

Sex-Related Differences in Short- and Long-Term Outcome among Young and Middle-Aged Patients for ST-Segment Elevation Myocardial Infarction Underwent Percutaneous Coronary Intervention

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Abstract

Background: Females with ST-segment elevation myocardial infarction (STEMI) have higher in-hospital and short-term mortality rates compared with males in China, suggesting that a sex disparity exists. The age of onset of STEMI is ahead of time and tends to be younger. However, there are relatively little data on the significance of sex on prognosis for long-term outcomes for adult patients with STEMI after percutaneous coronary intervention (PCI) in China. This study sought to analyze the sex differences in 30-day, 1-year, and long-term net adverse clinical events (NACEs) in Chinese adult patients with STEMI after PCI.

Methods: This study retrospectively analyzed 1920 consecutive STEMI patients (age ≤ 60 years) treated with PCI from January 01, 2006, to December 31, 2012. A propensity score analysis between males and females was performed to adjust for differences in baseline characteristics and comorbidities. The primary endpoint was the incidence of 3-year NACE. Survival curves were constructed with Kaplan-Meier estimates and compared by log-rank tests between the two groups. Multivariate analysis was performed using a Cox proportional hazards model for 3-year NACE.

Results: Compared with males, females had higher risk profiles associated with old age, longer prehospital delay at the onset of STEMI, hypertension, diabetes mellitus, and chronic kidney disease, and a higher Killip class (≥ 3), with more multivessel diseases ($P < 0.05$). The female group had a higher levels of low-density lipoprotein ($2.72 [2.27, 3.29]$ vs. $2.53 [2.12, 3.00]$, $P < 0.001$), high-density lipoprotein ($1.43 [1.23, 1.71]$ vs. $1.36 [1.11, 1.63]$, $P = 0.003$), total cholesterol (4.98 ± 1.10 vs. 4.70 ± 1.15 , $t = -3.508$, $P < 0.001$), and estimated glomerular filtration rate (103.12 ± 22.22 vs. 87.55 ± 18.03 , $t = -11.834$, $P < 0.001$) than the male group. In the propensity-matched analysis, being female was associated with a higher risk for 3-year NACE and major adverse cardiac or cerebral events compared with males. In the multivariate model, female gender (hazard ratio [HR]: 2.557, 95% confidence interval [CI]: 1.415–4.620, $P = 0.002$), hypertension (HR: 2.017, 95% CI: 1.138–3.576, $P = 0.016$), and family history of coronary heart disease (HR: 2.256, 95% CI: 1.115–4.566, $P = 0.024$) were independent risk factors for NACE. The number of stents (HR: 0.625, 95% CI: 0.437–0.894, $P = 0.010$) was independent protective factors of NACE.

Conclusions: Females with STEMI undergoing PCI have a significantly higher risk for 3-year NACE compared with males in this population. Sex differences appear to be a risk factor and present diagnostic challenges for clinicians.

Key words: Percutaneous Coronary Intervention; Prognosis; Sex; ST-Segment Elevation Myocardial Infarction

INTRODUCTION

Despite a significant decrease in mortality associated with cardiovascular disease in developed countries over the last decade,^[1,2] acute myocardial infarction (AMI) continues to be a major cause of morbidity and mortality.^[3,4] ST-segment elevation myocardial infarction (STEMI) constitutes more than 80% of MI patients in China,^[5] percutaneous coronary

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intervention (PCI) is the established acute reperfusion therapy for STEMI.^[6] STEMI remains the major contributor to death in females who suffer from cardiovascular disease at an older age compared with males,^[7-9] with females being reported to have more risk factors.^[10,11] However, these studies have only examined hospital mortality and short-term outcomes. Furthermore, the age of onset of STEMI is ahead of time and tends to be younger.^[12] Therefore, there is little evidence on the effect of sex on prognosis following long-term STEMI in developing countries such as China. Research is required to understand whether sex-dependent differences influence the long-term prognosis of Chinese patients with STEMI undergoing PCI in adults. This study analyzed differences between the sexes for 30-day, 1-year, and 3-year net adverse clinical events (NACEs) in 1920 Chinese adult patients (≤ 60 years old) with STEMI undergoing PCI.

METHODS

Ethical approval

The study was conducted in accordance with the *Declaration of Helsinki* and was approved by the local ethics committee of the hospital. As a retrospective study and data analysis was performed anonymously, this study was exempt from the informed consent from patients.

Study population and study design

This study retrospectively analyzed all consecutive patients admitted to the General Hospital of Shenyang Military Region with acute STEMI from January 01, 2006, to December 31, 2012, who underwent acute coronary angiography with the intention of PCI. Briefly, eligible patients aged 18–60 years had abnormal cardiac biomarkers, with at least one biomarker above the 99th percentile of the upper reference limit within 24 h of admission. Patients must have presented directly to the enrolling PCI site within the first 24 h of presentation to ensure that primary clinical decision-making occurred at the enrolling site. We included patients with STEMI ($n = 3179$) that was confirmed by discharge diagnosis and electrocardiogram results. We excluded patients who were missing hospitalization data ($n = 90$), older than 60 years ($n = 970$), without PCI ($n = 186$), and lost to follow-up ($n = 13$), resulting in a final cohort of 1920 patients (1693 males and 227 females).

