Original Article

Comparison of In-Hospital Mortality between Acute Inferior Wall STEMI Patients with and without Right Ventricular Infarction Undergoing Primary PCI

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Background: Patients with acute inferior wall ST elevation myocardial infarction (STEMI) with right ventricular infarction (RVI) involvement have a worse prognosis. However, most patients in the previous studies were treated with intravenous fibrinolysis and they were in the Caucasian populations.

Objective: To compare the in-hospital mortality rate of patients with acute inferior wall STEMI with and without RVI treated with primary percutaneous coronary intervention (PPCI).

Materials and Methods: The present study was a retrospective descriptive study that enrolled patients with acute inferior wall STEMI treated with PPCI in King Chulalongkorn Memorial Hospital between January 1, 2007, and December 31, 2016.

Results: Among 452 acute inferior wall STEMI patients treated with PPCI, 99 patients had RVI. The in-hospital mortality rate was 23.2% compared with 5.1% in patients without RVI (p<0.001). Patients with RVI had a significantly higher incidence of cardiogenic shock at 48.5% versus 15.6% (p<0.001), lower left ventricle ejection fraction at 51.15±17.27% versus 55.79±12.46% (p=0.037), higher incidence of complete heart block at 33.3% versus 11.9% (p<0.001), and ventricular tachycardia at 15.2% versus 5.9% (p=0.003). After adjustment for age, female gender, cardiogenic shock on admission, left ventricular ejection fraction (LVEF), ventricular tachycardia, and complete heart block, the RVI had a tendency to be the poor predictor for in-hospital death (adjusted HR 1.96; 95% CI 0.73 to 5.23; p=0.18). Nevertheless, the RVI was the significant independent predictor for 1-year mortality (adjusted HR 2.12; 95% CI 1.03 to 4.36; p=0.041).

Conclusion: Patients with acute inferior wall STEMI and RVI treated with PPCI had higher in-hospital mortality compared to no RVI. The RVI tended to have worse outcomes than those without RVI.

Keywords: Acute inferior wall STEMI; Right ventricular infarction; Mortality

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Patients with isolated right ventricular infarction (RVI) were rare. Most common was among the patients with acute inferior wall ST elevation myocardial infarction (STEMI). The RVI was occurred about 30 to 50% in these patients[1-3]. The patients with acute inferior wall STEMI, if there was RVI involvement, would associate with higher in-hospital morbidity and mortality compared to patients without RVI[4]. Furthermore, RVI increased the risk of death in patients with acute STEMI undergoing primary percutaneous coronary intervention (PPCI). Zehender et al[5] also found that among the patients with acute inferior wall STEMI, the in-hospital death was 19%. In addition, if there was right ventricular involvement, the in-hospital death was up to 31% compared to six percent of whom with no involvement, which was statistically significant.

Goldstein et al[6] reported on the patients with acute inferior wall STEMI undergoing PPCI. There was right ventricular involvement about 55%. Theses group had worse hemodynamic status (as they needed inotropes or vasopressors in 39 versus 15% p<0.0001, and intra-aortic balloon pump (IABP) in 32 versus

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13% (p<0.0001). In addition, the in-hospital mortality rate was higher at 14 versus three percent (p=0.0006).

Among patients with acute inferior wall STEMI who had reserved or slightly impaired LV systolic function, right ventricular systolic function was the only predictor associated with poor prognosis for death or hospitalization for cardiac problems at follow-up time for five years (odds ratio [OR] 0.56, 95% confidence interval [CI] 0.34 to 0.92, p=0.02)[7]. The cardiac problems were unstable angina, myocardial infarction, percutaneous coronary intervention (PCI), coronary artery bypass graft, decompensated heart failure, or pacemaker implantation. The positive predictive value (PPV), negative predictive value (NPV), sensitivity, and specificity for RV systolic myocardial velocity of less than 13 cm per second before hospital discharge were 44, 83, 80, and 49%, respectively, and the AUC was 0.7.

The aim of the present study was to compare the in-hospital mortality rate of patients with acute inferior wall STEMI with and without RVI involvement treated with PPCI in Thai population.

