AKUT İNFERİOR MİYOKARD ENFARKTÜSLÜ HASTALARDA LEAD III >LEAD II ST-ELEVASYONUNUN SAĞ VENTRİKÜL ENFARKTÜSÜNÜ VE HASTANE İÇİ MORTALİTEYİ ÖNGÖRDÜRÜCÜ DEĞERİ

Predictive Value of Lead III >Lead II ST Elevation for Ventricular Infarction and Hospital Mortality Rate in Patients with Acute Inferior Myocardial Infarction

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Geliş tarihi/Received: 12.02.2016
Kabul tarihi/Accepted: 28.03.2016

Bozok Tıp Derg 2016;6(2):1-9
Bozok Med J 2016;6(2):1-9

ÖZET

Amaç: Bu çalışmanın amacı akut inferior miyokard enfarktüsü ile başvuran primer perkütanöz koroner girişim yapılan hastalarda lead III 'deki ST- Elevasyonun lead II'deki ST-elevasyonundan fazla olmasının sağ ventrikül miyokard enfarktüsü ve hastaneiçi mortalite yi öngördürmedeki değerini araştırdık.

Yöntem: Çalışmaya sağ koroner arterden kaynaklanan ve primer perkütanöz koroner girişime giden 180 akut inferior miyokard enfarktüsü hasta alındı. Sağ ventrikül miyokard enfarktüsü sağ taraflı çekilen EKG'de V4R'daki ST-elevasyonu olması ile tanımlandı. V4R'daki ST-elevasyonu olmayan hastalar sağ ventrikül miyokard enfarktüsü olmayan akut inferior miyokard enfarktüsü, V4R'da ST-elevasyonu olan hastalar sağ ventrikül miyokard enfarktüsü olan akut inferior miyokard enfarktüsü hastalar olarak iki gruba ayrıldı. lead III 'deki ST- elevasyonun lead II'deki ST-elevasyonundan yüksek olmasının sağ ventrikül enfarktüsü belirlemesi ve hastaneiçi mortaliteyi öngörmesine bakıldı.

Bulgu: Lead III>II ST-elevasyonu sağ ventrikül miyokard enfarktüsü olan hastalarda oranı daha yüksek izlendi ($p<0.001$). Yapılan multivariate regresyon analizinde, lead III>II ST-elevasyonunun sağ ventrikül miyokard enfarktüsü bağımsız öngördürücü olduğu izlendi (odds ratio :2.8,95% CI 1.55-5.25; $p=0.008$). Ancak, hastaneiçi mortalite üzerindeki öngördürücülüğü izlenmedi.

Sonuç: Sağ koroner arterden kaynaklanan akut inferior miyokard enfarktüslü primer perkütanöz koroner girişime giden hastalarda Lead III>II ST-elevasyonu sağ ventrikül miyokard enfarktüsünün bağımsız öngördürücüsüdür. Ancak hastaneiçi mortalite üzerine bir öngördücülüğü yoktur.

Anahtar kelimeler: *Sağ ventriküler miyokard enfarktüsü; ST-elevasyon; Perkütanöz koroner girişim*

ABSTRACT

Objectives: The aim of this study was to evaluate ST-elevation in lead III more than II (III>II) findings in predicting right ventricular infarction (RVI) and in-hospital mortality in patients with acute inferior myocardial infarction (AIMI) undergoing primary percutaneous coronary intervention (pPCI).

Methods: A total of 180 AIMI patients undergoing pPCI and right coronary artery (RCA) as infarct-related artery were included in the study. The presence of RVI was determined by ST-elevation in right side lead (V4R). Patients were divided into 2 groups: patients without ST-elevation in lead V4R (AIMI without RVI), and patients with ST-elevation in lead V4R (AIMI with RVI). We assessed the diagnostic accuracy of ST-elevation in lead III more than II to identify RVI and predicting in-hospital mortality.

Results: A large proportion of ST-elevation in lead III>II ($p=0.001$) were observed in patients with RVI. In a multivariate regression analysis, ST-elevation in lead III>II remained an independent predictor of RVI (odds ratio :2.8,95% CI 1.55-5.25; $p=0.008$). However , this predictive effect was not observed in-hospital mortality.

Conclusion: ST-elevation in lead III>II was an independent predictor of RVI in patients with RCA related inferior myocardial infarction undergoing pPCI. However, ST-elevation in lead III>II was not predictor of in-hospital mortality.

