



Old Age and Myocardial Injury in ST-Segment Elevation Myocardial Infarction



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ABSTRACT

Background: Little is known about the causality and pathological mechanism underlying the association between old age and myocardial injury in patients with ST-segment elevation myocardial infarction (STEMI). We evaluated the association between old age and myocardial injury in STEMI patients undergoing primary percutaneous coronary intervention (PCI) using cardiovascular magnetic resonance imaging (CMR).

Methods: A total of 279 patients with STEMI who underwent primary PCI and CMR were enrolled. Of these, 52 patients were over the age of 70 years (18.6%, Age ≥ 70 group) and 227 patients were under the age of 70 years (81.4%, Age < 70 group) at STEMI occurrence. We compared myocardial infarct size on CMR according to age at STEMI occurrence and performed inverse probability of treatment weighting.

Results: On CMR analysis, myocardial infarct size on CMR tended to be greater in the Age ≥ 70 group than in the Age < 70 group ($21.2 \pm 10.2\%$ versus $19.5 \pm 11.1\%$; $p = 0.072$). After performing inverse probability of treatment weighting adjustment, myocardial infarct size was significantly greater in the Age ≥ 70 group compared with the Age < 70 group ($22.6 \pm 10.4\%$ versus $19.6 \pm 11.1\%$; $p = 0.001$). Subgroup analysis of patients older than 70 years revealed no significant difference in myocardial infarct size according to sex ($20.1 \pm 11.5\%$ in females versus $20.4 \pm 9.9\%$ in males; $p = 0.901$).

Conclusions: Despite appropriate coronary revascularization, old age was associated with greater extent of myocardial injury in STEMI patients.

Keywords: Aged; Myocardial ischemia; ST-segment elevation myocardial infarction; Magnetic resonance imaging. [Am J Med Sci 2021;362(6):592–600.]

INTRODUCTION

Worldwide life expectancy has increased dramatically and old age itself is an independent poor prognostic factor for various diseases including acute myocardial infarction (MI).¹ Previous studies reported that old age independently influenced short- and long-term mortality in patients with ST-segment elevation myocardial infarction (STEMI).^{2,3} Lonborg et al.⁴ found that aging and large infarct size in patients with STEMI were associated with higher all-cause-mortality. However, although the value of primary

percutaneous coronary intervention (PCI) over thrombolysis was established several years ago, the mechanisms underlying the direct relationship between old age and myocardial injury remain poorly understood in STEMI with prompt revascularization. Cardiovascular magnetic resonance imaging (CMR) can precisely assess the extent of myocardial injury and salvaged myocardium, providing valuable insight into the effects of old age in patients with STEMI.^{5,6} We evaluated the association between old age and myocardial injury using CMR in STEMI patients undergoing primary PCI.

METHODS

Ethics statement

The Institutional Review Board of Samsung Medical Center and Samsung Changwon Hospital, Sungkyunkwan University School of Medicine approved this study, and all subjects provided written informed consent to participate. All research was performed in accordance with the principals of the Declaration of Helsinki.

Study population

The study population consisted of patients on the Acute Myocardial Infarction-Cine Magnetic Resonance imaging registry at Samsung Medical Center in Seoul, Korea, from December 2007 to July 2016. A total of 439 consecutive patients were eligible for enrollment after presenting with STEMI. Inclusion criteria for this study were as follows: 1) patients treated with primary PCI within 12 hours after symptom onset, and 2) patients who underwent CMR after the index procedure. Exclusion criteria were as follows: 1) previous coronary artery bypass grafting, 2) history of MI, 3) patients who received reperfusion therapy more than 12 hours from symptom onset, 4) insufficient information regarding symptom onset time, 5) door to balloon time exceeding 90 minutes, and 6) patients with poor-quality CMR data for analysis.⁷ Finally, a total of 279 patients were included in this study (Fig. 1).

Definition of study group and study outcomes

Study patients were divided into two groups according to the age at STEMI occurrence: patients who

suffered from STEMI over the age of 70 years (Age ≥ 70 group) and patients who suffered from STEMI before the age of 70 years (Age < 70 group).⁸ The primary outcome was myocardial infarct size (% of left ventricle [LV]) assessed by CMR according to age at presentation. The secondary outcomes were the extent of the area at risk (AAR; % of LV), myocardial salvage index (MSI), and microvascular obstruction (MVO) area (% of LV).