Material and Method

Study design

The present study was a retrospective descriptive study conducted in King Chulalongkorn Memorial Hospital, Bangkok Thailand. The protocol for the present study was approved by the Med Chula Institutional Review Board (Med Chula IRB No. 097/61).

Study protocol

Patients with acute inferior wall STEMI treated with PPCI in Chulalongkorn Hospital between January 1, 2007, and December 31, 2016 were enrolled. The studied patients were collected from the database "PCI in Acute MI registry".

Inclusion criteria were patients aged 18 or above with diagnosis of acute inferior wall STEMI treated with PPCI.

Exclusion criteria were patients treated with thrombolysis, prior history of RVI, history of stenting at right coronary artery with in-stent restenosis (ISR) or stent thrombosis, mechanical complications due to myocardial infarction, and prior coronary artery bypass surgery (CABG).

The definition of RVI was patient recognized as clinical triad of hypotension, elevated jugular venous pressure (JVP), and clear lung fields or ST segment elevation of 0.1 mV or more in the right precordial leads, V3R or V4R of the 12 leads electrocardiography, echocardiogram demonstrated right ventricular dilatation, or impaired right ventricular systolic function[3-5,8-12].

Data collection

Data from patient's medical records included baseline clinical characteristics such as gender and age, cardiovascular risk factors such as diabetes mellitus, hypertension, hypercholesterolemia, and smoking history, Killip classification I-IV, systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, complete atrioventricular block and ventricular tachycardia or ventricular fibrillation, symptom-to-balloon time (minute), door-to-balloon time (minute), temporary pacemaker and IABP, ST segment elevation in V3R or V4R of the 12 lead electrocardiography and echocardiographic findings such as left ventricular ejection fraction (LVEF) and tricuspid annular plane systolic excursion (TAPSE), chemical profiles including creatinine, total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol, hs-Trop I, and CK-MB.

The primary endpoint of the present study was to compare the in-hospital mortality rate between acute inferior wall STEMI patients with and without RVI underwent a PPCI.

Secondary endpoint of the present study was to compare the 1-year mortality and the other prognostic factors between acute inferior wall STEMI patients with and without RVI underwent a PPCI.

Statistical analysis

Categorical data were reported as frequency and percentage. Continuous variables were reported as mean ± standard deviation when there was normal distribution or median (minimum, maximum) when there was skewed distribution.

Categorical data were compared using chi-square test or Fisher's exact test and continuous data were compared using Student's t-test or Mann-Whitney U test according to data distribution.

For relationship between baseline characteristics and the in-hospital and 1-year mortality were analyzed by Cox regression and presented with hazard ratio (HR) and 95% CI.

All statistical analyses were performed using IBM SPSS Statistics, version 22.0 (IBM Corp., Armonk, NY, USA).

Results

Between January 1, 2007, and December 31, 2016, 496 consecutive patients with acute inferior

wall STEMI were retrospectively identified. Forty-four patients (44/496, 8.87%) were excluded from the study, including 35 with prior thrombolytic treatment, five with history of stenting at right coronary artery with ISR or stent thrombosis, and four with prior CABG.

Four hundred fifty-two acute inferior wall STEMI treated with PPCI were analyzed in the present study. Among these, there were 99 patients (21.9%) with RVI. The average age in RVI group was higher than no RVI group at 64.63±15.07 versus 59.92±12.82 years (p=0.005). Male gender was predominant in both groups but significantly higher in no RVI group at 70.7% versus 80.2% (p=0.044). The LVEF in both groups was above 50% but also significantly higher in no RVI group at 51.15±17.27% versus 55.79±12.46% (p=0.037). The medical history such as diabetes mellitus, hypertension, history of smoking, hypercholesterolemia, and previous myocardial infarction were not different in both groups. However, the in-hospital death of RVI group was significantly higher than the non RVI group at 23.2% versus 5.1% (p<0.001). In addition, the 1-year mortality was still significantly higher in the RVI group compared to those without RVI at 29.3% versus 8.5% (p<0.001). The other complications such as complete heart block, ventricular tachycardia (VT), and presentation with Killip class 4 were significantly higher in RVI group for complete heart block at 33.3% versus 11.9% (p<0.001), ventricular tachycardia at 15.2% versus 5.9% (p=0.003), and Killip class 4 at 48.5% versus 15.6% (p<0.001) (Table 1).