Key words: *Right ventricular infarction; ST-elevation; Percutaneous coronary intervention*

INTRODUCTION

Acute Inferior myocardial infarction (AIMI) is usually considered to have a better prognosis in the short term than anterior MI (1,2), but there are subgroups of AIMI associated with increased mortality. Right ventricular Infarction (RVI) occurs in 30% to 50% of patients with AIMI (3,4). Although AIMI generally has a favorable prognosis, the presence of right ventricular (RV) involvement is associated with increased in hospital adverse events and mortality. Several studies demonstrated that patients with AIMI involving RV have a poor prognosis and increased mortality rates in the pre-primary angioplasty era (5-8).

The diagnosis of RVI is often based on clinical findings in patients with AIMI. The main clinical characteristics of a hemodynamically deteriorating RVI consist of hypotension, clear lung fields and increased jugular venous pressure. On the other hand, hemodynamic deterioration may not be manifested among nearly 60% of patients with RVI and concurrent presence of this clinical triad has a sensitivity of 10 to 25% (6, 9-11). For this reason, among patients with AIMI suffering hypotension, an electrocardiographic diagnosis of concurrent RVI should be considered. The ECG of patients with RVI may reveal ST elevation of more than 1 mm in the right-sided precordial derivations V4R to V6R. ST elevation in right-sided leads, especially in V4R, indicates acute RV injury (6,12-14). Also, ST-elevation in lead III more than II (III>II) indicates acute RV injury (15, 16).

Although predictive value of ST elevation in lead III>II in patients with AIMI is satisfactory, their predictive value for RVI and in-hospital mortality in patients with AIMI undergoing pPCI has not been evaluated prospectively. In this study, we aimed to evaluate the roles of ST-elevation in lead III>II findings in predicting RVI and in-hospital mortality considering ST-elevation in lead V4R for RVI among patients with AIMI undergoing pPCI.

METHODS

Study population

This prospective study was conducted between February 2012 and May 2015. A total of 180 right coronary artery related AIMI patients presented within 6 hours from the symptom onset were included in the study. AIMI was defined as ST segment elevation of ≥1 mV in inferior leads. RVI was defined according to ECG criteria as the recommendations of ESC guideline (17). Patients were divided into 2 groups according to ECG criteria as the ST-elevation in lead V4R before perfusion : patients without ST-elevation in lead V4R (AIMI without RVI), and patients with ST-elevation in lead V4R (AIMI with RVI). Patient delay time is the delay between symptom onset and first medical contact (17). All patients underwent emergency cardiac catheterization. All patients received dual antiplatelet theraphy with aspirin and clopidogrel (600 mg) or ticagrelor (180 mg) loading dose. Preprocedural anticoagulation consisted of intravenous unfractionated heparin (70 IU/kg) in all cases. PPCI with stent implantation was performed according to current guidelines (18). In patients who were treated with tirofiban, the agent was administrated after pPCI in the coronary care unit. The systemic bolus of tirofiban was used according to operator's decision, and continued for the following 12 hours accordingly. Exclusion criteria were concurrent pericardial disease, left anterior fascicular block, previous RV dysfunction, previous heart failure (defined as previously measured left ventricular ejection fraction of <50%), chronic pulmonary disease, pulmonary hypertension, valvular heart disease (moderate to severe insufficiency and/or stenosis), acute pulmonary embolism, inferior myocardial infarction due to circumflex artery occlusion and acute anterior myocardial infarction (shown to be present in 10 percent of patients with right ventricular involvement) (19). Informed consent of each subject and approval of the Local Ethics Committee was obtained.

Assessment of ECG

Standart 12-lead electrocardiograms and right precordial electrocardiograms (V3R through V6R) were recorded immediately after admission to the emergency room.

ST-segment elevation was measured 0.08 second after the J point in leads II, III, aVF and V4R . Three consecutive QRS complexes were measured with the PQ level used as the isoelectric line. All analyses were performed by a cardiologist blinded to the clinical data of the patient. By the help of ECG of each patient, the presence of ST elevation ≥1 mm in lead V4R, ST-elevation in lead III>II was searched accordingly.

Assessment of coronary angiography

Angiographic variables were multivessel coronary artery disease, the site of occlusion of the RCA. The site of the RCA was defined as proximal or distal based on the origin of the major (>1 mm in diameter) RV branch. The purpose of the primary PCI procedure was to obtain a residual stenosis of <20% in the infarct-related artery (IRA) by visual evaluation. A succesful angiographic result was defined as residual stenosis <20% associated with TIMI grade 3 flow.