Data collection and definition

Research coordinators for the dedicated registry prospectively recorded baseline characteristics, angiographic findings, and CMR data. Blood samples for N-terminal pro B-type natriuretic peptide (NT-proBNP) and creatine kinase myocardial band (CK-MB) were drawn from patients before primary PCI. Serum CK-MB levels were measured every eight hours from the index procedure until peak values were confirmed.⁷ Baseline left ventricular ejection fraction (LVEF; %) was measured by transthoracic echocardiography using Simpson's method just after primary PCI.⁹ Killip classification was determined upon arrival or before primary PCI.¹⁰ STEMI was defined as ST-segment elevation more than 1 mm in two or more contiguous leads or presumably new-onset left bundle branch block on electrocardiogram. Multivessel disease was defined as stenosis $> 50\%$ noted in more than two coronary arteries.⁷ Thrombolysis in myocardial infarction flow grade and myocardial blush grade were evaluated using the final angiogram, as defined previously.¹¹ All baseline and procedural cine coronary angiograms were reviewed and quantitatively analyzed at our institution's angiographic core laboratory.

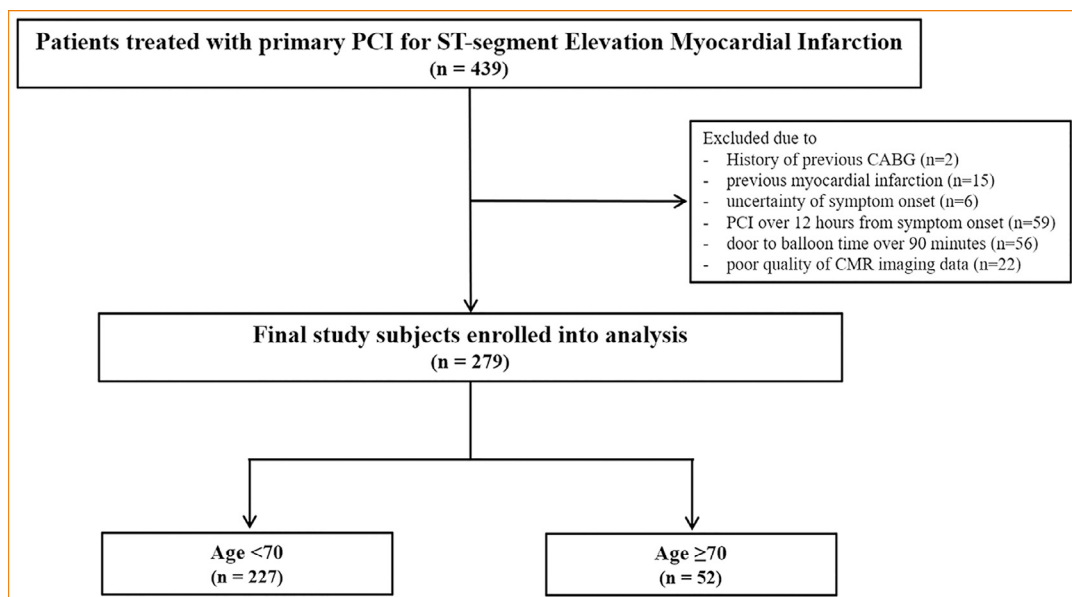


FIGURE 1. Schematic of study cohort selection.

Abbreviations: CABG, coronary artery bypass grafting; CMR, cardiac magnetic resonance imaging; PCI, percutaneous coronary infarction.