Using univariate analysis, RVI, age, female, cardiogenic shock, complete heart block, VT, cardiopulmonary resuscitation (CPR) prior PCI, Killip class 4, and chemical profiles such as higher value of CK-MB, white blood cell count, and fasting blood glucose were the strong predictors for in-hospital death (Table 2). The usage of invasive procedures such as Swan Ganz catheter, pacemaker, and IABP were also the predictor of in-hospital death (Swan Ganz catheter: HR 5.32, 95% CI 2.09 to 13.58, p<0.001, pacemaker: HR 4.09, 95% CI 2.19 to 7.61, p<0.001, and IABP: HR 9.54, 95% CI 5.11 to 17.79, p<0.001). On the contrary, higher value of echocardiographic findings such as LVEF, TAPSE, systolic and DBP as clinical presentations were the good prognostic factors for in-hospital death (LVEF: HR 0.94, 95% CI 0.91 to 0.96, p<0.001; TAPSE: HR 0.79, 95% CI 0.71 to 0.87, p<0.001; SBP: HR 0.97, 95% CI 0.96 to 0.98, p<0.001; and DBP: HR 0.96, 95% CI 0.94 to 0.97, p<0.001).

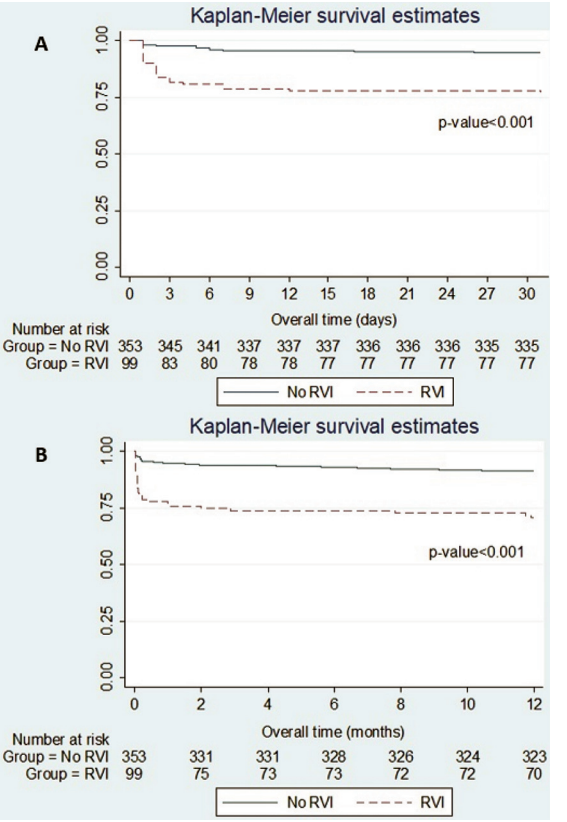

Figure 1. Kaplan-Meier survival curve for in-hospital mortality (A) and 1-year mortality (B) of ST elevation inferior myocardial infarction patients with and without right ventricular infarct.

Univariate cox regression analysis for 1-year mortality was also demonstrated (Table 2). The RVI, age, female gender, cardiogenic shock, VT, CPR prior PCI, and Killip class 4 were still the key factors to predict 1-year mortality except complete heart block. HR for RVI was 3.93 (95% CI 2.36 to 6.55), p<0.001. Kaplan Meier survival analysis demonstrated the worse outcomes of the patients who had inferior STEMI complicated with RVI (Figure 1).

Independent predictors for in-hospital death and 1-year mortality after multivariate cox regression analysis from selected prognostic factors such as RVI, age, female gender, LVEF, complete heart block, VT, and Killip class 4 were demonstrated (Table 3).