Assessment of ventricular function

Patients were underwent standard two-dimensional echocardiography with a digital ultrasonic device system (Philips IE-33, Holland) immediately after PCI. Left ventricular and right ventricular function was defined according to the rules set by American Society of Echocardiography (20). Echocardiographic evaluation of the RV function was completed by right ventricular fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE). Also, from the apical four-chamber view, the right ventricular free-wall was divided into three segments and the motion of each segment was scored on a scale of 1 to 4 (1= normal, 2= hypokinetic, 3= akinetic, 4=dyskinetic). The overall score for right ventricular free-wall motion was calculated as the average score for the segments. Modified Simpson‘s method was used to assess the left ventricular ejection fraction (LVEF).

Statistical Analysis

Statistical analysis was performed using SPSS 18.0 (SPSS, Inc., Chicago, IL, USA). Continuous variables were tested for normal distribution by the Kolmogorov-Smirnov test. Continuous variables are presented as mean+SD whereas categorical variables as count and percentages. Continuous variables were compared with Student’s t test. Categorical variables were compared with chi-square statistic or Fisher’s exact test as appropriate. Multiple logistic regression analysis was used to assess the independent predictors of RVI and in-hospital mortality estimated as relative risks with corresponding 95% confidence interval in leads III>II. A p value of <0.05 was considered statistically significant.

RESULTS

There were 90 patients (mean age 56.5±9.6 years, 74% men) without RVI and 90 patients (mean age 55.5±11.5 years, 82% men) with RVI. Baseline characteristics are listed in Table 1. There were no statistically significant differences in respect to demographic, coronary risk factors and in-hospital therapy among the groups. A large proportion of ST-elevation in lead V4R was observed in patients with RVI and a large proportion of ST-elevation in lead III>II in patients with RVI. Patient delay time (4.2±1.3 vs 4.6±1.5 , p=0.02) was significantly higher in patients with RVI , but there was no statistically significant difference in door to balloon time. At the time of admission, cardiogenic shock and ST-elevation in lead III>II were significantly higher in patients with RVI. Echocardiographic findings are listed in Table 1. Global left ventricular ejection fraction was similarly preserved in both groups. Patients with RVI had significantly worse regional right ventricular free-wall dysfunction (wall motion score index 2.0±0.6 vs.1.3±0.5 , p<0.001) ,as well as more severely depressed global right ventricular performance (TAPSE of 12.8±2.2 vs. 19.3±1.9 , p<0.001; RVFAC of 32.4±5.4 vs. 39±4.0, p<0.001). Angiographic procedural data of the groups were listed in Table 1. Coronary lesion location, multivessel coronary disease and succesful pPCI were significantly different between the groups. A higher number of patients with proximal coronary lesion was observed in patients with RVI. A higher number of unsuccesful pPCI was observed in patients with RVI. A higher number of multivessel coronary disease was observed in patients without RVI. In-hospital adverse cardiac events and in-hospital mortality were significantly higher in patients with RVI.

Table 1

Baseline characteristics

Variable	Right Ventricular Infarction Absent (n=90)	Present (n=90)	p Value
Age(years)	56.5±9.6	55.5±11.5	0.20
Male [n (%)]	67(74)	74(82)	0.45
Hypertension [n (%)]	47(52)	51(57)	0.65
Diabetes Mellitus [n (%)]	23(25.6)	22(24.4)	0.90
hyperlipidemia [n (%)]	35 (38.8)	38 (42.2)	0.83
Smoke [n (%)]	49(54)	54(60)	0.56
Body Mass Index(kg/m²)	25.2±2.8	24.3±3.2	0.25
Family History of CAD [n (%)]	15(16.5)	19(21)	0.67
Previous MI [n (%)]	20(22.2)	16(17.8)	0.46
Previous PCI [n (%)]	7(7.8)	9(10)	0.60
Previous CABG [n (%)]	3(3.3)	4(4.4)	0.38
Patient delay time (hours)	4.2±1.3	4.6±1.5	0.02
Door to balloon time (minutes)	46.5±10.5	48.5±11.8	0.12
Shock at time of admission [n (%)]	1 (1.1)	15 (16.6)	<0.001
ST Elevation in lead III>II [n (%)]	36(40)	84(93.3)	<0.001
Echocardiographic characteristics			
Left ventricular ejection fraction(%)	44.8±2.7	44±3.4	0.20
Right ventricular fractional area change (%)	39±4.0	32.4±5.4	<0.001
Tricuspit annular plane systolic excursion(mm)(TAPSE)	19.3±1.9	12.8±2.2	<0.001
Right ventricular free-wall index	1.3±0.5	2.0±0.6	<0.001
In-hospital therapy			
Aspirin [n (%)]	86(96)	87(96)	0.84
ACEI-ARA [n (%)]	55(61)	40(44.4)	0.10
Clopidogrel [n (%)]	78(87)	80(89)	0.64
Ticagrelor [n (%)]	12(13)	10(11.1)	0.60
Statin [n (%)]	84(93)	87(97)	0.53
Glycoprotein IIb /IIIa inhibitor [n (%)]	24(27)	26(29)	0.56
Coronary angiography characteristics			
Succesful PPCI [n (%)]	84 (93.3)	76 (84.4)	0.03
Multivessel coronary disease [n (%)]			0.02
1	36 (40)	52 (56.5)	
>1	54 (60)	38 (43.5)	
Coronary lesion location [n (%)]			0.001
Proximal	53 (59)	77 (85.5)	
Distal	37 (41)	13 (14.5)	
In-hospital adverse cardiac events [n (%)]			
Third-degree atrioventricular block	9(10)	19 (21)	0.03
Hypotension	8 (8.8)	30 (33.3)	<0.001
Ventricular Tachcardia/Fibrillation	5 (5.5)	13 (14.4)	0.03
Cardiogenic shock	4 (4.4)	20 (22.2)	0.008
In-hospital death	4(4.4%)	14(15.6%)	0.01

