의학석사 학위논문

Diabetes Mellitus is Associated with Increased Mortality after Acute Myocardial Infarction Only in Patients with Preserved Left Ventricular Systolic Function

급성 심근경색 후 좌심실 수축 기능부전 유무에 따라 달라지는 당뇨의 사망률에 대한 영향

2015 년 2 월

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지도 교수 김 용 진

이 논문을 의학석사 학위논문으로 제출함.

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의학과 임상의과학 전공

임 문 순

임문순의 의학석사 학위논문을 인준함.

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위원장 _____(인)

부위원장 _____(인)

위원 _____(인)

ABSTRACT

Background Left ventricular (LV) systolic function and diabetes mellitus (DM) are the primary determinants of clinical outcomes in patients with acute myocardial infarction (AMI). This study investigated the impact of DM on mortality according to LV systolic function after AMI.

Methods Between November 2005 and September 2009, a total of 12,486 patients from a nationwide database, who survived 30 days after AMI, were included in the study. Patients were stratified into two groups according to their LV ejection fraction (LVEF): LVEF<40% (n=1,732, 13.9%) and LVEF≥40% (n=10,754, 86.1%), and the effect of DM was analyzed in each group.

Results DM was more prevalent in patients with LVEF<40% than in those with LVEF≥40% (n=622, 36.1% vs n=2786, 25.9%; P<0.001). The 12-month mortality was higher in patients with with LVEF<40% than in those with LVEF≥40% (8.2% vs 2.0%, P<0.001) and DM than in those without DM (7.2% vs 10.1%, P=0.012; 1.6% vs 3.1%, P<0.001). In multivariate analysis, DM was an independent predictor of mortality in the LVEF ≥40% group (HR: 1.75; 95% CI: 1.23–2.49; P=0.002), but not in the LVEF<40% group (HR: 1.21; 95% CI: 0.80–1.83; P=0.364).

Conclusions DM was a significant independent risk factor for 12-month mortality in patients with LVEF≥40% but not in those with LVEF<40% after AMI.

Keywords: acute myocardial infarction, left ventricular systolic function, diabetes mellitus

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LIST OF ABBREVIATIONS

ACEI: angiotensin-converting enzyme inhibitor

AMI: acute myocardial infarction

ARB: agiotensin II receptor blocker

CAD: coronary artery disease

DM: diabetes mellitus

ECG: electrocardiographic

HDL: high-density lipoprotein

KAMIR: Korean Acute Myocardial Infarction Registry

KorMI: Korean Working Group on Myocardial Infarction

LDL: low-density lipoprotein

LV: left ventricular

LVEF: LV ejection fraction

LVSD: LV systolic dysfunction

MACE: major adverse cardiovascular event

PCI: percutaneous coronary intervention

RAS: renin-angiotensin system

STEMI: ST-segment elevation myocardial infarction

TIMI: thrombolysis in myocardial infarction

INTRODUCTION

Acute myocardial infarction (AMI) is one of the main causes of morbidity and mortality worldwide.[1] Numerous large clinical studies have identified several poor prognostic factors in patients with AMI including advanced age, female gender, left ventricular (LV) systolic dysfunction, and the presence of diabetes mellitus (DM), hypertension, renal insufficiency, or hemodynamic instability.[2-5] Among these factors, LV systolic dysfunction represented by LV ejection fraction (EF) is a major risk factor for mortality after AMI. [6] In addition, DM is another important prognostic factor after AMI. The risk of heart failure or death is higher in patients with DM when compared with those without DM despite a similar LVEF.[7] DM accelerates coronary atherosclerosis and increases coronary events after AMI. Moreover, it induces myocardial fibrosis and hypertrophy, causes myocardial systolic and diastolic dysfunction, and has an adverse effect on the process of post-infarct LV remodeling.[8,9] Because DM directly affects the vasculature and myocardium, the impact of DM on the prognosis may differ according to the LV systolic function. Therefore, we hypothesized that the impact of DM on mortality in patients with AMI may differ according to the LV systolic function. We analyzed the impact of DM on mortality in 30-day survivors after AMI according to LV systolic function.