Data collection

Baseline characteristics, including demographic, treatment, and clinically relevant comorbidities, were collected by medical chart abstraction and standardized in-person interviews. Demographic characteristics included sex, age, smoking, previous medical history of diabetes, hyperlipidemia, hypertension, family history of coronary heart disease (CHD), diabetes, hypertension, and stroke. Information on prior myocardial infarction, PCI, peripheral artery disease, stroke, and chronic kidney disease was also obtained. Data regarding medication management included the use of aspirin, clopidogrel,

β -blockers, angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists, diuretics, statins, and nitrates. Echocardiography was ordered during hospitalization. All patients were initially administered with a loading dose of 300 mg aspirin and 600 mg clopidogrel before all procedures, followed by 75 mg clopidogrel per day for 3–12 months, along with 100 mg aspirin daily indefinitely, and treated with PCI by two experienced interventional cardiologists. All definitions were in accordance with the American College of Cardiology. Data were extracted by review of records from the emergency room, catheterization laboratory, and intensive care. All patients gave their intervention informed consent.

Follow-up and endpoints

All the 1920 STEMI patients were followed by telephone or hospital visits at 30 days, 6 months, 12 months, and 3 years after the index procedure and every year thereafter. Outcome of patients was retrieved from the interhospital computer system or by telephone interview. The primary observational outcome for the study was the occurrence of long-term NACEs, defined as the composite of any bleeding or major adverse cardiac or cerebral events (MACCEs), including all-cause death, reinfarction, and clinically indicated target vessel revascularization (TVR) or stroke. Long-term NACE was defined as 3 years during the observation period. Secondary observational outcomes included all separate components of the primary outcome, at 30-day, 1-year, and 3-year MACCE as well as bleeding and 30-day and 1-year NACE.

Definitions

STEMI was diagnosed if there was evidence of persistent chest pain for longer than 30 min and electrocardiogram changes with ST elevation >2 mm in at least two precordial leads or >1 mm in the limb leads or a new left bundle branch block. Successful primary PCI was $<30\%$ residual stenosis of culprit lesions and three coronary flows, assessed visually. Multivessel disease was at least one additional $\geq 70\%$ stenosis in a major coronary vessel besides the culprit lesion. Cardiac death included any death due to an evident cardiac cause, any death related to PCI, or death from an unknown reason. Revascularization of the target vessel was defined as stenosis of any target vessel above 50% of the diameter of the vessel based on quantitative coronary angiography in the presence of objective evidence of ischemia from noninvasive or invasive testing or symptoms. The diagnosis of reinfarction was based on the evidence of new or presumably new ST-segment elevation in two consecutive leads and an increase in biochemical markers of myocardial necrosis.^[13] Stroke was defined as the rapid onset of a new, persistent, neurologic deficit lasting at least 24 h (or resulting in death before 24 h). To compare with other trials using Academic Research Consortium (ARC) MI definitions, we also adjudicated MI data according to ARC definitions.^[14]

Statistical analysis

Continuous and categorical variables are presented as mean \pm standard deviation (SD) and percentages. All data were compared using parametric Student's *t*-tests, Chi-square or Fisher's exact tests, and Wilcoxon tests, as appropriate. Propensity score matching between males and females was performed as a 1:1 propensity score-matching analysis using the nearest-neighbor matching within a caliper of 0.1 SD of pooled propensity scores. Cumulative incidence rates of unadjusted NACE were evaluated using the Kaplan–Meier method, and log-rank tests were used to calculate differences between females and males. Cox regression model analysis was used to identify factors associated with adjusted 3-year NACE. Multivariate analysis for 3-year NACE was performed using Cox proportional hazards regression modeling. The results are presented as adjusted hazard ratios (HRs) with a 95% confidence interval (CI). All analyses were performed using SPSS 22.0 software for Windows (IBM Corp., Armonk, NY, USA). *P* values were two sided and *P* < 0.05 was considered statistically significant.

RESULTS

Study population and baseline characteristics

Of 1920 patients, 227 (11.82%) were female and 1693 (88.18%) were male. Their baseline characteristics are presented in Table 1.1. Compared with males, females were slightly older (mean age 57.47 ± 6.14 years vs. 51.87 ± 8.30 years, $t = -12.317$, $P < 0.001$) and were more likely to have diabetes mellitus (25.55% in females vs. 17.25% in males, $\chi^2 = 9.258$, $P = 0.002$), hypertension (56.83% vs. 45.48%, $\chi^2 = 10.350$, $P = 0.001$), and chronic kidney disease (2.20% vs. 0.65%, $\chi^2 = 4.113$, $P = 0.043$). More males had a history of smoking (76.90% in males vs. 28.19% in females, $\chi^2 = 231.355$, $P < 0.001$) and were less likely to have hyperlipidemia (30.01% vs. 30.84%, $\chi^2 = 0.066$, $P = 0.798$). After propensity matching, the demographics and clinical parameters were well balanced between the groups; there were 290 propensity-matched patients [145 females and 145 males, Table 1.2].

