and 90 mg two times per day as a maintenance dose) in addition to conventional treatment (beta-blocker, Angiotensin-converting enzyme inhibitors (ACEI), and statin).

(a) Coronary angiography:
   (i) All patients received informed written consent.
   (ii) Glycoprotein IIb/IIIa receptor antagonists may be given at the operator's discretion.
   (iii) Sterilization and local infiltration of anesthesia of the site of puncture was routinely undertaken.
   (iv) Right femoral or right radial artery puncture using Seldinger's technique was done.
   (v) Selective left and right coronary angiographies in multiple views starting with the noninfarct related artery.
   (vi) Percutaneous transluminal coronary angioplasty (PTCA) was optionally done using a suitable balloon (usually undersized).

(vii) A stent suitable in diameter and length was inserted according to the angiographic findings in each case to the LAD artery.

(viii) The sheath removal was undertaken after normalization of the activated clotting time (ACT) or activated partial thromboplastin clotting time (APTT).

TIMI flow was evaluated as follows, before and after PCI:
1. Gr 0 (no perfusion): there is no flow of antegrade beyond the occlusion stage.
2. Gr II:(perfusion-free penetration): the contrast material goes past the obstruction region but 'hangs up' and does not opacify the entire coronary bed distally to the obstruction for the duration of the filming series.
3. Gr II (partial perfusion): the fluid of contrast passes through the obstruction and opacifies the coronary bed distally from the obstruction. However, the entry rate of contrast material into the vessel distal to the obstruction or its clearance rate from the distal bed (or both) is perceptibly slower than its entry into or clearance from comparable areas not perfused by the previous occlusion.
4. Gr III (complete perfusion): natural flow that fully fills the distal coronary bed; antegrade flow into the bed distal to the obstruction happens as rapidly as antegrade flows into the bed from the bed concerned and is as rapid as clearance from the uninvolved bed in the same vessel or artery opposite [5].

Transthoracic echocardiography was done before and after 3 months of PCI stressing on LVEF using the commercially available equipment, the digital ultrasound system with a 2–3 MHz transducer.

M-mode, two-dimensional, and Doppler echocardiographic assessments were performed for all patients.

Examinations were performed in left semilateral position with the patient, using left parasternal long, short-axis views apical four chambers, apical two chambers, and apical five champers. The LV dimensions (end-systolic and end-diastolic) were determined through acquisitions in parasternal M-mode. The LVEF percentage was determined using the following formula, from traditional apical two-chamber and four-chamber images:

$$EF\% = (EDV - ESV) / EDV \times 100 = SV / EDV \times 100$$

Where SV is stroke volume.

LV systolic dysfunction was defined as LVEF% less than 45%.

Patients were grouped into two groups those with EF more than 45% and those with LV systolic dysfunction less than 45%.

Clinical in-hospital follow-up of MACE was as follows:

Cardiovascular mortality: defined as sudden unexpected death or death correlated with acute myocardial infarction (AMI), heart failure, or arrhythmia.

Morbidity:
(1) Hospital readmission as for major arrhythmias, heart failure, or others.
(2) Reinfarction after PCI: the term reinfarction is used for an AMI that occurs within 28 days of an incident or recurrent MI [6].
(3) Target vessel revascularization: defined as repeated PCI or coronary artery bypass grafting owing to stenosis or occlusion in the infarcted related artery (IRA).
(4) Contrast-induced nephropathy: acute renal dysfunction after PCI (caused by radiographic contrast content) occurs in up to 2% of patients. Contrast-induced nephropathy is characterized as renal dysfunction and is calculated either as a 25% increase in baseline serum creatinine or an increase in absolute value of 0.5 mg/dl (44 µmol/l) within 48–72 h of intravenous contrast administration [7].

## Ethical considerations

(1) All the steps of the study were explained to the participants with its possible complications, stressing on the importance of data they were going to offer.
(2) Written informed consent was taken from patients participating in the study; this meant that the participants in the prospective study were fully informed about the procedures and risks involved in the study.
(3) All data and results of the study of the participants were confidential and were not being made available to anyone who was not directly involved in the study.
(4) Patients were informed about any abnormal results of procedures and tests performed.
(5) The patients had the right to refuse participation without affecting the medical care expected to be offered to the patients.