METHODS

Study population

The study population consisted of consecutive patients with AMI who were registered in national databases (Korean Acute Myocardial Infarction Registry [KAMIR] and Korean Working Group on Myocardial Infarction [KorMI]). The registry database was constructed in 2005 with a prospective, multicenter, observational design, and 59 university or community hospitals with a large patient population and facilities for primary percutaneous coronary intervention (PCI) participated. At the participating sites, consecutive patients admitted with AMI were asked to register to be included in this study. Data were collected at each institution by trained study coordinators using a web-based, computerized database program and standardized case report form and protocol. Between November 2005 and September 2009, a total of 16,587 patients with presumed AMI were identified from the KAMIR and KorMI databases. Of these patients, 573 were excluded because their final diagnosis was other than AMI. In addition, 1,708 patients whose LV systolic function information was unavailable and 1,318 patients who had insufficient clinical parameters available for the study analysis were also excluded. Since 501 patients died within 30 days of AMI, the remaining 12,468 patients who survived 30 days were included for the final analysis in this study. The flow of the patient enrollment is shown in Figure 1. The Institutional Review Board of each participating center approved the study protocol, and written informed consent was obtained from each study patient.

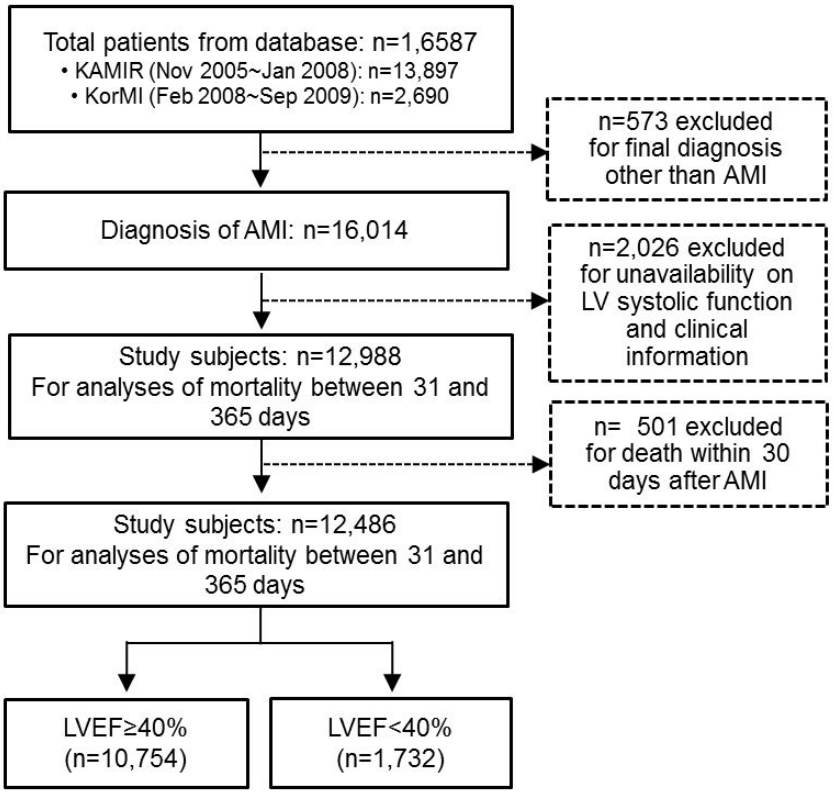


Figure 1. Flow chart of patient enrollment

Clinical information

The diagnosis of AMI was based on clinical symptoms, cardiac enzyme elevation, and electrocardiographic (ECG) changes suggesting infarction.10 ST-segment elevation myocardial infarction (STEMI) was diagnosed by the cardiologists in patients with ST elevation in more than 2 ECG leads or a new left bundle branch block with a concomitant increase in cardiac enzymes. Information on demographic and clinical characteristics, including age, gender, body mass index (BMI), smoking status, history of DM, hypertension, dyslipidemia, ischemic heart disease, and other comorbidities, were obtained