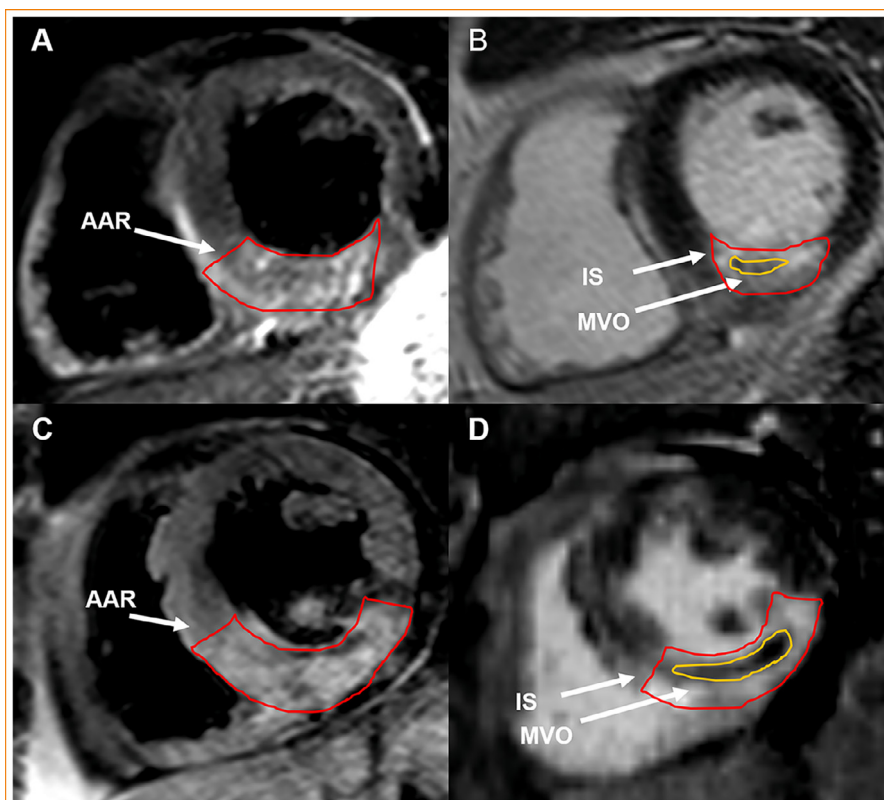


FIGURE 2. Cardiac magnetic resonance images of ST-segment elevation myocardial infarction. (A) AAR (22.4%) in T2-weighted image of a patient in the Age <70 group. (B) IS (13.2%) and MVO (1.23%) in late gadolinium hyperenhancement image of the patient in the Age <70 group. (C) AAR (38.9%) in T2-weighted image of a patient in the Age ≥70 group and (D) IS (20.6%) and MVO (11.15%) in late gadolinium hyperenhancement image of the patient in the Age ≥70 group.

Abbreviations: AAR, area at risk; IS, infarct size; MVO, microvascular obstruction.

CMR imaging analysis

CMR was performed using a 1.5-T scanner (Achieva, Philips Medical Systems, Best, Netherlands). All measurements were taken at Samsung Medical Center-CMR core laboratory using validated software (ARGUS; Siemens Medical System, Erlangen, Germany). Infarct size and extent of MVO were assessed using late gadolinium enhancement images, whereas the AAR was measured on T2-weighted images.¹² Two experienced radiologists who were blinded to patient information measured CMR parameters using the software mentioned above. Endocardial borders were traced after acquiring short-axis images at end-diastole and end-systole. The Simpson algorithm was then used to calculate LV end-diastolic volume, LV end-systolic volume, and LVEF. Infarct size was calculated from summation of the area with delayed hyperenhancement within each segment of the short-axis images. This value was multiplied by slice thickness to cover the entire LV. Endocardial and epicardial borders were planimtered to calculate the myocardial area, and they were then summed to calculate LV myocardial volume using the same method. Infarct size was expressed as a percentage of affected LV myocardial volume.¹² T2-weighted images were used to determine the presence of hemorrhagic infarction.¹³ AAR was

quantified on T2-weighted images using a similar algorithm and was expressed as a percentage of LV myocardial volume affected.¹² MSI was computed as follows: $MSI = (AAR - \text{infarct size}) / AAR \times 100$.¹⁴ Fig. 2 shows representative CMR images of STEMI treated with revascularization.

Statistical analysis

Continuous variables were expressed as mean $\pm$ standard deviation or median when variables lacked a normal distribution. An analysis of continuous variables was performed using Student's t-test or the Wilcoxon rank-sum test. Categorical variables were described as numbers (n) with percentages (%), and differences were analyzed using Pearson χ^2 or Fisher's exact tests. We performed inverse probability of treatment weighting (IPTW) adjustment to reduce treatment selection bias according to treatment strategy and other potential confounding factors. The IPTW method was performed using generalized boosted models for baseline characteristics to evaluate if interactions affected clinical outcomes. Covariates that were either statistically significant on univariable analysis (p value < 0.1) or considered clinically important were included in

TABLE 1. Baseline characteristics.

	Overall population (n = 279)	Age ≥ 70 (n = 52)	Age <70 (n = 227)	p value
Male	225 (80.7)	29 (55.8)	196 (86.3)	<0.001
Body mass index, kg/m ²	24.8 $\pm$ 3.4	23.8 $\pm$ 3.4	25.1 $\pm$ 3.4	0.017
Current smoker	144 (51.6)	12 (23.1)	132 (58.2)	<0.001
Hypertension	125 (44.8)	30 (57.7)	95 (41.9)	0.038
Diabetes mellitus	60 (21.5)	14 (26.9)	46 (20.3)	0.292
Dyslipidemia	50 (17.9)	8 (15.4)	42 (18.5)	0.597
Previous PCI	6 (2.2)	1 (1.9)	5 (2.2)	>0.999
Previous CVA	10 (3.6)	4 (7.7)	6 (2.6)	0.094
LVEF (%)	53.2 $\pm$ 10.8	51.6 $\pm$ 12.3	53.6 $\pm$ 10.4	0.266
^a NT-proBNP, pg/mL	426.1 $\pm$ 1035.8	1055.4 $\pm$ 1986.6	277.5 $\pm$ 543.8	0.015
Peak CK-MB, ng/mL	214.6 $\pm$ 230.3	242.1 $\pm$ 415.5	208.3 $\pm$ 161.5	0.566
Concomitant medications				
Aspirin	277 (99.3)	52 (100.0)	225 (99.1)	>0.999
P2Y12 inhibitors	277 (99.3)	52 (100.0)	225 (99.1)	>0.999
Statins	269 (96.4)	50 (96.2)	219 (96.5)	>0.999
Beta blockers	255 (91.4)	47 (90.4)	208 (91.6)	0.785
ACE inhibitors/ARB	234 (83.9)	39 (75.0)	195 (85.9)	0.054
^b Killip class score ≥ 3	19 (7.7)	4 (8.9)	15 (7.4)	0.732

Data are presented as n (%) or mean $\pm$ standard deviation.