Data are expressed as mean± SD for normaly distributed data or count (percentage) for categorical variables; CAD, Coronary artery disease; MI, Myocardial infarction; PCI, Perc-utaneous coronary intervention; CABG, Coronary artery by-pass greft; ACEI-ARA, angio-tensin-converting enzyme inhibitor-angiotensin II receptor antagonist; PPCI, primary percutaneous intervention

Baseline characteristics of patients with or without ST-segment elevation in lead III>II (III<II and III>II, respectively) is summarized in Table 2. There were no statistically significant differences in respect to demographic, coronary risk factors, patient delay time door to balloon time , and shock at time of admission among the groups. Global left ventricular ejection fraction, TAPSE and RVFAC was similarly preserved in both groups. Patients with ST-segment elevation in lead III>II had significantly worse regional right ventricular free-wall dysfunction (wall motion score index 1.75±0.6 vs.1.5±0.55 , p=0.02). There were no statistically significant differences between the groups in respect to presence of coronary lesion location, multivessel coronary disease and succesful pPCI. There were no statistically significant differences between the groups in respect to in-hospital adverse cardiac events and in-hospital mortality.

Table 2

Clinical, echocardiographic and coronary angiography characterisitics, IACE and in-hospital death according to the presence of ST-elevation in lead III>II

Variable	ST-Elevation in Lead III>II		p value
	III<II (n=60)	III>II (n=120)	
Age (years)	56.5±9.1	55.8±11.3	0.28
Male [n (%)]	49(81.6)	92(76.6)	0.68
Hypertension [n (%)]	33(55)	65(54)	0.87
Diabetes Mellitus [n (%)]	13(21.7)	32(26.7)	0.92
Smoke [n (%)]	34(56.6)	75(62.5)	0.56
Hyperlipidemia [n (%)]	23(38.3)	50(41.6)	0.47
Family History of CAD [n (%)]	10(16.6)	24(20)	0.63
Patient delay time (hours)	6.0±1.3	6.2±1.4	0.52
Door to ballon time (minutes)	46±10	47±12	0.48
Shock at time of admission [n (%)]	4(6.7)	12(10)	0.45
Left Ventricular Ejection Fraction (%)	44.6±3.2	44.3±3.1	0.48
TAPSE	17.4±2.7	15.8±3.3	0.08
Right ventricular fractional area change (%)	37.7±5.2	34.5±5.6	0.15
Right ventricular free-wall index	1.5±0.55	1.7±0.6	0.02
Coronary angiographic characteristics			
Succesful PPCI	55(91.6)	105(87.5)	0.35
Multivessel disease			0.15
1	28(47)	61)51)	
>1	32(53)	59(49)	
coronary lesion location			0.25
proximal	42(70)	88(73)	
distal	18(30)	32(27)	
IACE [n (%)]			
Third-degree atrioventricular block	7(11.7)	21(14.2)	0.35
Hypotension	10(16.6)	28(23.3)	0.20
Ventricular Tachycardia/Fibrillation	4(6.7)	14(11.6)	0.15
Cardiogenic shock	6(10)	18(15)	0.10
Death	5(8.3)	13(10.8)	0.60