The RVI was still the significantly independent predictor for 1-year mortality (adjusted HR 2.12, 95% CI 1.03 to 4.36, p=0.041). In addition, age, VT, and Killip class 4 were significantly prognostic factors for both in-hospital death and 1-year mortality. The significantly good prognostic factor for both in-hospital and 1-year mortality was only LVEF

Table 1. Demographic data of the patients

Variable	RVI (n=99)	No RVI (n=353)	p-value
Age (year); mean±SD	64.63±15.07	59.92±12.82	0.005*
Sex: male; n (%)	70 (70.7)	283 (80.2)	0.044*
Cardiovascular risk factors; n (%)			
Diabetes mellitus	33 (33.3)	107 (30.3)	0.566
Hypertension	53 (53.5)	177 (50.1)	0.551
Smoking	42 (42.4)	158 (44.8)	0.679
Hypercholesterolemia	65 (65.7)	250 (70.8)	0.323
Family history of ischemic heart disease	1 (1.0)	16 (4.5)	0.104
Clinical presentations; n (%)			
Systolic blood pressure (mmHg); mean±SD	93.21±25.16	120.79±26.95	<0.001*
Diastolic blood pressure (mmHg); mean±SD	57.48±16.84	71.09±15.1	<0.001*
Heart rate (beats/minute); mean±SD	68.07±22.63	72.18±20.09	0.105
Complete heart block	33 (33.3)	42 (11.9)	<0.001*
Ventricular tachycardia	15 (15.2)	21 (5.9)	0.003*
CPR prior PCI	17 (17.2)	19 (5.4)	<0.001*
Previous myocardial infarction	4 (4.0)	20 (5.7)	0.524
Killip classification; n (%)			
1	41 (41.4)	262 (74.2)	<0.001*
2	5 (5.1)	25 (7.1)	0.473
3	5 (5.1)	10 (2.8)	0.276
4	48 (48.5)	55 (15.6)	<0.001*
Chemical profiles; median (min, max)			
Creatinine kinase (U/L)	2,628.5 (1,114.5, 3,895)	1,689 (756, 3,170)	0.003*
hsTroponinI (ng/L)	43,976 (7,351, 50,000)	14,584 (2,138, 50,000)	0.069
CK-MB (ng/mL)	290.5 (153.5, 539.5)	194 (81, 347)	<0.001*
Creatinine (mg/dL)	1.23 (0.9, 2.04)	0.98 (0.83, 1.3)	0.001*
Fasting blood glucose (mg/dL)	151 (113, 200)	127 (104, 173)	0.067
HbA1c (%)	5.9 (5.6, 7.3)	5.9 (5.5, 7.5)	0.516
Hematocrit (%)	39 (36, 42)	41 (36, 44)	0.250
White blood cell count ($\times 10^3/\mu L$)	13,290 (10,850, 16,330)	11,760 (9,190, 14,225)	0.004*
Cholesterol (mg/dL); mean±SD	176.12±50.95	198.26±50.77	0.002*
Triglyceride (mg/dL); mean±SD	118.8±56.42	158.81±149.8	<0.001*
HDL-cholesterol (mg/dL); mean±SD	41.83±13.79	41.17±20.39	0.801
LDL-cholesterol (mg/dL); mean±SD	114.89±48.01	129.38±42.91	0.016*
Door to balloon time (minute); median (min, max)	31 (20, 79)	34 (20, 65)	0.294
≤60 minute; n (%)	68 (68.7)	250 (70.8)	0.681
≤90 minute; n (%)	81 (81.8)	301 (85.3)	0.402
Pain to FMC time (minute); median (min, max)	107 (50, 210)	130 (60, 230)	0.262
FMC to balloon time (minute); median (min, max)	205 (122, 326)	162.5 (110, 255)	0.104
≤120 minute; n (%)	8 (8.1)	49 (13.9)	0.125
Pain to balloon time <6 hours; n (%)	41 (41.4)	113 (32)	0.081
Refer; n (%)	55 (55.6)	176 (49.9)	0.316
Refer time (minutes); mean±SD	76.14±52.33	64.94±36.83	0.089
Infarct related artery; n (%)			
Proximal RCA	65 (65.7)	93 (26.3)	<0.001*
Mid RCA	28 (28.3)	126 (35.7)	0.169
Distal RCA	6 (6.1)	78 (22.1)	<0.001*
Others	0 (0.0)	56 (15.9)	N/A

RVI=right ventricular infarct; CPR=cardiopulmonary resuscitation; PCI=percutaneous coronary intervention; FMC=first medical contact; RCA=right coronary artery; LVEF=left ventricular ejection fraction; TAPSE=tricuspid annular plane systolic excursion; IABP=intra-aortic balloon pump