^a Data of NT-proBNP was available for 225 (80.6%) patients.

^b Data of Killip class was available for 248 (88.9%) patients.

Abbreviations: ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker; CK-MB, creatine kinase-myocardial band; CVA, cerebrovascular accident; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

multivariate models to determine the independent predictors of large myocardial infarcts (percent infarct volume $\geq$ mean infarct size in the present study). Analyzed covariates were age, sex, smoking, hypertension, dyslipidemia and multi-vessel disease. All tests were two-tailed, and $p < 0.05$ was considered statistically significant. Statistical analyses were performed using SAS software (Version 9.2; SAS Institute Inc, Cary, NC, USA).

RESULTS

Baseline clinical, angiographic, and procedural characteristics

The 279 patients were sorted into two groups: Age ≥ 70 group (n = 52; 18.6%) and Age <70 group (n = 227; 81.4%) (Fig. 1). Median age of the total study population was 59 years. Compared with the Age <70 group, the Age ≥ 70 group had a higher proportion of males ($p < 0.001$), lower body mass index ($p = 0.017$) and fewer current smokers ($p < 0.001$). There was a higher incidence of hypertension ($p = 0.038$) and higher NT-proBNP ($p = 0.015$) in the Age ≥ 70 group than in the Age <70 group. Other baseline clinical characteristics were not significantly different between the two groups (Table 1). Table 2 shows the angiographic and procedural findings in the Age ≥ 70 and the Age <70 groups. In the Age ≥ 70 group, symptom onset-to-balloon time tended to be longer ($p = 0.059$), and door-to-balloon time was significantly longer than in the Age <70 group ($p = 0.038$). The Age ≥ 70 group had a higher incidence of multi-vessel

disease ($p = 0.015$), but a lower frequency of GPIIb/IIIa inhibitor use ($p = 0.009$) compared with the Age <70 group. The myocardial blush grade was lower ($p < 0.001$), and stent diameter was smaller ($p = 0.031$) in the Age ≥ 70 group than in the Age <70 group, but these differences were minimal. Types of drug-eluting stents were not significantly different between the two groups ($p = 0.189$).

Analysis of CMR findings

CMR was performed a median of 3.3 days (interquartile range: 2.8-4.2) after the index procedure, and the interval from primary PCI to CMR was not significantly different between the two groups. Myocardial infarct size tended to be greater in the Age ≥ 70 group than in the Age <70 group (21.2 $\pm$ 10.2% versus 19.5 $\pm$ 11.1%; $p = 0.072$). The extent of AAR was greater in the Age ≥ 70 group than in the Age <70 group (37.1 $\pm$ 17.7 versus 34.2 $\pm$ 15.3; $p = 0.039$). MSI (42.2 $\pm$ 16.7 versus 44.3 $\pm$ 19.3; $p = 0.166$) and MVO area (4.6 $\pm$ 4.4 versus 4.2 $\pm$ 6.2; $p = 0.402$) were similar between the two groups (Table 3). After performing IPTW adjustment, myocardial infarct size was significantly greater in the Age ≥ 70 group compared with the Age <70 group (22.6 $\pm$ 10.4 versus 19.6 $\pm$ 11.1; $p = 0.001$). The extent of AAR was greater in the Age ≥ 70 group than in the Age <70 group (38.8 $\pm$ 17.8 versus 34.3 $\pm$ 15.3; $p = 0.001$). MSI was lower in the Age ≥ 70 group than in the Age <70 group (40.4 $\pm$ 15.9 versus 44.4 $\pm$ 19.1; $p = 0.007$), and

TABLE 2. Angiographic and procedural characteristics.