Data are expressed as mean± SD for normaly distributed data or count (percentage) for categorical variables; IACE, In-hospital adverse clinical events; MI, Myocardial infarction; PPCI, Primary Percutaneous coronary intervention; RCA, right coronary artery

We identified several univariate predictors of RVI in patients: Door to balloon time, patient delay time, multivessel coronary disease, coronary lesion location, unsuccesful pPCI and ST-segment elevation in lead III>II (p<0.25 for all). Patient delay time (odds ratio [OR] 1.25, 95% CI 1.11 to 1.70, p=0.02), coronary lesion location (OR 1.6, 95% CI 1.25 to 2.85, p=0.04), unsuccesful pPCI (OR 1.8, 95% CI 1.15 to 3.25, p=0.03) and ST-segment elevation in lead III>II (OR 2.8, 95% CI 1.55 to 5.25,p=0.02) were independent predictors of RVI after multivariate analysis (Table 3).

Table 3
Univariate and multivariate logistic regression analysis for prediction of RVI

Variable	Univariate			Multivariate		
	Unadjusted (	95% CI	p value	.djusted O	95%CI	p value
Door to balloon time	1.01	0.90-1.08	0.20			
Patient delay time	1.30	1.01-1.75	0.01	1.25	1.11-1.70	0.02
Multivessel coronary disease	1.2	0.30-4.72	0.15			
Coronary lesion location	1.8	1.12-3.13	0.03	1.6	1.25-2.85	0.04
Unsuccesful PPCI	2.1	1.25-6.54	0.02	1.8	1.15-3.25	0.03
ST-Elevation İn Lead III>II	3.5	1.35-11.5	0.004	2.8	1.55-5.25	0.008

OR=odds ratio; CI=confidence interval;MI, Myocardial infarction; PPCI, Primary Percutaneous coronary interention

We identified several univariate predictors of in-hospital mortality in patients: Age, door to balloon time, patient delay time, shock at the time of admission, LV ejection fraction, multivessel coronary disease, coronary lesion location, unsuccesful pPCI and ST-segment elevation in lead III>II (p<0.25 for all). Age (OR 1.04, 95% CI 1.04 to 1.65, p=0.04), patient delay time (OR 1.4, 95% CI 1.15 to 2.9, p=0.04), shock at the time of admission (OR 11.05, 95% CI 3.80 to 38.30, p=0.001) and unsuccesful pPCI (OR 2.0, 95% CI 1.18 to 3.75, p=0.02) were independent predictors of RVI after multivariate analysis (Table 4).

Table 4
Univariate and multivariate logistic regression analysis for prediction of mortality

Variable	Univariate			Multivariate		
	Unadjusted O	95% CI	p value	Adjusted OR	95%CI	p value
Age	1.15	1.02-1.08	0.01	1.1	1.03-1.6	0.04
Patient delay time	1.5	1.25-3.45	0.03	1.4	1.15-2.9	0.04
Door to balloon time	1.03	0.54-2.20	0.18			
Shock at the time of admission	12.1	3.80-45.40	<0.001	11.05	3.80-38.30	<0.001
Left ventricular ejection fraction on admission	0.90	0.08-12.2	0.22			
Multivessel coronary disease	1.2	0.30-4.72	0.20			
Coronary lesion location	1.8	1.12-3.13	0.02			
Unsuccesful PPCI	2.5	1.08-7.15	0.01	2.0	1.18-3.75	0.02
ST-Elevation İn Lead III>II	1.5	0.68-3.15	0.40			

OR=odds ratio; CI=confidence interval;MI, Myocardial infarction; PCI, Percutaneous coronary interention; RV,Right ventricular

DISCUSSION

In the present study, ST-elevation in lead III >II was an independent predictor of RVI in patients with AIMI undergoing pPCI. This association remained significant after adjusting for other confounding factors that were identified in multivariate analysis. However, this predictive effect was not observed in-hospital mortality. The standard 12 lead ECG, right-sided chest leads and posterior chest leads, in conjuction with clinical findings often provide the necessary information for physician to predict complications.