## Data management and data analysis

The data gathered have been updated, coded, tabulated, and added to a PC using the Social Science Statistical Kit (SPSS 23). Data were processes and analyses using IBM SPSS Statistics for Windows, Version 25.0. (IBM Corp, 2017). Data were presented, and an appropriate analysis was performed for each parameter according to the type of data collected.

Descriptive statistics were as follows:
(1) Mean and SD for numerical data.
(2) Frequency and percentage of nonnumerical data.

Analytical statistics were as follows:
(1) Student $t$ test has been used to test the statistical significance of the discrepancy between means of two study groups. $\chi^2$ test was used to examine the relationship between two qualitative variables.
(2) Fisher's exact test was used to analyze the relationship between two qualitative variables if the predicted number in more than 20% of cells is less than 5.
(3) Of the same research sample, the McNemar test was used to determine the statistical significance of the difference between a qualitative variable measured twice.

# RESULTS

The study included 50 patients. Their mean age was 57.2 ± 12.7 years. Average heart rate was 93 ± 18.9 beats/min.

Average blood pressure was 133/75.5 mmHg (Table 1).

Regarding the sex, 26 (52%) were male patients and 24 (48%) were female patients. A total of 34 (68%) patients had diabetes mellitus, 29 (58%) patients had hypertension, 31 (62%) patients had dyslipidemia, and 25 (50%) patients were smokers (Table 2).

Regarding ECG rhythm, 45 (90%) patients were presented by sinus rhythm, and five (10%) patients were presented by atrial fibrillation (Table 2).

Regarding ECG findings, 34 (68%) patients presented with acute STEMI, and 16 (32%) patients presented with NSTEMI. The ST-segment elevation magnitude was ~2 mm in eight (16%) patients, ~3 mm in seven (14%) patients, 4 mm in 11 (22%) patients, and ~5 mm in two (4%) patients.

Regarding the angiographic data and number of vessels affected, 25 (50%) patients had only LAD artery affected, and 25 (50%) patients had multivessel disease (Table 3).

Regarding the preprocedural TIMI flow, 31 patients had a pre-TIMI flow 0 (62%), 19 (38%) patients had a pre-TIMI flow I, 0 (0%) patients had a pre-TIMI flow II, and 0 (0%) patients

had a pre-TIMI flow III. Regarding the postprocedural TIMI flow, two (4%) patients had post-TIMI flow I, 14 (28%) patients had post-TIMI flow II, and 32 (64%) patients has post-TIMI flow III (Table 4).

Regarding echocardiographic LV systolic function, 34 (68%) patients had preprocedural LVEF less than 45, and 16 (32%) patients had LVEF more than 45. After 3 months of primary PCI, 23 (46%) patients had LVEF less than 45 and 27 (54%) patients had LVEF more than 45 (Table 4).

Regarding age, blood pressure, and heart rate, there was no statistically significant difference among patients with LV systolic function less than 45 and those with LV systolic function more than 45 (Table 5).

Regarding the sex and risk factors, there was no statistically significant difference among patients with LV systolic function less than 45 and those with LV systolic function more than 45 (Table 6).

Regarding STEMI and NSTEMI patients, there was no statistically significant difference in patients with LV dysfunction before and after primary PCI (Table 7). As for

### Table 1: Demographic and clinical feature data distribution of the study group

|  | Mean | SD |
|---|---|---|
| Age | 57.2 | 12.7 |
| Systolic blood pressure | 133.0 | 33.2 |
| Diastolic blood pressure | 75.5 | 14.7 |
| Heart rate | 93.0 | 18.9 |

### Table 2: Risk factor distribution of the study group

|  | n (%) |
|---|---|
| Sex |  |
| Male | 26 (52.0) |
| Female | 24 (48.0) |
| Smoking |  |
| No | 25 (50.0) |
| Yes | 25 (50.0) |
| HTN |  |
| No | 21 (42.0) |
| Yes | 29 (58.0) |
| DM |  |
| No | 16 (32.0) |
| Yes | 34 (68.0) |
| Dyslipidemia |  |
| No | 19 (38.0) |
| Yes | 31 (62.0) |

DM, diabetes mellitus; HTN, hypertension.