	Overall population (n = 279)	Age ≥70 (n = 52)	Age <70 (n = 227)	p value
Times, minute				
Symptom onset-to-balloon time	209.0 ± 136.8	243.3 ± 145.0	201.1 ± 134.0	0.059
Door-to-balloon time	61.9 ± 17.6	66.6 ± 18.0	60.8 ± 17.4	0.038
Infarct-related artery				0.863
Left anterior descending artery	138 (49.5)	26 (50.0)	112 (49.3)	
Left circumflex artery	29 (10.4)	4 (7.7)	25 (11.0)	
Right coronary artery	110 (39.4)	22 (42.3)	88 (38.8)	
Left main artery	2 (0.7)	0 (0.0)	2 (0.9)	
Multi-vessel disease	124 (44.4)	31 (59.6)	93 (41.0)	0.015
TIMI flow grade before PCI				0.899
0	215 (77.1)	39 (75.0)	176 (77.5)	
1	18 (6.5)	4 (7.7)	14 (6.2)	
2 or 3	46 (16.5)	9 (17.3)	37 (16.3)	
Presence of Collateral flow	127 (51.6)	23 (52.3)	104 (51.5)	0.925
Final TIMI flow grade 3 after PCI	264 (94.6)	49 (94.2)	215 (94.7)	0.889
Angiographic no reflow phenomenon	14 (5.0)	0 (0.0)	14 (6.2)	0.080
Myocardial blush grade				<0.001
1	2 (0.8)	1 (2.2)	1 (0.5)	
2	20 (8.1)	11 (24.4)	9 (4.4)	
3	226 (91.1)	33 (73.3)	193 (95.1)	
Aspiration thrombectomy	174 (62.4)	32 (61.5)	142 (62.6)	0.891
Use of GP IIb/IIIa inhibitor	43 (17.3)	2 (4.4)	41 (20.2)	0.009
PCI using Stent	266 (95.3)	48 (92.3)	218 (96.0)	0.272
Number of implanted stents	1.2 ± 0.6	1.3 ± 0.6	1.2 ± 0.6	0.320
Stent diameter, mm	3.2 ± 0.6	3.1 ± 0.4	3.2 ± 0.6	0.031
Stent length, mm	30.1 ± 14.9	32.3 ± 17.0	29.6 ± 14.4	0.321
Types of drug-eluting stents				0.189
Biolimus-eluting stent	28 (11.9)	6 (14.6)	22 (11.3)	
Everolimus-eluting stent	53 (22.5)	4 (9.8)	49 (25.1)	
Zotarolimus-eluting stent	147 (62.3)	29 (70.7)	118 (60.5)	
Other drug-eluting stents	8 (3.4)	2 (4.9%)	6 (3.1%)	

Data are presented as n (%) or mean ± standard deviation.
Abbreviations: GP, glycoprotein; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

TABLE 3. Analysis of cardiac magnetic resonance findings.

	Overall population (n = 279)			IPTW population (n = 559)		
	Age ≥70 (n = 52)	Age <70 (n = 227)	p value	Age ≥70 (n = 280)	Age <70 (n = 279)	p value
Myocardial Infarct size (% of LV)	21.2 ± 10.2	19.5 ± 11.1	0.072	22.6 ± 10.4	19.6 ± 11.1	0.001
Area at risk (% of LV)	37.1 ± 17.7	34.2 ± 15.3	0.039	38.8 ± 17.8	34.3 ± 15.3	0.001
Myocardial salvage index	42.2 ± 16.7	44.3 ± 19.3	0.166	40.4 ± 15.9	44.4 ± 19.1	0.007
MVO area (% of LV)	4.6 ± 4.4	4.2 ± 6.2	0.402	5.0 ± 4.4	4.2 ± 6.2	0.082
Hemorrhagic infarction	27 (52.0)	127 (45.5)	0.127	163 (58.4)	125 (44.8)	0.001
LV end diastolic volume (mL)	136.2 ± 47.9	146.3 ± 32.3	0.003	138.5 ± 47.5	145.9 ± 32.8	0.032
LV end systolic volume (mL)	69.1 ± 50.0	69.5 ± 26.9	0.894	71.7 ± 49.6	69.5 ± 27.4	0.520
LV ejection fraction (%)	52.4 ± 12.1	53.5 ± 9.7	0.248	51.2 ± 11.9	53.4 ± 9.7	0.020
LV stroke volume (mL)	67.1 ± 13.4	76.9 ± 16.4	<0.001	66.8 ± 13.2	76.4 ± 16.5	<0.001
LV cardiac output (L/min)	4.6 ± 0.8	5.2 ± 1.1	<0.001	4.4 ± 0.8	5.2 ± 1.1	<0.001

Data are presented as n (%) or mean ± standard deviation.
Abbreviations: IPTW, Inverse probability of treatment weighting; LV, left ventricle (-ular); MVO, microvascular obstruction.