at initial admission. Hypertension was defined as a history of hypertension or drug treatment for hypertension instead of high blood pressure. DM was defined as a history of drug treatment for DM instead of high blood glucose. Dyslipidemia was defined as a diagnosis previously made by physicians or treatment with lipid lowering medications. BMI was calculated by weight in kilograms divided by height in meters squared (kg/m2). Systolic and diastolic blood pressure and heart rate were checked by trained nurses at admission, and LVEF was determined by 2-dimensional echocardiography before hospital discharge. The Killip classification categorized patients based on the presence of heart failure, pulmonary edema, and shock at initial admission.11 Blood samples for baseline laboratory tests other than the lipid profile were collected at admission before initial treatment. Blood samples for the lipid profile including total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, and fasting glucose, were obtained after an 8-hour overnight fast. The initial treatment strategy for AMI patients was PCI, thrombolysis, or conservative management, which was determined by the attending physicians based on the recommendations in the guidelines.[12,13] Primary PCI was defined as emergency angioplasty and/or stent implantation in patients with STEMI within 12 hours after admission or in patients with continuing symptoms or cardiogenic shock even after 12 hours. Early invasive treatment was defined as urgent PCI within 24 hours of admission in patients with non-STEMI.

Clinical events

Follow-up hospital visits were scheduled at 1, 6, and 12 months after discharge. Information on death, nonfatal myocardial infarction (MI), and revascularization (PCI or coronary bypass surgery) was collected by medical record review or telephone interviews, if necessary. Only the first major adverse cardiovascular event (MACE) was considered for each patient. When two or more events were recorded during the same hospitalization, the most serious event was determined in the following order: death, non-fatal MI, and revascularization.

Statistical analysis

Data are expressed as mean±SD or median (interquartile range). For study analysis, the patients were divided into two groups according to LVEF, with a cutoff value of 40%.[14,15] Univariate comparisons between the two groups were made using Pearson's Chi-square tests for categorical variables or Student's t tests for continuous variables. Multiple logistic regression and Cox proportional regression analyses were conducted to identify the independent prognostic factors and determine the impact of the presence of DM on mortality. Several clinical variables including age, gender, BMI, DM, hypertension, dyslipidemia, smoking history, past history of MI, Killip class, PCI, multi-vessel disease, and serum creatinine were initially analyzed using the univariate regression model. Variables with P values <0.1 in univariate analysis were incorporated into a multivariate regression model. Significance levels for all analyses were set at a $P<0.05$. All statistical analyses were performed using SPSS 18.0 (IBM Co., Armonk, NY, USA).

RESULTS

Baseline characteristics

The baseline characteristics of the patients are shown in Table 1. The differences between DM and non-DM patients were amplified when the EF category was considered. The overall prevalence of DM was 27.3%, and it was higher in patients with LVEF<40% than in those with LVEF>40% (35.9% vs 25.9%, P<0.001). Compared to those without DM, there was a higher female predominance, Killip class, creatinine level, and BMI, and a lower LVEF, in patients with DM; they also had more frequent co-morbidities including hypertension and dyslipidemia. In addition, patients with DM had a lower rate of PCI (76.5% vs 82.3%) only in those with LVEF<40%.