AIMI patients may have associated RVI, evaluation using standard 12-lead electrocardiography (ECG) often reveals corresponding ST segment elevations in leads II, III and aVF. Standard 12-lead ECG images mainly assess the LV. However, Lead III only directly images RV. In the literature there has been only one study searching the diagnostic value of ratio of ST-elevation in lead III>II for RVI and it was retrospectively designed (15). In this study, Saw et al. found that this ratio had sensitivity of 97%, but relatively nonspecific when compared to ST-elevation in lead V4R (56% vs. 78%, respectively). The presence of acute ST-elevation, Q waves or both in the right precordial leads (V3R-V6R) was found to be reliable in the diagnosis of right ventricular infarction compared to the gold standard methods such as hemodynamic assessment or autopsy. The right-sided precordial leads can show ST-elevation across the entire right precordium from V1R through V6R ; a sole ST-elevation in lead V4R>1 is a reliable marker of an RVI, with sensitivity 88%, specificity 78 % using findings from results of autopsy, cardiac catheterization, hemodynamic monitoring and radionuclide imaging as the ''gold standard''(6). Also, the diagnostic accuracy of this finding is greatest within the first 10 h of symptoms, as it disappears in 50% of patients after this time period (6,21). In the literature, there has not been any prospective study to evaluate ST-elevation in lead III>II findings in predicting right ventricular infarction (RVI) in primary angioplasty era. In this study, we found that odds ratio of the ST-elevation in lead III>II for predict of RVI was 2.8 . Also, patients with ST-elevation in lead III>II had significantly worse regional right ventricular free-wall motion. In this case we thought that lead III images directly RV free wall. In the setting of AIMI, if the larger ST-elevation in lead III>II, RVI is also suggested. The prognostic effect of ECG criteria pertaining to ratio of the ST-elevation in lead III>II for RVMI is uncertain in AIMI patients undergoing primary PCI. There have been only one study searching predictive value of ST-elevation in III>II for in-hospital mortality in trombolitic era(15). In this study, 70% of the patients were given thrombolytic therapy while 30% of the patients took only medical therapy without reperfusion. They found that odds ratio of ST-elevation in lead III>II for predicting in-hospital mortality was 5.0. In our study, we applied mechanical perfusion therapy to all patients and the patients were homogenously distributed in respect to the type of supplementary medical treatment. Based on this population, we found that odds ratio of ST elevation in lead III>II for in-hospital mortality was 1.5. In this study , patients with ST-elevation in lead III>II had significantly worse regional right ventricular free-wall motion, but there were no significant difference RVFAC and TAPSE between groups. RVFAC and TAPSE defines global right ventricular performance (20). In this study RVFAC was observed close to normal values in patients with ST-elevation in lead III>II. Since the global right ventricular sistolic functional deterioration, in-hospital adverse events and hemodynamically deteriorating were not observed different than in patients with ST-elevation in lead III<II. RVFAC and TAPSE values of patients with ratio of the ST-elevation in lead III>II were similar to those of the patients with ratio of the ST-elevation in lead III<II Additionally there was no significant difference between these two groups in respect to in-hospital adverse events and in-hospital mortality. Also, we did not find any significant relation between in-hospital adverse events/in-hospital mortality and RVFAC/TAPSE indicating echocardographically RV hemodynamical comprimise. Altogether, ratio of the ST-elevation in lead III>II was not predictor for in-hospital mortality. Actually, the main reason of such result was due to mechanical intervention rather than thrombolytic therapy. Otherwise, it is well known that primary PCI reduces in-hospital mortality and it has been proven by the previous studies. That is the reason that ratio of the ST-elevation in lead III>II was not helpful predictor for in-hospital mortality. Thus, it meant that ST-elevation in lead III>II did not have sufficient correlation with global RV performance in pPCI era.

Another main finding that we also found that proximal coronary lesion location and longer delay time have an independent predictor of RVI. RVI usually develops upon total occlusion of the proximal right coronary artery (RCA)(22-24). Some studies described proximal RCA occlusion compromising flow to the major RV branches as the most common anatomic substrate of RV dysfunction.

In this study confirmed that proximal RCA lesion location was associated with RVI. In our recently published, we demostrated that time duration from symtom onset to PCI (delay time) has an independent predictor of RV dysfunction(25). It meant that the patient has longer delay time increases risk of RVI.

Limitation toour studies were as follows: 1) In enrollment of patients, nearly gold standard method which is hemodynamic assessment was not used for diagnosis of RVI. 2) Echocardiographic examination was carried out following PCI due to short of time, thus possibility of recovery in RV functions just after PCI and inability to assess interobserver variability 3) inability to evaluate RV functions by strain and strain rate.

CONCLUSION

ST-elevation in lead III>II was an independent predictor of RVI in patients with RCA related AIMI undergoing pPCI. However, ST-elevation in lead III>II was not predictor of in-hospital mortality.

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