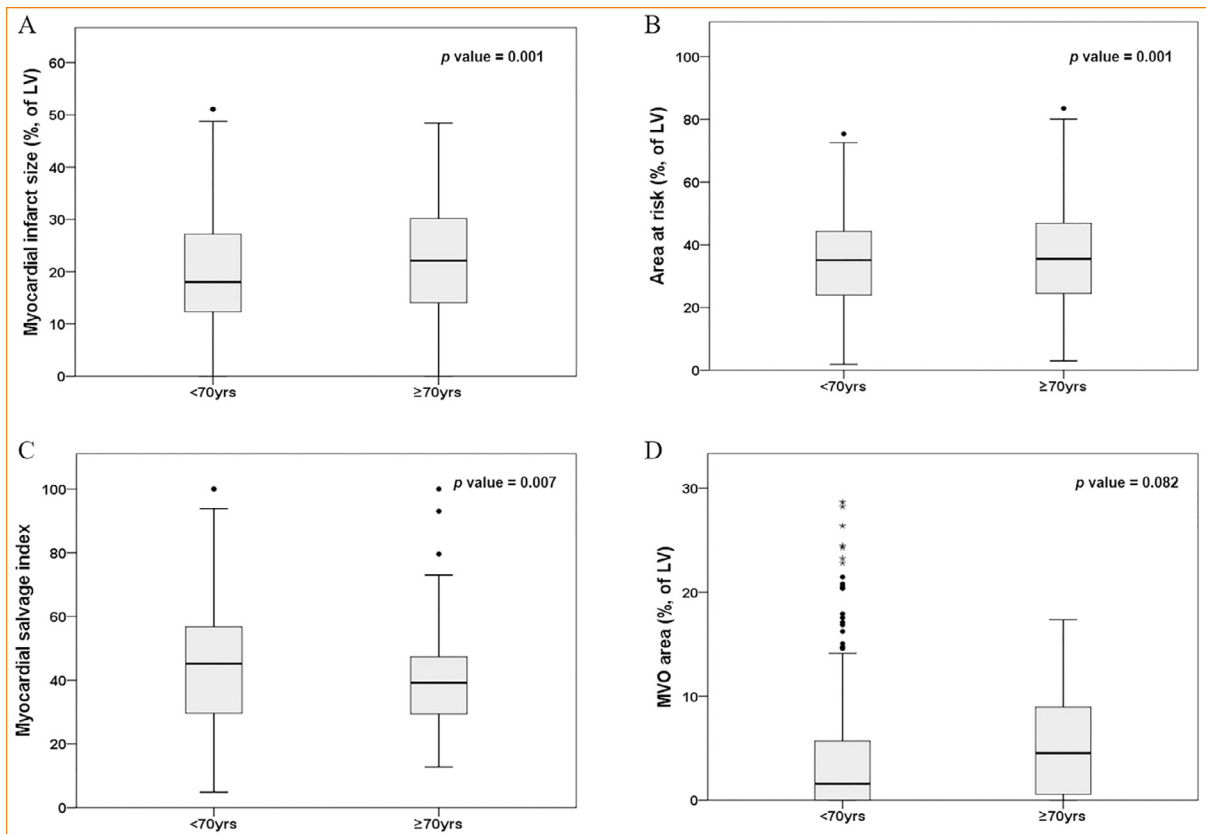


FIGURE 3. Cardiac magnetic resonance findings according to age categories after IPTW analysis. The boxplots show cardiac magnetic resonance data according to age at occurrence of STEMI and (A) myocardial infarct size, (B) extent of AAR, (C) MSI, and (D) MVO area after IPTW adjustment.

Abbreviations: AAR, area at risk; IPTW, inverse probability of treatment weighting; LV, left ventricle; MSI, myocardial salvage index; MVO, microvascular obstruction; STEMI, ST-segment elevation myocardial infarction.

MVO area tended to be greater in the Age ≥ 70 group compared with the Age < 70 group, but these differences were not significant (5.0 ± 4.4 versus 4.2 ± 6.2 ; $p = 0.082$) (Table 3 and Fig. 3).

Predictors of large myocardial infarct

On multivariate logistic regression analysis, independent predictors of large myocardial infarct (percent infarct volume of LV $\geq 20\%$) included age ≥ 70 years (odds ratio [OR] 2.02; 95% confidence interval [CI] 1.02–4.00; $p = 0.044$) and male sex (OR 2.12; 95% CI 1.04–4.31; $p = 0.038$) (Fig. 4).

Sex and myocardial injury in older STEMI patients

We performed subgroup analysis to identify the association between sex and myocardial injury and found no significant difference with respect to myocardial infarct size among female and male subgroups after IPTW adjustment (20.1 ± 11.5 , females versus 20.4 ± 9.9 , males; $p = 0.901$). The extent of AAR (38.0 ± 15.3 versus 35.5 ± 18.2 ; $p = 0.460$), MSI (46.3 ± 21.9 versus 41.9 ± 15.2 ; $p = 0.281$), and MVO

area (4.0 ± 5.1 versus 4.4 ± 4.2 ; $p = 0.685$) were also similar between the sex subgroups (Table 4).