Table 1. Baseline characteristics

Variable	Overall (n= 12,486)		LVEF <40% (1,732)		LVEF ≥40% (10,754)	
	Non-Diabetes (n=9,078)	Diabetes (n=3,408)	Non-Diabetes (n=1,110)	Diabetes (n=622)	Non-Diabetes (n=7,965)	Diabetes (n=2,789)
Age, years	62.8±13.1	64.8±11.3**	66.0±13.3	65.9±10.9	62.3±13.0	64.6±11.4**
Male sex, n (%)	6641(73.5)	2231(65.5)**	801(72.9)	408(65.6)*	5840(73.6)	1823(65.4) **
Body mass index, kg/m^2	24.0±3.5	24.2±3.3*	23.2±3.5	23.6±3.2	24.1±3.5	24.3±3.3*
Hypertension, n (%)	3830(42.4)	2162(63.5)**	482 (43.9)	405(65.1) **	3348(42.2)	1757(63.1) **
Dyslipidemia, n (%)	730(8.1)	384(11.3)**	73(6.7)	66(10.6) **	657(8.3)	318(11.5) **
Smoking, n (%)	5657(62.9)	1713(50.5)**	625(57.3)	284(45.9) **	5032(63.7)	1429(51.5) **
Previous MI, n (%)	325(3.6)	157(4.6)*	87(7.9)	42(6.8)	238(3.0)	115(4.1) *
SBP, mmHg	128.8±27.0	130.0±27.7	125.4±27.0	127.3±27.9	129.1±27.0	130.5±27.6
DBP, mmHg	79.2±16.2	78.4±15.9	77.0±16.6	77.4±16.2	79.5±16.2	78.7±15.8
Killip class ≥ 2, n (%)	1958(22.3)	1032(31.1)**	463 (43.3)	355(58) **	1495(19.4)	677(25) **
LVEF, %	53.1±11.9	50.8±12.7**	32.3±5.6	31.7±5.9**	56.0±9.4	55.1±9.4
STEMI, n (%)	5421(60.1)	1763(51.9)**	730(66.5)	316(50.88) **	4691(59.2)	1447(52.1) **
Total-cholesterol, mg/dL	185.3±43.1	179.0±45.2**	180.8±45.4	174.3±44.4*	185.9±42.8	180.0±45.3**
creatinine (mg/dL)	1.1±1.1	1.4±1.5**	1.2±1.6	1.7±2.0**	1.1±1.0	1.3±1.4**
Multivessel disease, n (%)	1762(20.4)	1088(33.7)**	292(29.5)	280(49) **	1470(19.2)	808(30.4) **
Initial treatment strategy, n (%)						
STEMI						
Primary PCI	4253(79.1)	1380(78.3)	558(77.1)	225(71.2)	3695(79.4)	1155(79.9)
Facilitated PCI	303(6.6)	90(6.1)	32(5.1)	19(6.9)	271(6.9)	71(5.9)

Thrombolysis	583(6.5)	148(4.4)*	59(5.4)	25(4.0)	524(6.7)	123(4.4) **
Conservative	557(10.4)	229(13)*	105(14.5)	64(20.3)	452(9.7)	165(11.4)
NSTEMI						
Early invasive	2176(62.3)	946(59.2)	162 (46.2)	149(50.5)	2014(64.1)	797(61.2)
Conservative	1317(37.7)	651(40.8)	189(53.8)	146(51.9)	1128(35.9)	505(38.8)
PCI done, n (%)	7952(88.1)	2944(86.4)	904(82.3)	476(76.5) *	7048(88.9)	2468(88.6)
Discharge medication, n (%)						
Aspirin	8739(97.9)	3271(97.8)	1050(98.1)	580(96.5)	7689(97.9)	2691(98.1)
Clopidogrel	8287(92.8)	3130(93.5)	980(91.6)	554(92)	7307(93.0)	2576(93.8)
ACEI/ARB	7343(82.2)	2778(83.0)	862(80.6)	476(79.1)	6481(82.5)	2302(83.9)
Beta-blockers	6955(77.9)	2638(78.8)	784(73.3)	444(73.8)	6174(78.5)	2194(79.9)
Statins	6794(76.1)	2463(73.6)*	764(71.4)	416(69.1)	6030(76.7)	2047(74.6)