For biochemistry, compared with males, females had higher average low-density lipoprotein cholesterol (2.72 [2.27, 3.29] vs. 2.53 [2.12, 3.00], $P < 0.001$), high-density lipoprotein (1.43 [1.23, 1.71] vs. 1.36 [1.11, 1.63], $P = 0.003$), total cholesterol (4.98 ± 1.10 vs. 4.70 ± 1.15 , $t = -3.508$, $P < 0.001$), and estimated glomerular filtration rate (103.12 ± 22.22 vs. 87.55 ± 18.03 , $t = -11.834$, $P < 0.001$) whereas hemoglobin (144.57 ± 15.83 vs. 125.73 ± 14.51 , $t = 16.894$, $P < 0.001$) in males was significantly higher than females. There were no differences in pharmacologic management during hospitalization between sexes. Females had a higher Killip class (≥ 3) than males (8.37% vs. 4.96%, $\chi^2 = 8.578$, $P = 0.035$). Females had significantly longer time from symptom onset to treatment (4.50 [3.00, 7.00] h vs. 7 h [4.00, 10.00],

$P < 0.001$). In angiographic characteristics and procedural results, females had a worse risk profile than males, with more multivessel diseases (80.17% vs. 67.92%, $\chi^2 = 18.284$, $P < 0.001$). Males had a relatively larger reference vessel diameter ($[3.16 \pm 1.17]$ mm vs. $[3.01 \pm 0.38]$ mm, $t = 1.942$, $P = 0.052$). In propensity score-adjusted data, females had also significantly longer time from symptom onset to treatment (4 [2.00, 5.00] h vs. 7 [4.25, 11.00] h, $P < 0.001$), and other clinical characteristics were no significant differences between females and males [Table 1.2].

Survival analysis of clinical outcomes

The results of 30-day, 1-year, and 3-year clinical outcomes for both groups are shown in Table 2.1. The incidence of 30-day NACE occurred in 12 (5.28%) female patients and 35 (2.07%) male patients (log rank $P = 0.002$). The rates of MACCE and myocardial infarction for females were significantly higher than males (females vs. males 4.84% vs. 1.59%, log rank $P = 0.001$; 1.32% vs. 0.12%, log rank $P = 0.001$). After propensity-matched adjustment, 30-day NACE was similar in both groups [Table 2.2].

At the 1-year follow-up, females continued to show increases in NACE compared with males (12.78% vs. 7.09%, log rank $P = 0.002$). In addition, females had a higher incidence of MACCE (11.89% vs. 5.91%, log rank $P = 0.001$), stroke (1.32% vs. 0.30%, log rank $P = 0.024$), and TVR (4.84% vs. 2.48%, log rank $P = 0.029$) compared with males. In the propensity-matched analysis, sex was no longer associated with a higher 1-year NACE [16.55% vs. 11.03%, log rank $P = 0.164$, Table 2.2].

Females had a significantly higher unadjusted 3-year NACE (21.59% vs. 11.40%, log rank $P < 0.001$) and a higher incidence of MACCE (18.94% vs. 9.92%, log rank $P < 0.001$), stroke (1.76% vs. 0.41%, log rank $P = 0.011$), and TVR (8.37% vs. 4.31%, log rank $P = 0.015$) compared with males. There were no differences in all-cause death (females vs. males) (4.85 vs. 3.43%, log rank $P = 0.253$), myocardial infarction (3.52% vs. 1.83%, log rank $P = 0.089$), and bleeding [3.08% vs. 1.42%, log rank $P = 0.063$, Table 2.1]. In the propensity-matched analysis, the 3-year incidence of NACE (26.21% vs. 13.79%, log rank $P = 0.008$) and MACCE (22.76% vs. 10.34%, log rank $P = 0.005$) were significantly higher in females than males. There were no significant differences between groups for all-cause death (females vs. males) (6.90% vs. 2.76%, log rank $P = 0.100$), cardiac death (5.52% vs. 2.07%, log rank $P = 0.125$), myocardial infarction (4.83% vs. 1.38%, log rank $P = 0.091$), stroke (2.07% vs. 0, log rank $P = 0.082$), TVR (8.97% vs. 6.21%, log rank $P = 0.400$), and bleeding [3.45% vs. 3.45%, log rank $P = 1.000$; Figure 1 and Table 2.2].

Multivariate analysis

Cox regression analysis with propensity matching showed that female (HR: 2.557, 95% CI: 1.415–4.620, $P = 0.002$), hypertension (HR: 2.017, 95% CI: 1.138–3.576, $P = 0.016$), and family history of CHD (HR: 2.256,

Table 1.1: Baseline characteristics of STEMI patients treated with PCI according to gender