DISCUSSION

We investigated the association between old age and myocardial injury using CMR findings of myocardial and microvascular damage in patients with STEMI. The main finding of our study was that old age is associated with greater myocardial infarct size, AAR and MVO area on CMR in STEMI patients undergoing primary PCI. This association between old age and progressed myocardial injury remained after IPTW-adjusted analysis. In subgroup analysis of sex-specific effects for older STEMI patients, myocardial injury was not significantly different between females and males. To the best of our knowledge, this is a unique study evaluating the clinical significance of old age on myocardial injury assessed via CMR in older STEMI patients. Our findings corresponded well with those of earlier studies that established an association between old age and adverse clinical outcomes after STEMI.^{15,16} Indeed, the results of our study may provide

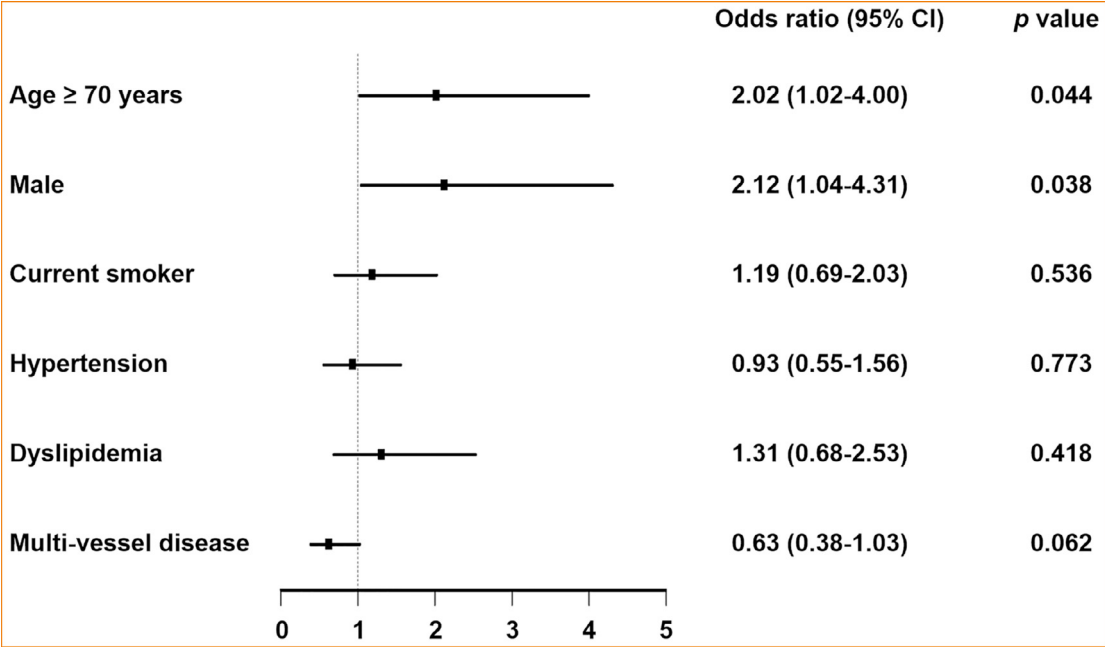


FIGURE 4. Predictors of large myocardial infarct. Forest plots show the results of multivariable analysis of predictors of large myocardial infarct.

TABLE 4. Subgroup analysis of sex differences in cardiac magnetic resonance findings after inverse probability of treatment weighting adjustment.

	IPTW population (n = 100)	Female (n = 61)	Male (n = 39)	p value
Myocardial Infarct size (% of LV)	20.2 ± 10.8	20.1 ± 11.5	20.4 ± 9.9	0.901
Area at risk (% of LV)	37.0 ± 16.4	38.0 ± 15.3	35.5 ± 18.2	0.460
Myocardial salvage index	44.6 ± 19.6	46.3 ± 21.9	41.9 ± 15.2	0.281
MVO area (% of LV)	4.1 ± 4.8	4.0 ± 5.1	4.4 ± 4.2	0.685

Data are presented as n (%) or mean ± standard deviation.
Abbreviations: IPTW, Inverse probability of treatment weighting; LV, left ventricle (-ular); MVO, microvascular obstruction.

a causal mechanism linking old age to adverse clinical outcomes after STEMI.

Elderly patients with STEMI tend to present frequently with symptoms other than chest pain, which may divert diagnostic suspicion away from acute MI.¹⁷ Brieger et al. reported that patients with acute coronary ischemia in the absence of chest pain were older than those with chest pain and experienced higher mortality.¹⁸ The more frequent presentation of atypical chest pain in older STEMI patients could contribute to the longer symptom-to-door- and door-to-balloon times, resulting in a higher mortality rate.¹⁵ Moreover, compared to younger patients, older patients are less likely to undergo invasive treatment for acute MI because of their functional and socioeconomic status and complications arising from coronary revascularization.¹⁹ Lonborg et al.⁴ analyzed the prognostic importance of infarct size in STEMI patients and reported that old age could be one predictor of poor clinical outcomes, but they were not able to elucidate the underlying mechanisms.