*P <0.01, **P <0.001

Independent predictors of 12-month mortality

Table 2 shows the outcome of the study population at 12 months after AMI. Approximately 95.6% of the patients were available for a 12-month clinical follow-up during which the DM group had significantly higher incidences of all-cause death, cardiac death, and MACE, regardless of LVEF (2.2% vs 4.4%, $P<0.001$; 1.3% vs 2.9%, $P<0.001$; 9.0% vs 13.4%, $P<0.001$, respectively) At 12 months, death occurred in 352 patients (3.4%). The mortality rate was higher in patients with LVEF<40% than in those with LVEF≥40% (8.2% vs 2.0%, $P<0.001$). Multiple logistic regression analyses were performed to determine independent predictors of 12-month mortality in each group according to LVEF (**Table 3, Figure 2**). In multiple logistic regression analysis, age, DM, Killip class on admission, multi-vessel disease, PCI, and serum creatinine level were identified as independent predictors of mortality in patients with LVEF≥40%. In contrast, in patients with LVEF<40%, age, hypertension, Killip class on admission, and serum creatinine level were determined to be independent predictors of mortality, whereas DM was not an independent predictor of mortality.

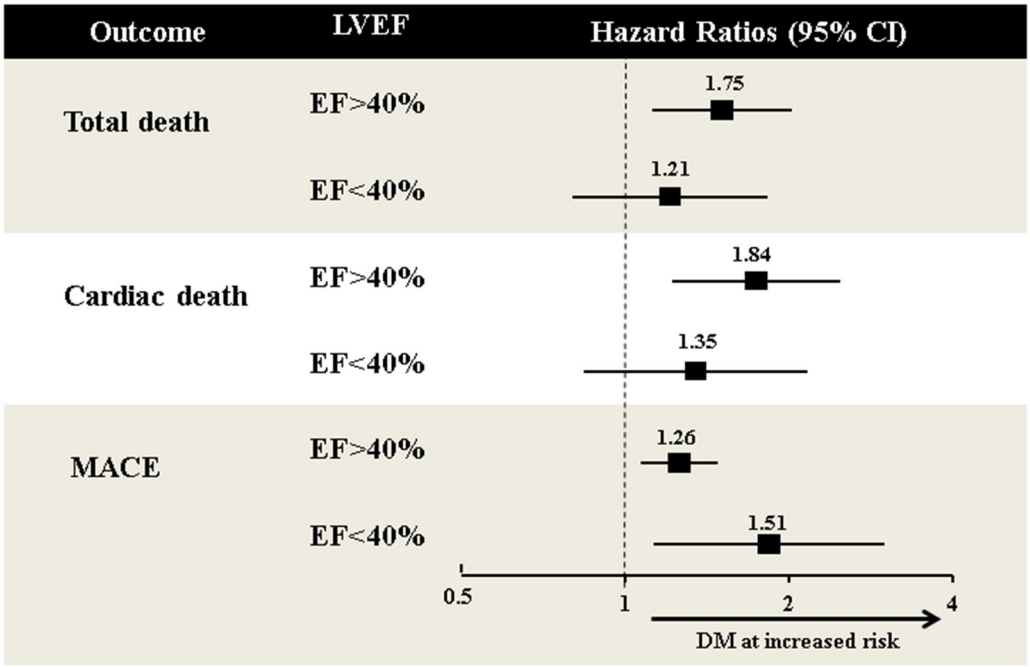
Table 2.Clinical events at 12 month following acute myocardial infarction

		Total (n=12486)	DM(-) (n=9078, 72.7%)	DM(+) (n=3408, 27.3%)
Total death, n (%)	EF≥40%	210/10754 (2.0%)	123/7932 (1.6%)	87/2789 (3.1%)
	EF<40%	142/1732 (8.2%)	79/1099 (7.2%)	63/622 (10.1%)
	Overall	352/12486 (3.4%)	202/9078 (2.2%)	150/3408 (4.4%)
Cardiac death, n (%)	EF≥40%	114/10754(1.1%)	65/7932 (0.8%)	49/2789 (1.8%)
	EF<40%	105/1732 (6.1%)	56/1099 (5.1%)	49/622 (7.9%)
	Overall	214/12486 (2.1%)	121/9078 (1.3%)	98/3408 (2.9%)
MACE, n (%)	EF≥40%	997/10754 (9.2%)	670/7932 (8.4%)	327/2789 (11.5%)
	EF<40%	283/1732 (16.3%)	151/1099 (13.7%)	132/622 (21.2%)
	Overall	1280/12486 (11.8%)	821/9078 (9.0%)	459/3408 (13.4%)