Table 1. (continued)

Variable	RVI (n=99)	No RVI (n=353)	p-value
Initial TIMI flow grade; n (%)	0.156		
0-1	82 (82.8)	262 (74.4)	
2	11 (11.1)	47 (13.4)	
3	6 (6.1)	43 (12.2)	
Final TIMI flow grade; n (%)	0.017*		
0-1	7 (7.1)	6 (1.7)	
2	11 (11.1)	35 (9.9)	
3	81 (81.8)	311 (88.4)	
Number of diseased vessels; n (%)			
1	29 (29.3)	136 (38.5)	0.092
2	23 (23.2)	107 (30.3)	0.169
3	33 (33.3)	93 (26.3)	0.171
Thrombectomy; n (%)	77 (77.8)	255 (72.2)	0.270
Echocardiographic findings; mean±SD			
LVEF (%)	51.15 ± 17.27	55.79 ± 12.46	0.037*
TAPSE (mm)	11.64 ± 3.63	20.59 ± 4.01	<0.001*
The 12-lead echocardiography; n (%)			
Elevated V3r	41 (41.4)	19 (5.4)	<0.001*
Elevated V4r	42 (42.4)	18 (5.1)	<0.001*
Medications used; n (%)			
Nitroglycerine	1 (1.0)	34 (9.6)	0.005*
Aspirin	99 (100)	352 (99.7)	0.596
Heparin	93 (93.9)	343 (97.2)	0.125
Integrilin	80 (80.8)	285 (80.7)	0.987
Dopamine	62 (62.6)	86 (24.4)	<0.001*
Dobutamine	10 (10.1)	6 (1.7)	<0.001*
Adranaline	32 (32.3)	27 (7.6)	<0.001*
Betablocker	26 (26.3)	154 (43.6)	0.002*
Angiotensin converting enzyme inhibitor	33 (33.3)	174 (49.3)	0.005*
Statin	72 (72.7)	328 (92.9)	<0.001*
Calcium channel blocker	3 (3.0)	6 (1.7)	0.402
Digoxin	1 (1.0)	2 (0.6)	0.631
Antiarrhythmic agents	2 (2.0)	5 (1.4)	0.667
Invasive procedures; n (%)			
Swan Ganz catheter	9 (9.1)	4 (1.1)	<0.001*
Pacemaker	36 (36.4)	36 (10.2)	<0.001*
IABP	33 (33.3)	34 (9.6)	<0.001*
In-hospital mortality; n (%)	23 (23.2)	18 (5.1)	<0.001*
1-year mortality; n (%)	29 (29.3)	30 (8.5)	<0.001*

RVI=right ventricular infarct; CPR=cardiopulmonary resuscitation; PCI=percutaneous coronary intervention; FMC=first medical contact; RCA=right coronary artery; LVEF=left ventricular ejection fraction; TAPSE=tricuspid annular plane systolic excursion; IABP=intra-aortic balloon pump

(adjusted HR 0.95, 95% CI 0.92 to 0.98, p<0.001) (Table 3).

Discussion

Most of acute inferior wall STEMI resulted from an occlusion of the right coronary artery. Although, the occlusion was proximal to the acute marginal artery arteries, there was no evidence of RVI in a half of patients[3]. The present study demonstrated that right coronary artery occlusion was the major vessel of acute inferior wall STEMI as high as 93% (n=422/452), and only 41% of proximal RCA occlusion were involved with RVI. There was 6% of distal RCA occlusion that developed RV infarction

Table 2. Univariate: cox regression analysis for in-hospital death and 1-year mortality