Characteristics	Males (n = 1693)	Females (n = 227)	Statistics	P
Demographics				
Age (years)	51.87 ± 8.30	57.47 ± 6.14	-12.317*	<0.001
Measurements				
SBP (mmHg)	126.11 ± 37.70	126.22 ± 20.32	-0.041*	0.967
DBP (mmHg)	77.27 ± 14.44	76.67 ± 13.73	0.596*	0.551
Heart rate (beats/min)	76.80 ± 14.89	77.97 ± 15.14	-1.114*	0.266
Medical history				
Hypertension	770 (45.48)	129 (56.83)	10.350†	0.001
Diabetes mellitus	292 (17.25)	58 (25.55)	9.258†	0.002
Hyperlipidemia	508 (30.01)	70 (30.84)	0.066†	0.798
Peripheral artery disease	17 (1.00)	3 (1.32)	0.009†	0.925
Stroke	164 (9.69)	24 (10.57)	0.178†	0.673
Chronic kidney disease	11 (0.65)	5 (2.20)	4.113†	0.043
Smoking	1302 (76.90)	64 (28.19)	231.355†	<0.001
Prior history				
Prior MI	153 (9.04)	18 (7.93)	0.303†	0.582
Prior PCI	121 (7.15)	10 (4.41)	2.367†	0.124
Family history				
Family history of CHD	171 (10.10)	21 (9.25)	0.160†	0.464
Family history of hypertension	157 (9.27)	19 (8.37)	0.196†	0.658
Family history of DM	53 (3.13)	8 (3.52)	0.101†	0.751
Family history of stroke	53 (3.13)	12 (5.29)	2.844†	0.092
Medication				
Aspirin	1691 (99.88)	227 (100.00)	0.000†	1.000
Clopidogrel	1690 (99.82)	226 (99.56)	0.002†	0.967
ACEI/ARBs	1223 (72.24)	161 (70.93)	0.172†	0.679
Beta-blocker	1404 (82.93)	185 (81.50)	0.288†	0.592
Nitrates	1422 (83.99)	193 (85.02)	0.159†	0.690
Statin	1298 (76.67)	167 (73.57)	1.064†	0.302
Diuretic	901 (53.22)	136 (59.91)	3.610†	0.057
Laboratory				
Hemoglobin (g/L)	144.57 ± 15.83	125.73 ± 14.51	16.894*	<0.001
Triglyceride (mmol/L)	1.63 (1.21, 2.36)	1.80 (1.21, 2.49)		0.110
LDL (mmol/L)	2.53 (2.12, 3.00)	2.72 (2.27, 3.29)		<0.001
HDL (mmol/L)	1.36 (1.11, 1.63)	1.43 (1.23, 1.71)		0.003
Total cholesterol (mmol/L)	4.70 ± 1.15	4.98 ± 1.10	-3.508*	<0.001
eGFR (ml/min)	87.55 ± 18.03	103.12 ± 22.22	-11.834*	<0.001
Killip class				
1	1391 (82.16)	169 (74.45)	8.578†	0.035
2	218 (12.88)	39 (17.18)		
3	39 (2.30)	9 (3.96)		
4	45 (2.66)	10 (4.41)		
Symptom onset time (h)	4.50 (3.00, 7.00)	7.00 (4.00, 10.00)		<0.001
Angiographic characteristics				
Diseased vessels				
Single vessel disease	543 (32.07)	45 (19.82)	18.284†	<0.001
Double vessel disease	587 (34.67)	79 (34.80)		
Three vessel disease	563 (33.25)	103 (45.37)		
Number of stents	1.00 (1.00, 2.00)	1.00 (1.00, 2.00)		0.189
Average stent diameter (mm)	3.16 ± 1.17	3.01 ± 0.38	1.942*	0.052
Average stent length (mm)	26.56 ± 5.91	26.03 ± 5.33	1.274*	0.203

Data are presented as mean ± SD, n (%) or median (25th, 75th). **t* values; † χ^2 values. SD: Standard deviation; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CHD: Coronary heart disease; DM: Diabetes mellitus; ACEI: Angiotensin-converting enzyme inhibitors; ARB: Angiotensin II receptor blockers; LDL: Low-density lipoprotein-cholesterol; HDL: High-density lipoprotein-cholesterol; eGFR: Estimated glomerular filtration rate; STEMI: ST-elevation myocardial infarction.

95% CI: 1.115–4.566, *P* = 0.024) were independent risk factors for NACE. The number of stents (*HR*: 0.625, 95%

CI: 0.437–0.894, *P* = 0.010) was independent protective factors of NACE [Table 3].

Table 1.2: Baseline characteristics of STEMI patients treated with PCI according to gender by propensity score matching

Characteristics	Males (n = 145)	Females (n = 145)	Statistics	P
Demographics				
Age (years)	54.97 ± 7.54	56.40 ± 6.65	-1.717*	0.087
Measurements				
SBP (mmHg)	127.32 ± 21.48	125.50 ± 20.76	0.734*	0.464
DBP (mmHg)	76.27 ± 14.13	77.02 ± 14.16	-0.453*	0.651
Heart rate (beats/min)	77.00 ± 15.38	77.57 ± 15.20	-0.319*	0.750
Medical history				
Hypertension	74 (51.03)	78 (53.79)	0.221 [†]	0.638
Diabetes mellitus	28 (19.31)	35 (24.14)	0.994 [†]	0.319
Hyperlipidemia	40 (27.59)	46 (31.72)	0.595 [†]	0.442
Peripheral artery disease	2 (1.38)	0	0.503 [†]	0.478
Stroke	19 (13.10)	13 (8.97)	1.265 [†]	0.261
Chronic kidney disease	2 (1.38)	3 (2.07)	0.000 [†]	1.000
Smoking	62 (42.76)	57 (39.31)	0.356 [†]	0.551
Prior history				
Prior MI	9 (6.21)	11 (7.59)	0.215 [†]	0.643
Prior PCI	8 (5.52)	6 (4.14)	0.300 [†]	0.584
Family history				
Family history of CHD	13 (8.97)	13 (8.97)	0.000 [†]	1.000
Family history of hypertension	14 (9.66)	12 (8.28)	0.169 [†]	0.681
Family history of DM	4 (2.76)	4 (2.76)	0.000 [†]	1.000
Family history of stroke	9 (6.21)	6 (4.14)	0.633 [†]	0.426
Medication				
Aspirin	145 (100.00)	145 (100.00)	0.000 [†]	1.000
Clopidogrel	145 (100.00)	144 (99.31)	0.000 [†]	1.000
ACEI/ARBs	89 (61.38)	104 (71.72)	3.485 [†]	0.062
Beta-blocker	122 (84.14)	115 (79.31)	1.131 [†]	0.288
Nitrates	130 (89.66)	124 (85.52)	1.142 [†]	0.285
Statin	100 (68.97)	105 (72.41)	0.416 [†]	0.519
Diuretic	84 (57.93)	81 (55.86)	0.127 [†]	0.722
Laboratory				
Hemoglobin (g/L)	130.48 ± 14.84	129.20 ± 14.52	0.744*	0.457
Triglyceride (mmol/L)	1.40 (1.04, 2.07)	1.80 (1.21, 2.49)		0.069
LDL (mmol/L)	2.58 (2.18, 3.18)	2.72 (2.27, 3.29)		0.138
HDL (mmol/L)	1.41 (1.15, 1.66)	1.43 (1.23, 1.71)		0.367
Total cholesterol (mmol/L)	4.78 ± 1.26	4.86 ± 1.05	-0.610*	0.542
eGFR (ml/min)	92.69 ± 22.36	96.72 ± 20.64	-1.595*	0.112
Killip class				
1	111 (76.55)	109 (75.17)	0.110 [†]	0.979
2	19 (13.10)	20 (13.79)		
3	7 (4.83)	8 (5.52)		
4	8 (5.52)	8 (5.52)		
Symptom onset time (h)	4.00 (2.00, 5.50)	7.00 (4.25, 11.00)		<0.001
Angiographic characteristics				
Diseased vessels				
Single vessel disease	29 (20.00)	33 (22.76)	0.918 [†]	0.632
Double vessel disease	48 (33.10)	52 (35.86)		
Three vessel disease	68 (46.90)	60 (41.38)		
Number of stents	1.00 (1.00, 2.00)	1.00 (1.00, 2.00)		0.391
Average stent diameter (mm)	3.26 ± 0.47	3.18 ± 0.45	1.405*	0.161
Average stent length (mm)	26.30 ± 5.49	26.25 ± 5.29	0.076*	0.939