Table 3 Regression Analysis: Factor Associated with 12month mortality

Variable	Univariate (LVEF≥40%)		Multivariate (LVEF≥40%)		Univariate (LVEF〈40%)		Multivariate (LVEF〈40%)	
	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
Age	1.07 (1.06-1.09)	<0.001	1.06 (1.04-1.08)	<0.001	1.04 (1.03-1.06)	<0.001	1.04 (1.02-1.06)	<0.001
Male gender	0.52 (0.39-0.71)	0.001	1.13 (0.95-1.71)	0.558	0.84 (0.57-1.23)	0.360		
BMI ≥ 25kg/m^2	0.51 (0.36-0.72)	<0.001	0.79 (0.54-1.14)	0.206	0.84 (0.55-1.27)	0.402		
Hypertension	1.57 (1.18-2.12)	0.003	0.89 (0.63-1.26)	0.519	2.04 (1.40-2.99)	<0.001	1.78 (1.16-2.72)	0.008
DM	2.03 (1.51-2.74)	<0.001	1.75 (1.23-2.49)	0.002	1.56 (1.08-2.25)	0.018	1.21 (0.80-1.83)	0.364
Dyslipidemia	1.03 (0.62-1.71)	0.905			1.60 (0.90-2.85)	0.110		
Smoking	0.57 (0.42-0.76)	<0.001	0.92 (0.64-1.33)	0.663	0.81 (0.56-1.16)	0.248		
PrevMI	1.78 (0.93-3.41)	0.081	1.22 (0.57-2.61)	0.603	1.21 (0.63-2.33)	0.567		
Killip class ≥ 2	3.82 (2.84-5.14)	<0.001	2.48 (1.77-3.47)	<.0011	2.54 (1.72-3.74)	<0.001	1.95 (1.29-2.94)	0.002
MultiVD	3.05 (2.12-4.24)	<0.001	2.16 (1.53-3.03)	<0.001	2.05 (1.38-3.05)	<0.001	1.42 (0.94-2.13)	0.094
PCI done	0.23 (0.17-0.32)	<0.001	0.52 (0.33-0.81)	0.004	0.43 (0.29-0.64)	<0.001	0.76 (0.44-1.30)	0.309
Creatinine	1.18 (1.12-1.25)	<0.001	1.11 (1.04-1.19)	0.002	1.13 (1.05-1.22)	0.001	1.13 (1.04-1.24)	0.005