	In hospital death		1-year mortality	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Right ventricular infarction	4.98 (2.69 to 9.24)	<0.001*	3.93 (2.36 to 6.55)	<0.001*
Age	1.05 (1.02 to 1.07)	<0.001*	1.05 (1.03 to 1.07)	<0.001*
Female	1.92 (1.01 to 3.67)	0.047*	2.08 (1.22 to 3.54)	0.007*
Cardiovascular risk factors				
Diabetes mellitus	1.65 (0.88 to 3.09)	0.117	1.41 (0.83 to 2.41)	0.206
Hypertension	1.8 (0.94 to 3.45)	0.076	1.6 (0.94 to 2.72)	0.082
Smoking	0.59 (0.3 to 1.15)	0.12	0.54 (0.31 to 0.94)	0.03*
Hypercholesterolemia	0.89 (0.45 to 1.76)	0.737	0.98 (0.55 to 1.75)	0.953
Family history of CAD	0.76 (0.1 to 5.63)	0.79	0.46 (0.06 to 3.37)	0.447
Clinical presentations				
Systolic blood pressure	0.97 (0.96 to 0.98)	<0.001*	0.98 (0.97 to 0.99)	<0.001*
Diastolic blood pressure	0.96 (0.94 to 0.97)	<0.001*	0.96 (0.94 to 0.98)	<0.001*
Heart rate	1.01 (1 to 1.02)	0.175	1.01 (1 to 1.02)	0.038*
Complete heart block	2.47 (1.28 to 4.76)	0.007*	1.68 (0.92 to 3.05)	0.092
Ventricular tachycardia	5.38 (2.74 to 10.55)	<0.001*	3.89 (2.1 to 7.2)	<0.001*
CPR prior PCI	7.71 (4.07 to 14.58)	<0.001*	5.4 (3.03 to 9.6)	<0.001*
Previous myocardial infarction	0.42 (0.06 to 3.07)	0.395	0.29 (0.04 to 2.07)	0.216
Killip class 4	9.42 (4.8 to 18.47)	<0.001*	7.05 (4.15 to 11.96)	<0.001*
Chemical profiles				
Creatinine kinase	1.01 (1.01 to 1.02)	0.043*	1.01 (1.01 to 1.02)	0.029*
hsTroponinI	1 (0.99 to 1)	0.779	1 (0.99 to 1)	0.328
CK-MB	1.01 (1.01 to 1.02)	<0.001*	1.01 (1.01 to 1.02)	<0.001*
Creatinine	1 (0.98 to 1.02)	0.947	1 (0.98 to 1.02)	0.988
Fasting blood sugar	1.01 (1.01 to 1.02)	<0.001*	1.01 (1 to 1.01)	0.009*
HbA1c	1.13 (0.93 to 1.37)	0.211	1.01 (0.84 to 1.22)	0.903
Hematocrit	0.99 (0.92 to 1.05)	0.648	0.95 (0.9 to 1.01)	0.078
White blood cell count	1.01 (1.01 to 1.02)	<0.001*	1.01 (1 to 1.021)	<0.001*
Cholesterol	0.99 (0.98 to 1)	0.07	0.99 (0.99 to 1)	0.054
Triglyceride	1 (0.99 to 1)	0.303	1 (0.99 to 1)	0.178
HDL-cholesterol	1 (0.99 to 1.02)	0.498	1 (0.99 to 1.02)	0.694
LDL-cholesterol	0.99 (0.98 to 1)	0.157	0.99 (0.99 to 1)	0.195
Echocardiographic findings				
LVEF	0.94 (0.91 to 0.96)	<0.001*	0.95 (0.93 to 0.97)	<0.001*
TAPSE	0.79 (0.71 to 0.87)	<0.001*	0.84 (0.77 to 0.92)	<0.001*
Invasive procedures				
Swan Ganz catheter	5.32 (2.09 to 13.58)	<0.001*	4.7 (2.02 to 10.94)	<0.001*
Pacemaker	4.09 (2.19 to 7.61)	<0.001*	3.31 (1.94 to 5.63)	<0.001*
IABP	9.54 (5.11 to 17.79)	<0.001*	6.15 (3.68 to 10.28)	<0.001*

CAD=coronary artery disease; CPR=cardiopulmonary resuscitation; PCI=percutaneous coronary intervention; LVEF=left ventricular ejection fraction; TAPSE=tricuspid annular plane systolic excursion; IABP=intra-aortic balloon pump; HR=hazard ratio; CI=confidence interval

Table 3. Multivariate: cox regression analysis for in-hospital death and 1-year mortality