Data are presented as mean ± SD, n (%) or median (25th, 75th). **t* values; [†] χ^2 values. SD: Standard deviation; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CHD: Coronary heart disease; DM: Diabetes mellitus; ACEI: Angiotensin-converting enzyme inhibitors; ARB: Angiotensin II receptor blockers; LDL: Low-density lipoprotein-cholesterol; HDL: High-density lipoprotein-cholesterol; eGFR: Estimated glomerular filtration rate; STEMI: ST-elevation myocardial infarction.

Table 2.1: Unadjusted outcomes of STEMI patients treated with PCI according to gender

Characteristics	Males (n = 1693)	Females (n = 227)	Statistics	P*
30-day outcomes				
NACE	35 (2.07)	12 (5.28)	9.142	0.002
MACCE	27 (1.59)	11 (4.84)	11.476	0.001
All-cause death	14 (0.83)	5 (2.20)	3.791	0.052
Cardiac death	14 (0.83)	4 (1.76)	1.858	0.173
All MI	2 (0.12)	3 (1.32)	11.160	0.001
Stroke	1 (0.06)	1 (0.44)	2.798	0.094
Target vessel revascularization	10 (0.59)	2 (0.88)	0.427	0.513
Bleeding	8 (0.47)	1 (0.44)	0.005	0.943
1-year outcomes				
NACE	120 (7.09)	29 (12.78)	9.456	0.002
MACCE	100 (5.91)	27 (11.89)	11.794	0.001
All-cause death	35 (2.07)	9 (3.96)	3.250	0.071
Cardiac death	31 (1.83)	7 (3.08)	1.657	0.198
All MI	18 (1.06)	4 (1.76)	0.881	0.348
Stroke	5 (0.30)	3 (1.32)	5.104	0.024
Target vessel revascularization	42 (2.48)	11 (4.84)	4.747	0.029
Bleeding	20 (1.18)	2 (0.88)	0.161	0.689
3-year outcomes				
NACE	193 (11.40)	49 (21.59)	19.258	<0.001
MACCE	168 (9.92)	43 (18.94)	16.960	<0.001
All-cause death	58 (3.43)	11 (4.85)	1.035	0.253
Cardiac death	44 (2.60)	10 (4.41)	2.608	0.106
All MI	31 (1.83)	8 (3.52)	2.899	0.089
Stroke	7 (0.41)	4 (1.76)	6.433	0.011
Target vessel revascularization	73 (4.31)	19 (8.37)	5.896	0.015
Bleeding	24 (1.42)	7 (3.08)	3.453	0.063

Data are presented as *n* (%). *Log-rank tests. STEMI: ST-elevation myocardial infarction; PCI: Percutaneous coronary intervention; MACCE: Major adverse cardiac and cerebrovascular event; NACE: Net adverse clinical event; MI: Myocardial infarction.

DISCUSSION

We retrospectively analyzed adult females and males with STEMI who underwent PCI. Females were found to have higher 30-day and long-term unadjusted outcomes. However, in a propensity-matched analysis, no differences were found between females and males for 30-day outcomes. There was, however, an apparent difference in long-term outcomes.

For the Chinese population, most studies aim to study the results of short-term follow-up of different gender.^[15-17] Our findings extend the previous literature on possible gender differences in short- and long-term outcomes in patients undergoing PCI in China. This population is large, and is a potentially different patient population from other countries, as China is preparing to embark on national efforts to improve the quality of AMI care.