CMR is uniquely positioned to comprehensively evaluate the morphological, functional, and microvascular

sequelae of postinfarct myocardium.⁶ Therefore, we evaluated the association of old age with the prognostic pathophysiology of myocardial ischemia by using CMR data in STEMI patients. We identified old age as a negative predictor of large myocardial infarct and found that older patients suffering from STEMI was associated with extensive myocardial and microvascular damage despite prompt revascularization. Our results provide substantial support to the notion that old age is a reasonable and accurate surrogate for increased myocardial injury in patients with acute MI.

Interestingly, acute MI occurs more often in men than in women below the age of 60 years, but among the older aged 70 or over, women represent the majority of patients.²⁰ Premenopausal women have fewer underlying diseases known as cardiovascular risk factors including hypertension, and hyperlipidemia compared to similarly aged men, which is attributed to cardioprotective effects of estrogen such as vasodilatation, but this reverses in older women and age-matched men.^{20,21} Nevertheless, there have been a small number of studies on prognosis according to sex in older patients with

ischemic heart disease and there is still debate regarding whether outcomes are worse in women, with a few studies reporting that poorer outcomes are related to older age and more comorbidities in women suffering from acute MI.^{22,23} Zhang et al.²⁴ reported that among patients with non-STEMI, women ≥ 70 years old had lower in-hospital mortality than their male counterparts, but this tendency was not observed in patients with STEMI. Cenko et al.¹⁶ showed no significant association between sex and 30-day mortality in STEMI patient ≥ 75 years old. The reason why previous studies have reported these heterogeneous clinical outcomes was presumed to be because estrogen exhibits cardioprotective effects in a complex manner that requires further research.²⁵ However, no previous study proved causality or identified the underlying pathophysiological mechanisms. In subgroup analysis, we investigated the relationship between sex and myocardial injury assessed by CMR in older STEMI patients treated with prompt primary PCI, and we found that myocardial infarct size, the extent of AAR, MSI, and MVO area were similar between men and women aged ≥ 70 years. Our findings suggest that sex differences alone are not a poor prognostic factor in older patients with STEMI. Further larger-scale investigations of the clinical implications of these findings should be considered.

Study limitations

This study has several limitations. First, it was non-randomized, retrospective, and observational, and thus confounding factors may have significantly affected the results. Second, since patients undergoing CMR may be clinically stable with modest myocardial injury and only patients available for CMR were included, intervals from primary PCI to CMR were heterogeneous; moreover, older patients with comorbidities were unlikely to have undergone CMR, which may limit our study results. Third, we included patients treated with primary PCI within 12 hours after symptom onset. Considering the tendency of older patients to have longer symptom onset-to-balloon time leading to a larger infarction and worse outcomes, the 12-hour inclusion criterion may be too long to assess the true impact of primary PCI alone. Moreover, in terms of primary PCI as an inclusion criterion, patients with acute occlusion of left circumflex coronary arteries who often present with non-diagnostic ECG would not have received primary PCI, resulting in a relatively lower proportion of left circumflex coronary arteries as the culprit vessel and, angiographic findings regarding coronary artery dominance or the number of non-culprit lesions that can affect infarct size were not considered in our analysis, which may have affected our results. Finally, due to the retrospective nature of our registry, we could not thoroughly identify the detailed data regarding clinical outcomes including mortality, and the procedures, such as whether complete revascularization was performed during the index period.

CONCLUSIONS

In the present study, older patients had greater myocardial infarct size, less myocardial salvage, and greater extent of myocardial edema despite appropriate primary PCI for STEMI compared to younger patients. Based on our results, old age could be a poor prognostic factor in patients with STEMI and close attention needs to be paid to older STEMI patients after primary PCI. Further research on the potential therapeutic implications of these findings should be conducted.

AUTHOR CONTRIBUTIONS

Conceptualization: Oh J.H. and Chun W.J.; Data curation: Park Y.H. and Kim J.; Formal analysis: Park I.H. and Cho H.K.; Investigation: Choi S.-H. and Lee S.-C.; Methodology: Gwon H.-C. and Jang W.J.; Software: Choe Y.H.; Validation: Cho H.K., Song Y.B. and Hahn J.-Y.; Supervision: Oh J.H. and Chun W.J.; Visualization: Park Y.H., Choe Y.H. and Kim J.; Writing – original draft: Park I.H., Cho H.K. and Jang W.J.; Writing – review & editing: Park I.H. and Jang W.J.

CONFLICTS OF INTEREST

None.

SOURCE OF FUNDING

None.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.amjms.2021.06.023>.

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