### Table 3: Clinical presentation and angiographic result distribution of the study group

|  | n (%) |
|---|---|
| STEMI |  |
| No | 16 (32.0) |
| Yes | 34 (68.0) |
| Non-STEMI |  |
| No | 34 (68.0) |
| Yes | 16 (32.0) |
| ST-segment magnitude |  |
| 1 | 22 (44.0) |
| 2 | 8 (16.0) |
| 3 | 7 (14.0) |
| 4 | 11 (22.0) |
| 5 | 2 (4.0) |
| Multivessel |  |
| No | 25 (50.0) |
| Yes | 25 (50.0) |

STEMI, ST-segment elevation myocardial infarction.

### Table 4: TIMI flow and echocardiographic characteristic distribution of the study group

|  | Before [n (%)] | After [n (%)] |
|---|---|---|
| TIMI flow |  |  |
| 0 | 31 (62.0) | 2 (4.0) |
| 1 | 19 (38.0) | 2 (4.0) |
| 2 | 0 | 14 (28.0) |
| 3 | 0 | 32 (64.0) |
| LV systolic function |  |  |
| <45 | 34 (68.0) | 23 (46.0) |
| >45 | 16 (32.0) | 27 (54.0) |

LV, left ventricular.

**Table 5: Comparison between left ventricular systolic function regarding age, blood pressure, and heart rate in the study group**

| | LV systolic function after | | | | t test | | |
| | <45 | | >45 | | | | |
| | Mean | SD | Mean | SD | t | P | Significance |
|---|---|---|---|---|---|---|---|
| Age | 54.7 | 15.4 | 59.3 | 9.6 | −1.28 | 0.205 | NS |
| Systolic blood pressure | 126.0 | 35.1 | 139.0 | 30.8 | −1.40 | 0.169 | NS |
| Diastolic blood pressure | 72.0 | 17.5 | 78.4 | 11.4 | −1.50 | 0.142 | NS |
| Heart rate | 88.7 | 23.2 | 96.7 | 13.8 | −1.45 | 0.157 | NS |

LV, left ventricular.

**Table 6: Comparison between left ventricular systolic function regarding sex, smoking, diabetes, and rhythm in the study group**

| | LV systolic function after | | | | $\chi^2$ test | | |
| | <45 | | >45 | | | | |
| | Mean | SD | Mean | SD | $\chi^2$ | P | Significance |
|---|---|---|---|---|---|---|---|
| Sex | | | | | | | |
| Male | 14 | 60.9 | 12 | 44.4 | 1.34 | 0.247 | NS |
| Female | 9 | 39.1 | 15 | 55.6 | | | |
| Smoking | | | | | | | |
| No | 12 | 52.2 | 13 | 48.1 | 0.08 | 0.777 | NS |
| Yes | 11 | 47.8 | 14 | 51.9 | | | |
| HTN | | | | | | | |
| No | 9 | 39.1 | 12 | 44.4 | 0.14 | 0.704 | NS |
| Yes | 14 | 60.9 | 15 | 55.6 | | | |
| DM | | | | | | | |
| No | 8 | 34.8 | 8 | 29.6 | 0.15 | 0.697 | NS |
| Yes | 15 | 65.2 | 19 | 70.4 | | | |
| Dyslipidemia | | | | | | | |
| No | 10 | 43.5 | 9 | 33.3 | 0.54 | 0.461 | NS |
| Yes | 13 | 56.5 | 18 | 66.7 | | | |
| Rhythm | | | | | | | |
| Sinus | 21 | 91.3 | 24 | 88.9 | 0.08 | 1.000 | NS |
| AF | 2 | 8.7 | 3 | 11.1 | | | |

AF, atrial fibrillation; DM, diabetes mellitus; HTN, hypertension; LV, left ventricular.

patient rhythm, there was also no statistically significant difference (Table 6).

In patients with multivessel coronary artery disease, there was a statistically significant difference in prediction of LV systolic dysfunction as compared by single-vessel disease as well as magnitude of ST-segment elevation. Patients with LVEF less than 45 before MI had also LV systolic dysfunction after primary PCI. In patients with TIMI flow less than III after PCI, there was a statistically significant difference in patient with LVEF less than 45 after primary PCI (Tables 7 and 8).

## DISCUSSION

AMI is usually caused by a rupture or erosion of the plaque within the coronary artery, which can lead to many complications. Approximately 30–40% of patients with STEMI experience decreased LVEF or cardiac dysfunction [8].