Some differences between developed countries and China exist. Females in developed countries with STEMI undergoing PCI have a significantly increased risk for short-term NACE.^[18] In the present study of Chinese patients, the short-term risk was not found to be different. Based on the current data, and on our knowledge of gender differences in STEMI, there are several potential explanations for the difference of short-term risk in adult women compared with similarly aged men. Females with STEMI tended to

be older, had more comorbidities including hypertension, diabetes, chronic kidney disease, and a higher Killip class, and more diseased vessels.^[19] Furthermore, sex differences in NACE became more pronounced by adjusted age and clinical characteristics,^[14] a finding that became attenuated and nearly disappeared. When the age of two groups of males and females is similar to the comorbidities, the effect of estrogen is highlighted. The main cause of this difference is the direct protective effect of hormone on the coronary artery and its impact on the risk factors of cardiovascular disease. Protections of estrogens on the coronary artery system include accelerating endothelial cell growth, inhibiting the migration and proliferation of smooth-muscle cells, and influencing the bioavailability of endothelial-derived nitric oxide.^[20] Other reasons for this international difference include different patient populations (≤ 60 years old) and type of AMI. This difference between previous studies and the current study requires further investigation.

For long-term NACE, females with STEMI after PCI had higher rates of NACE than males when developed countries were compared with China. After adjustment for confounders, gender was an important risk factor for STEMI with PCI and other secondary risk factors, such as hypertension and family history of CHD. There are several potential explanations for the higher risk for adverse

Table 2.2: Adjusted outcomes of STEMI patients treated with PCI according to gender by propensity score matching

Characteristics	Males (n = 145)	Females (n = 145)	Statistics	P*
30-day outcomes				
NACE	7 (4.83)	10 (6.90)	0.519	0.471
MACCE	5 (3.45)	9 (6.21)	1.105	0.293
All-cause death	1 (0.69)	5 (3.45)	2.670	0.102
Cardiac death	1 (0.69)	4 (2.76)	1.808	0.179
All MI	0	3 (2.07)	3.021	0.082
Stroke	0	1 (0.69)	1.000	0.317
Target vessel revascularization	4 (2.76)	0	4.042	0.044
Bleeding	2 (1.38)	1 (0.69)	0.355	0.551
1-year outcomes				
NACE	16 (11.03)	24 (16.55)	1.937	0.164
MACCE	12 (8.28)	22 (15.17)	3.228	0.072
All-cause death	2 (1.38)	8 (5.52)	3.705	0.054
Cardiac death	2 (1.38)	6 (4.14)	2.015	0.156
All MI	1 (0.69)	4 (2.76)	1.834	0.176
Stroke	0	3 (2.07)	3.021	0.082
Target vessel revascularization	4 (2.76)	7 (4.83)	0.277	0.599
Bleeding	4 (2.76)	2 (1.38)	0.688	0.407
3-year outcomes				
NACE	20 (13.79)	38 (26.21)	7.058	0.008
MACCE	15 (10.34)	33 (22.76)	7.802	0.005
All-cause death	4 (2.76)	10 (6.90)	2.709	0.100
Cardiac death	3 (2.07)	8 (5.52)	2.354	0.125
All MI	2 (1.38)	7 (4.83)	2.857	0.091
Stroke	0	3 (2.07)	3.021	0.082
Target vessel revascularization	9 (6.21)	13 (8.97)	0.079	0.400
Bleeding	5 (3.45)	5 (3.45)	0.000	0.988

Data are presented as n (%). *Log-rank tests. STEMI: ST-elevation myocardial infarction; PCI: Percutaneous coronary intervention; MACCE: Major adverse cardiac and cerebrovascular event; NACE: Net adverse clinical event; MI: Myocardial infarction.

Table 3: Independent predictors of 3-year NACE by propensity score matching

Characteristics	HR	95% CI	P
Female	2.557	1.415–4.620	0.002
Symptom onset time	0.948	0.890–1.009	0.093
Killip class	1.160	0.851–1.582	0.347
Hypertension	2.017	1.138–3.576	0.016
Chronic kidney disease	2.716	0.495–9.570	0.303
Cholesterol	0.845	0.659–1.083	0.183
Diseased vessels	1.153	0.809–1.642	0.431
Family history of CHD	2.256	1.115–4.566	0.024
Family history of hypertension	1.855	0.882–3.901	0.103
The number of stents	0.625	0.437–0.894	0.010
Average stent diameter	0.513	0.249–1.051	0.069

Adjusted for age, sex, symptom onset time, Killip class, hypertension, diabetes, chronic kidney disease, cholesterol, LDL, hyperlipidemia, diseased vessels, number of stents, average stent diameter, prior MI, family history of CHD, family history of hypertension. NACE: Net adverse clinical event; HR: Hazard ratio; CI: Confidence interval; MI: Myocardial infarction; CHD: Coronary heart disease; LDL: Low-density lipoprotein-cholesterol.

outcomes in adult females compared with similarly aged males.