Figure 2. Risk of different outcomes associated with DM

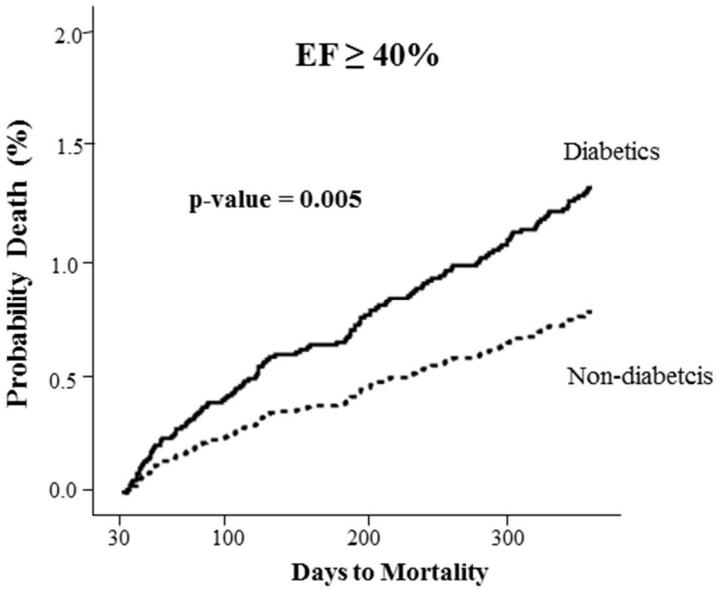


Interaction between the presence of DM and LVEF

DM was an independent risk factor for each component of MACE in patients with LVEF≥40% and LVEF<40%. However, DM was an independent risk factor for mortality only in patients with LVEF≥40% and not in those with LVEF<40%. The adjusted HR for mortality in patients with DM compared with non-DM patients was 1.75 (1.23–2.49, P=0.002) in those with LVEF≥40% and 1.21 (0.80–1.83, P=0.364) in those with LVEF<40%. Cox regression plots on the incidence of 12-month mortality between the 2 groups according to LVEF are shown in **Figure 3**.

Cox regression plots after adjustment for confounders showed that the cumulative incidence of mortality within 12 months was significantly higher in DM patients than in non-DM patients among those with LVEF<40%.

Figure 3. COX regression plot for Mortality



DISCUSSION

Using unselected consecutive patients with AMI from a large number of hospitals, our study showed that the prognostic impact of DM was different according to LV systolic function in patients with AMI. DM was an independent risk factor for mortality in patients with LVEF≥40% but not in those with LVEF<40%.

LVEF and DM are important predictors of clinical outcomes after AMI, as shown in many large clinical studies. The detrimental effect of LVEF on long-term prognosis was also shown in a study by the European Cooperative Study Group and the Argatroban in the Acute Myocardial Infarction-2 (ARGAMI-2) trial. The ARGAMI-2 trial reported that 211 patients (28%) with LVEF<40% showed a higher 6-month mortality rate than 541 patients (72%) with LVEF≥40% (13.9% vs 2.6%, P<0.001).[14] These findings are consistent with ours that also demonstrated a higher mortality rate in patients with an LVEF<40% (8.2% vs 2.0%, P<0.001). Higher mortality rate of patients with low LVEF has been shown to be associated with ventricular arrhythmia and pump failure. Patients with low LVEF are more likely to have larger myocardial scars and sustained ventricular arrhythmia.[16] In addition, LV dysfunction leads to post-infarct LV remodeling and increased wall stress that causes progressive dilation and deterioration in contractile function.[17]

Numerous studies have advocated that DM is associated with poor cardiovascular outcomes in patients with coronary heart disease. Hyperglycemia, insulin resistance, abnormal platelet function, and coagulation abnormalities have been reported to be associated with excessive thrombus formation and neointimal formation leading to stent thrombosis and in-stent restenosis.[18] Increased susceptibility to myocardial

pump failure and diastolic dysfunction by diminished collagen cross-linking have also been reported to contribute to poor outcomes in DM patients.[19] In addition, a higher incidence of silent myocardial ischemia leads to late presentation and may be another reason for poorer outcomes in DM patients.[20] Furthermore, it has been reported that patients with DM have worse baseline clinical characteristics compared to those without DM.[21] Consistent with previous findings, our patients with DM were found to have worse LV and renal function as well as more severe coronary artery disease than those without DM. However, our study showed that DM was associated with 12-month mortality only in patients with LVEF≥40%, but not in those with LVEF<40%. A few previous studies have demonstrated the prognostic impact of DM in AMI patients according to LV systolic function, and the results are in line with our findings. Deedwania *et al*.[21] studied 6,632 patients with LVEF<40% after AMI for more than two years and demonstrated that DM is a significant independent risk factor for recurrent AMI, although DM is not associated with mortality. Andersson *et al*.[22] investigated 16,912 patients with AMI, who were screened for three clinical trials in 7 years, and reported that patients with LVEF≥40% after AMI have a greater mortality risk attributable to DM than those with LVEF<40%. However, those studies were mainly conducted in the pre-drug-eluting stent (DES) era, and the data were largely collected from clinical trials. Unlike previous studies, the strength of the present study lies in the use of nationwide estimates with a large sample size, unselected population, current treatment strategies with DES, and proper adjustment for major potential confounders. The association of DM with a greater risk of mortality only in patients with LVEF≥40%