The present study showed no statistically significant difference between the two groups regarding the age, sex, blood pressure, diabetes mellitus, smoking, and dyslipidemia. This was in concordance with Hoffmayer et al. [9].

For age, our study was not in concordance with Rybicki et al. [10], who stated that older age may predict higher rates of depressed LV function in patients with STEMI. This discordance could be explained by larger sample volume included in their study, higher mean age, and different modalities of revascularization.

Regarding female sex, our result was not in concordance with Topilsky et al. [11], who made a comparison of LV systolic function after STEMI treated with primary PCI and concluded that female patients demonstrated lower systolic LV function, despite receiving similar treatment as male patients. This discrepancy could be related to higher ages of females in their study and other comorbidities.

## Table 7: Comparison between left ventricular systolic function regarding angiographic findings in the study group

| | LV systolic function after [n (%)] | | Test of significance | | |
| --- | --- | --- | --- | --- | --- |
| | <45 | >45 | Value of significace | P | Significance |
| STEMI | | | | | |
| Non-STEMI | 6 (26.1) | 10 (37.0) | $\chi^2$=0.68 | 0.408 | NS |
| STEMI | 17 (73.9) | 17 (63.0) | | | |
| ST-segment magnitude | | | | | |
| 1 | 9 (39.1) | 13 (48.1) | Fisher exact=1.05 | 0.04 | S |
| 2 | 4 (17.4) | 4 (14.8) | | | |
| 3 | 3 (13.0) | 4 (14.8) | | | |
| 4 | 6 (26.1) | 5 (18.5) | | | |
| 5 | 1 (4.3) | 1 (3.7) | | | |
| Multivessel | | | | | |
| No | 8 (34.8) | 17 (63) | $\chi^2$=3.95 | 0.047 | S |
| Yes | 15 (65.2) | 10 (37) | | | |
| TIMI flow before | | | | | |
| 0 | 16 (69.6) | 15 (55.6) | $\chi^2$=1.04 | 0.309 | NS |
| 1 | 7 (30.4) | 12 (44.4) | | | |
| TIMI flow after | | | | | |
| 0 | 1 (4.3) | 1 (3.7) | Fisher exact=8.54 | 0.017 | S |
| 1 | 2 (8.7) | 0 | | | |
| 2 | 10 (43.5) | 4 (14.8) | | | |
| 3 | 10 (43.5) | 22 (81.5) | | | |

LV, left ventricular; S, significant; STEMI, ST-segment elevation myocardial infarction.

## Table 8: Relation between left ventricular systolic function before and after primary percutaneous coronary intervention

| | LV systolic function after | | Total | McNemar test | |
| --- | --- | --- | --- | --- | --- |
| | <45 | >45 | | P | Significance |
| LV systolic function before | | | | | |
| <45 | 16 (32) | 0 | 16 (32) | 0.016 | S |
| >45 | 7 (14) | 27 (54) | 34 (68) | | |
| Total | 23 (46) | 27 (54) | 50 (100) | | |

LV, left ventricular; S, significant.

There was no statistically significant increased incidence of LV systolic dysfunction after PCI among patient with DM than those without DM. This was not in concordance with von Bibra and Sutton [12], which may be related to the decreased myocardial energy supply, endothelial dysfunction, and oxidative stress; however, this may be attributed to the small number of patients involved in this study.

Regarding ST-segment elevation, there was a significantly increased statistical difference for LV systolic dysfunction among patient with ST-segment elevation more than 2 mm. This finding was in concordance with the study of Hoffmayer et al. [9], who found that among patients with ST elevation MI, ST-segment elevation more than 2 mm was found to be an independent predictor of depressed LV function. This goes with the fact that the magnitude of ST-segment elevations may be a surrogate for the size of the myocardial territory being injured, so it is a fact that greater height of ST-segment elevations correlates with ventricular depressed function.

In patients with multivessel coronary artery disease, there was a significantly increased incidence of LV systolic dysfunction than in patients with single-vessel disease. This was in concordance with Yee et al. [13], who found greater incidence in LV dysfunction among patients undergoing PCI and with multivessel disease.

We found that the no-reflow phenomenon represents the myocardial status of the post-AMI PCI, resulting in a significant decrease in LV systolic function with adverse long-term outcomes in patients with AMI. This was in concordance with Wang et al. [14].