The reasons for the differences in NACE for female patients who showed poorer outcomes for STEMI following PCI

are likely to be multifactorial and may include sex-specific biology, pathophysiology of myocardial infarction, psychosocial stressors, and potential intrinsic differences in angiogenesis. First, accompanying with the increase of aging, estrogen deficiency in adult women may be a potent risk factor for STEMI.^[21] Females were almost 5 years older than males in this study, and tended to have more hemodynamic impairment, which is in accordance with previous observations.^[22–25] Second, 39.31% females had the history of smoking. Compared with males, smoking provides an increased risk for STEMI in females.^[26] Several studies have reported a particularly harmful effect of smoking on females, showing that the dose-dependent risk associated with smoking is significantly higher among females.^[14] Cigarette smoking induces oxidized stress and stimulates the release of vascular inflammatory cytokines, which result in endothelial dysfunction,^[15] and antagonizes the protective vasodilatory effects of estrogen in premenopausal females.^[27] Third, women ≤ 60 years of age had higher rates of any composite complications, such as hypertension than males in the study. Moreover, such risk factor was more strongly associated with the development of myocardial infarction in females than in males, as well as in younger females (≤ 60 years).^[28] What is more, uncontrolled blood pressure would be expected to increase vessel wall stress, a known stimulus to left ventricle remodeling, and result

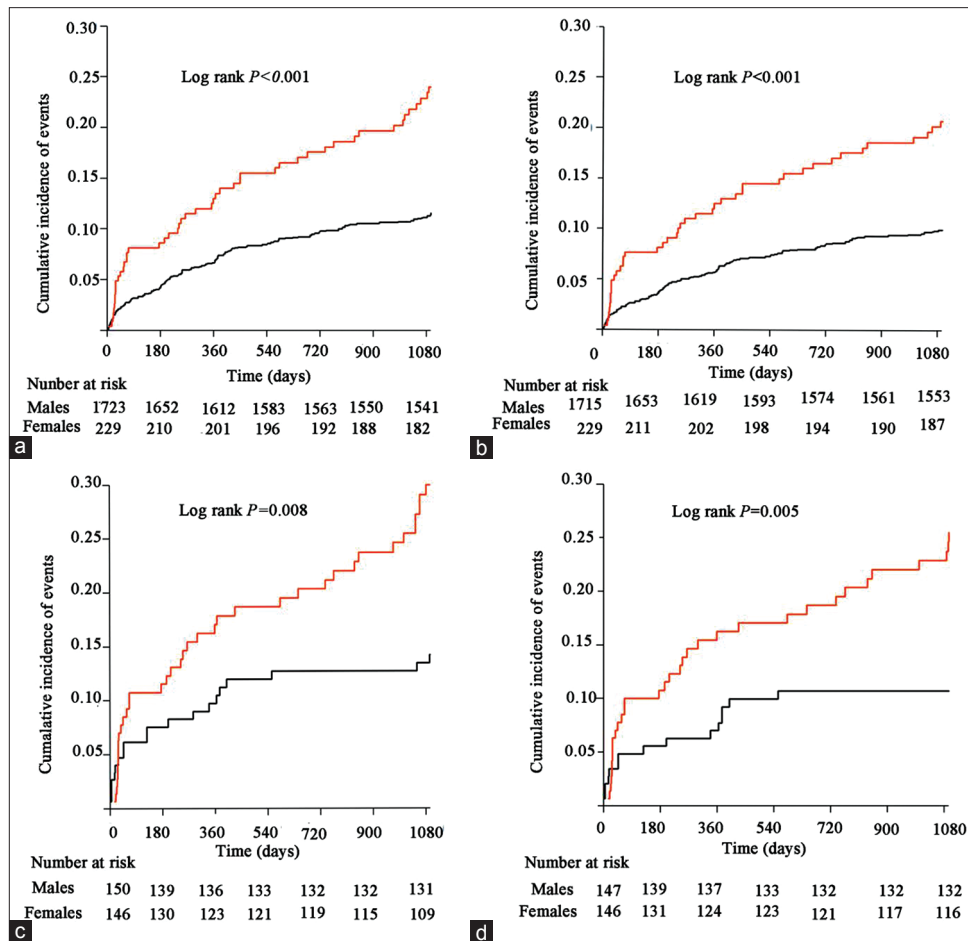


Figure 1: Survival salvage curves of the study. (a) Unadjusted 3-year cumulative incidence of NACE; (b) unadjusted 3-year cumulative incidence of MACCE; (c) adjusted 3-year cumulative incidence of NACE by propensity score matching; (d) adjusted 3-year cumulative incidence of MACCE by propensity score matching. Red line represents females group; black line represents males group. NACE: Net adverse clinical event; MACCE: Major adverse cardiac and cerebrovascular event.

in interstitial fibrosis.^[29] The damage to target organ is more obvious over time, and these are the causes of poor prognosis in female AMI patients with hypertension. We also supposed that the relative risk conveyed by diabetes in females is greater than that in males. The incidence of coronary artery disease in patients with AMI is significantly higher than that in patients without diabetes. Meanwhile, diabetic patients have microvascular and myocardial damage caused by abnormal glucose metabolism. These are the causes of poor prognosis in diabetic female AMI patients.^[30] Fourth, females had significantly longer time from symptom onset to treatment. Apparently, females tended to postpone seeking medical care longer than males. We suppose that atypical symptoms are often a reason for patient's delay in seeking medical attention and should be incorporated in these education efforts. The symptom onset time of Chinese is longer than that of foreign standards time. In China, public recognition of the symptoms of common AMI should be raised, and the start of the catheter room as soon as possible can not only treat the delay but also reduce the patient's mortality. What is more, the incidence of plaque ruptures and subsequent thrombosis are more common in young male and older female patients with STEMI, while

plaque erosion and microvascular embolization are more frequently reported in young female patients.^[31] It has been reported that erosive plaques have a higher level of critical stenosis as a result of greater maturation of thrombus material in comparison with ruptured plaques in pathology study.^[32] When plaque ruptures occur, thrombus formation is more frequently observed in females than males.^[33] Furthermore, in the current study, the number of stents were protective factors. Compared with females, the number of implanted stents was relatively much more in males, which might also account for the lower occurrence of 3-year NACE in males. At last, genes that might impact the effect of cardiovascular risk factors differentially by sex are still being investigated. For example, the Ser843 variant of glycoprotein IIb may be associated with an increased risk of MI in young women with other cardiovascular risk factors.^[34] These results suggest that family history of CHD is a risk factor for the poor prognosis of MI and more strict control of risk factors is needed for females, with estrogens decreasing, to reduce the incidence of STEMI.