	In hospital death		1-year mortality	
	Adjusted HR (95%CI)	p-value	Adjusted HR (95%CI)	p-value
Right ventricular infarction	1.96 (0.73 to 5.23)	0.180	2.12 (1.03 to 4.36)	0.041*
Age	1.05 (1.01 to 1.09)	0.016*	1.04 (1.02 to 1.07)	0.002*
Ventricular tachycardia	5.27 (1.87 to 14.85)	0.002*	3.93 (1.64 to 9.45)	0.002*
Killip class 4	4.91 (1.8 to 13.39)	0.002*	3.1 (1.51 to 6.37)	0.002*
LVEF	0.95 (0.92 to 0.98)	<0.001*	0.96 (0.94 to 0.98)	<0.001*

LVEF=left ventricular ejection fraction; HR=hazard ratio; CI=confidence interval

due to distal take off of RV branch.

As the previous studies[3,4], the in-hospital mortality was 31% in patients with acute inferior wall STEMI with RVI as compared with 6% for patients without right ventricular involvement. In addition, specific high-risk subgroups of patients, such as older patient of 75 years of age or older with RVI had an in-hospital mortality as high as 47% compared with 10% in patients without right ventricular involvement (p<0.001). In the present study, the result was not so different to the previous studies that the in-hospital mortality was 23.2%, as compared with 5.1%, respectively, (p<0.001). However, the lower number of in-hospital mortality in both groups from the present study, especially in patients with RVI might be due to all of them being complete revascularization with primary PCI and similar effective both door-to-balloon time of 60 minutes or less at 68.7% versus 70.8% (p=0.681) and 90 minutes or less at 81.8% versus 85.3% (p=0.402), and technique, such as thrombectomy at 77.8% versus 72.2% (p=0.27).

The present study demonstrated that patients with acute inferior wall STEMI with RVI related to major complications needed specific treatment and medications such as dopamine, dobutamine, adrenaline, and invasive procedures such as Swan Ganz catheter, pacemaker, and IABP more than those without right ventricular involvement. The major complications were cardiogenic shock at 48.5% versus 15.6% (p<0.001), cardiac arrest needed CPR before PCI at 17.2% versus 5.4% (p<0.001), complete heart block at 33.3% versus 11.9% (p<0.001), and ventricular tachycardia at 15.2% versus 5.9% (p=0.003). These results were similar to the previous studies[4,13-16]. A significantly higher incidence of cardiogenic shock was independent of LVEF, complete heart block, and ventricular tachycardia that induced a greater in-hospital mortality. In addition, Mehta et al[17]. proposed the meta-analysis of six studies that consisted of 1,198 patients. The results confirmed that RVI was the strong predictor for death, cardiogenic shock, ventricular tachycardia or fibrillation, and complete heart block (OR 3.2, 95% CI 2.4 to 4.1; OR 3.2, 95% CI 2.4 to 3.5; OR 2.7, 95% CI 2.1 to 3.5; and OR 3.4, 95% CI 2.7 to 4.2, respectively).

From the previous study[3], there was conflicting data about the RVI and long-term prognosis, as studies showed that it was an independent risk factor for long-term mortality, but others had not demonstrated any difference in long-term mortality. The present study demonstrated that long-term mortality at 1-year follow-up of patients with acute inferior wall STEMI with RVI was as high as 29.3%, compared with 8.5% for patients with no right ventricular involvement and the RVI was also an independent risk factors for 1-year mortality (adjusted HR 2.12, 95% CI 1.03 to 4.36, p=0.041).

In the present study demonstrated the in-hospital and 1-year mortality for patients with acute inferior wall STEMI who were with or without RVI. As compared with the previous study[18,19], it was able to assess the prognostic significance of the presence of RVI and explore the factors associated with the improved outcomes in patients that underwent primary PCI that is currently and practically used. Moreover, the raw data in the present study came from one of the largest PCI databases in Thailand.

Limitation

The present study was a single center retrospective study and registry-based. It had limitation in incomplete collection data including chemical profiles such as creatinine, total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol, hs-Trop I, and CK-MB, and echocardiographic findings such as LVEF and TAPSE, especially within the first five-year of data collections.

Conclusion

In-hospital death and 1-year mortality of patients with acute inferior wall STEMI and RVI involvement had worse outcomes than those without RVI involvement treated with primary PCI.

What is already known on this topic?