### Limitations
Our research has obvious drawbacks, mainly related to the relatively small sample volume and the possible effect on statistical analysis, despite the fact that the substantial level of PCI-based improvement in segmental and regional ventricular function indicates it is simple, regardless of the sample size.

## CONCLUSION
Our findings may help in the management of patients with STEMI in the early stages of their presentations. To recognize

patients who are likely to have more harm after primary PCI, a high prediction score for LV dysfunction may be needed. In addition, high-risk scores may serve as a warning to providers that certain patients require closer monitoring for congestive heart failure development and arrhythmias. Such patients may also need to be repeatedly assessed for select postinfarction therapies, such as eplerenone or an implantable cardioverter-defibrillator.

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### Conflicts of interest

There are no conflicts of interest.

## References

1. Jernberg T, Johanson P, Held C, Svennblad B, Lindbäck J, Wallentin L, SWEDEHEART/RIKS-HIA. Association between adoption of evidence-based treatment and survival for patients with ST-elevation myocardial infarction. JAMA 2011; 305:1677–1684.
2. Tomas Jernberg CH, Rydberg E, Hambraeus K. SWEDEHEART Annual report 2014. 2015. World Health Organisation. Cardiovascular diseases (CVDs) Fact sheet No 317 2015; 2016.
3. Baek JY, Seo SM, Park HJ, Kim PJ, Park MW, Koh YS, et al. Clinical outcomes and predictors of unprotected left main stem culprit lesions in patients with acute ST segment elevation myocardial infarction. Catheter Cardiovasc Interv 2014; 83:E243–E250.
4. Califf RM, Armstrong PW, Carver JR, D'Agostino R, Strauss WE. Task force 5. Stratification of patients into high, medium and low risk subgroups for purposes of risk factor management. J Am Coll Cardiol 1996; 27:1007–1019.
5. Gibson CM, Cannon CP, Murphy SA, Ryan KA, Mesley R, Marble SJ, et al. Relationship of TIMI myocardial perfusion grade to mortality after administration of thrombolytic drugs. Circulation 2000; 101:125–130.
6. Mendis S, Thygesen K, Writing Group on Behalf of the Participating Experts of the WHO Consultation for Revision of WHO Definition of Myocardial Infarction. World Health Organization definition of myocardial infarction. Int J Epidemiol 2011; 40:139–146.
7. Lakhal K, Ehrmann S, Chaari A, Laissy JP, Regnier B, Wolff M, Pajot O. Acute Kidney Injury Network definition of contrast-induced nephropathy in the critically ill: incidence and outcome. J Crit Care 2011; 6:593–599.
8. Campo G, Saia F, Guastaroba P, Marchesini J, Varani E, Manari A, et al. Prognostic impact of hospital readmissions after primary percutaneous coronary intervention. Arch Intern Med 2011; 171:1948–1949.
9. Hoffmayer KS, Armstrong EJ, Garg S, Patel A, Macgregor JS, Stein JC, et al. Predictors of depressed left ventricular function in patients presenting with ST-elevation myocardial infarction. Am J Cardiol 2012; 109:327–331.
10. Rybicki BA, Alam M, Wulbrecht N, Richer-Cornish K, Khaja F, Sabbah HN, Goldstein S. Clinical predictors of heart failure in patients with first acute myocardial infarction. Am Heart J 1999; 138:1133–1139.
11. Topilsky Y, Leshem-Rubinow E, Laufer-Perl M, Keren G, Roth A, Steinvil A, Arbel Y. Comparison of left ventricular function following first ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention in men versus women. Am J Cardiol 2014; 113:1941–1946.
12. von Bibra H, Sutton MS. Impact of diabetes on postinfarction heart failure and left ventricular remodeling. Curr Heart Fail Rep 2011; 8:242–251.
13. Yee NP, Siu AM, Davis J, Kao J. Recovery of left ventricular function after percutaneous coronary intervention compared to coronary artery bypass grafting in patients with multi-vessel coronary disease and left ventricular dysfunction. Hawaii J Med Public Health 2016; 75:273–277.
14. Wang L, Liu G, Liu J, Zheng M, Li L. Effects of no-reflow phenomenon on ventricular systolic synchrony in patients with acute anterior myocardial infarction after percutaneous coronary intervention. Ther Clin Risk Manag 2016; 12:1017–1022.