but not in those with LVEF<40% requires an explanation. Patients with LVEF<40% have a high rate of early death that conceals the 'effect' of DM on late mortality. Patients with preserved LV function have more residual myocardium that can be effected by diabetes and more prevalence of diastolic heart failure. Moreover, DM may interact with other risk factors such as hypertension and be the primary cause of cardiac dysfunction in patients with preserved LV systolic function[23]. In addition, DM has previously been identified as an independent predictor of the development of atrial fibrillation that was reported to be associated with increase in mortality only in patients with preserved EF.[24]

The prevalence of DM in the present study was 27.3%, which is similar to that reported in prior AMI studies.[3,21] However, the percentage of patients in our study with LVEF<40% (13.9%) was lower than has been previously reported (25%–43%).[14,25,26] This discrepancy may be attributed to different patient characteristics and treatment strategies. In particular, rapid reperfusion therapy with recent advanced techniques may decrease the incidence of LV systolic dysfunction caused by ischemia. The first limitation of this study is its retrospective design. Second, 1,708 patients were excluded due to the unavailability of information on LV systolic function by echocardiography, which may have affected the results. Third, we did not have any data on the type and duration of DM and information regarding the management of DM that may have impacted the prognosis.

CONCLUSION

The results of this study suggest that the prognostic impact of DM differs

according to the LV systolic function. DM was a significant independent risk factor for 12-month mortality in patients with LVEF≥40% but not in those with LVEF<40% after AMI. These results provide insight into the pathophysiology of the link between DM and cardiovascular disease after AMI.

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국 문 초 록

서론 급성 심근경색 환자에서 좌심실 기능저하와 당뇨는 임상적 결과를 결정하는 주요한 요소이다. 본 연구에서는 급성 심근 경색 이후 당뇨의 좌심실 기능저하 유무에 따라 다른 영향에 대하여 분석하고자 하였다.

방법 2005년 11월부터 2009년 8월까지 심근경색 30일 이후 생존한 급성 심근경색(acute myocardial infarction, AMI) 데이터베이스에 등록된 12,486명의 급성 심근경색 심근경색 환자를 연구 대상으로 하여 좌심실 수축기능 40%을 기준으로 두 그룹으로 나누고: 좌심실 수축기능 <40% (n=1,732, 13.9%)과 좌심실 수축기능 ≥40% (n=10,754, 86.1%), 각 그룹에서의 당뇨 영향을 분석하였다.

결과 좌심실 수축기능 40% 미만의 급성 심근경색 환자 그룹에서 40%이상의 그룹보다 당뇨 유병률이 높았다 (n=622, 36.1% vs n=2786, 25.9%; *P*<0.001). 12개월 사망률은 좌심실 수축 기능 40% 미만인 그룹이 40%이상인 그룹 보다 높았으며 (8.2% vs 2.0%, *P*<0.001)두 그룹 모두 에서 모두 당뇨가 있는 그룹이 당뇨가 없는 그룹보다 사망률이 높았다 (7.2% vs 10.1%, *P*=0.012; 1.6% vs 3.1%, *P*<0.001). 당뇨는 다변량분석에서 좌심실 수축기능 40%이상에서는 사망을 예측하는 독립 인자였으나 (HR: 1.75; 95% CI: 1.23–2.49; *P*=0.002) 좌심실 수축기능 40%미만에서는 독립 인자로 작용하지 못하였다 (HR: 1.21; 95% CI: 0.80–1.83; *P*=0.364).

결론 심근경색 12개월후 사망률의 독립적인 위험요인으로 당뇨는 좌심실 수축기능 40%이상에서는 작용하였지만 40%미만에서는 그렇지 못하였다.

주요어: 급성심근경색, 좌심실 수축기능, 당뇨

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