RVI associated with inferior wall STEMI is a well-known poor prognostic factors for patients presenting with acute myocardial infarction. Prompt treatments including reperfusion therapy and the invasive management are required.

What this study adds?

This study adds up the detail of demographic data and prognostic factors for in-hospital and one-year follow up for the patients who presented with inferior wall STEMI with RVI in Thailand.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size and topography in coronary

heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. J Am Coll Cardiol 1987;10:1223-32.
2. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease. Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol 1978;42:885-94.
3. Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med 1994;330:1211-7.
4. Bueno H, López-Palop R, Bermejo J, López-Sendón JL, Delcán JL. In-hospital outcome of elderly patients with acute inferior myocardial infarction and right ventricular involvement. Circulation 1997;96:436-41.
5. Zehender M, Kasper W, Kauder E, Schönthaler M, Geibel A, Olschewski M, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 1993;328:981-8.
6. Goldstein JA, Kommuri N, Dixon SR. Left ventricular systolic dysfunction is associated with adverse outcomes in acute right ventricular infarction. Coron Artery Dis 2016;27:277-86.
7. Smarz K, Zaborska B, Jaxa-Chamiec T, Tysarowski M, Budaj A. Right ventricular systolic function as a marker of prognosis after ST-elevation inferior myocardial infarction 5-year follow-up. Int J Cardiol 2016;221:549-53.
8. Kakouros N, Cokkinos DV. Right ventricular myocardial infarction: pathophysiology, diagnosis, and management. Postgrad Med J 2010;86:719-28.
9. Braat SH, Brugada P, de Zwaan C, Coenegracht JM, Wellens HJ. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. Br Heart J 1983;49:368-72.
10. Goldberger JJ, Himelman RB, Wolfe CL, Schiller NB. Right ventricular infarction: recognition and assessment of its hemodynamic significance by two-dimensional echocardiography. J Am Soc Echocardiogr 1991;4:140-6.
11. Kakouros N, Kakouros S, Lekakis J, Rizos I, Cokkinos D. Tissue Doppler imaging of the tricuspid annulus and myocardial performance index in the evaluation of right ventricular involvement in the acute and late phase of a first inferior myocardial infarction. Echocardiography 2011;28:311-9.
12. Jacobs AK, Leopold JA, Bates E, Mendes LA, Sleeper LA, White H, et al. Cardiogenic shock caused by right ventricular infarction: a report from the SHOCK registry. J Am Coll Cardiol 2003;41:1273-9.
13. Braat SH, de Zwaan C, Brugada P, Coenegracht JM, Wellens HJ. Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrioventricular nodal conduction disturbances. Am Heart J 1984;107:1183-7.
14. Mavrić Z, Zaputović L, Matana A, Kucić J, Roje J, Marinović D, et al. Prognostic significance of complete atrioventricular block in patients with acute inferior myocardial infarction with and without right ventricular involvement. Am Heart J 1990;119:823-8.
15. Ricci JM, Dukkipati SR, Pica MC, Haines DE, Goldstein JA. Malignant ventricular arrhythmias in patients with acute right ventricular infarction undergoing mechanical reperfusion. Am J Cardiol 2009;104:1678-83.
16. Brodie BR, Stuckey TD, Hansen C, Bradshaw BH, Downey WE, Pulsipher MW. Comparison of late survival in patients with cardiogenic shock due to right ventricular infarction versus left ventricular pump failure following primary percutaneous coronary intervention for ST-elevation acute myocardial infarction. Am J Cardiol 2007;99:431-5.
17. Mehta SR, Eikelboom JW, Natarajan MK, Diaz R, Yi C, Gibbons RJ, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. J Am Coll Cardiol 2001;37:37-43.
18. Antoni ML, Scherptong RW, Atary JZ, Boersma E, Holman ER, van der Wall EE, et al. Prognostic value of right ventricular function in patients after acute myocardial infarction treated with primary percutaneous coronary intervention. Circ Cardiovasc Imaging 2010;3:264-71.
19. Assali AR, Teplitsky I, Ben-Dor I, Solodky A, Brosh D, Battler A, et al. Prognostic importance of right ventricular infarction in an acute myocardial infarction cohort referred for contemporary percutaneous reperfusion therapy. Am Heart J 2007;153:231-7.