In summary, higher mortality in females was mainly attributed to older age, worse risk-factor profiles, and greater

comorbidities. After the propensity-matched adjustment in this study, the most of females with more risk factors were removed. By this way, female was still an independent predictor long-term NACE in STEMI patients undergoing PCI, suggesting that female was a special group. Therefore, for the management of STEMI, the 2012 European Society of Cardiology Guidelines consider female a special patient subset that requires specific awareness and attention during diagnosis and treatment.^[35] Particular attention should be given to females with STEMI so as to provide them with an equal quality of treatment as males. Thus, each sex should be considered with a distinct group of risk factors, undergoing temporal changes in an independent way. For this reason, reporting of future sex-specific results should be encouraged. Furthermore, baseline risks should be lowered with focused strategies for secondary prevention, particularly for females. It is vital that these comorbidities, including hypertension and diabetes mellitus, are actively improved in the general population to prevent the occurrence of cardiovascular disease in adults. To this end, a national prevention program for the management of cardiovascular diseases is urgently required because there is an absence of public preventive care programs or projects for coronary risk factors to reduce cardiovascular diseases in China.

There are some limitations to this study. First, as this was a clinical observational and a single-center study, it remains unclear why females had a greater prevalence of comorbidities than males. Second, this study was a retrospective analysis; the impact of the undervaluation of unmeasured factors on sex-related outcomes has not been addressed. Third, stent thrombosis was missing or lacking. Furthermore, other demographic factors, such as economic status, education background, and geographical region, were not considered. There were no data available regarding menopausal status and hormone replacement. Finally, the analysis was restricted to patients 60 years of age and who had STEMI, limiting the sample size and preventing definitive conclusions. Our observations should be regarded as hypothesis generating.

In conclusion, female gender was independently and significantly associated with increased 3-year NACE in this population of patients. Health-care providers should improve STEMI treatment at the national level by taking into consideration this specific at-risk group of females. Further studies are required to clarify the underlying mechanisms for this increased risk to develop improved therapeutic strategies for females with STEMI after PCI.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV, Go AS. Population trends in the incidence and outcomes of acute myocardial infarction. *New England J Med* 2010;362:2155-65. doi: 10.1056/NEJMoa0908610.
2. Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, *et al.* Executive summary: Heart disease and stroke statistics—2016 update: A Report from the American Heart Association. *Circulation* 2016;133:447-54. doi: 10.1161/CIR.0000000000000366.
3. Kolte D, Khera S, Aronow WS, Mujib M, Palaniswamy C, Sule S, *et al.* Trends in incidence, management, and outcomes of cardiogenic shock complicating ST-elevation myocardial infarction in the United States. *J Am Heart Assoc* 2014;3:e000590. doi: 10.1161/JAHA.113.000590.
4. Khera S, Kolte D, Aronow WS, Palaniswamy C, Subramanian KS, Hashim T, *et al.* Non-ST-elevation myocardial infarction in the United States: Contemporary trends in incidence, utilization of the early invasive strategy, and in-hospital outcomes. *J Am Heart Assoc* 2014;3. pii: e000995. doi: 10.1161/JAHA.114.000995.
5. Li GX, Zhou B, Qi GX, Zhang B, Jiang DM, Wu GM, *et al.* Current trends for ST-segment elevation myocardial infarction during the past 5 years in rural areas of China's Liaoning Province: A Multicenter Study. *Chin Med J* 2017;130:757-66. doi: 10.4103/0366-6999.202742.
6. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr., Chung MK, de Lemos JA, *et al.* 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation* 2013;127:e362-425. doi: 10.1016/j.jacc.2012.11.019.
7. Kang SH, Suh JW, Yoon CH, Cho MC, Kim YJ, Chae SC, *et al.* Sex differences in management and mortality of patients with ST-elevation myocardial infarction (from the Korean Acute Myocardial Infarction National Registry). *Am J Cardiol* 2012;109:787-93. doi: 10.1016/j.amjcard.2011.11.006.
8. Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC), Steg PG, James SK, Atar D, Badano LP, Blomstrom-Lundqvist C, *et al.* ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33:2569-619. doi: 10.1093/eurheartj/ehs215.
9. Zhang Z, Fang J, Gillespie C, Wang G, Hong Y, Yoon PW, *et al.* Age-specific gender differences in in-hospital mortality by type of acute myocardial infarction. *Am J Cardiol* 2012;109:1097-103. doi: 10.1016/j.amjcard.2011.12.001.
10. Jneid H, Fonarow GC, Cannon CP, Hernandez AF, Palacios IF, Maree AO, *et al.* Sex differences in medical care and early death after acute myocardial infarction. *Circulation* 2008;118:2803-10. doi: 10.1161/CIRCULATIONAHA.108.789800.
11. Champney KP, Frederick PD, Bueno H, Parashar S, Foody J, Merz CN, *et al.* The joint contribution of sex, age and type of myocardial infarction on hospital mortality following acute myocardial infarction. *Heart* 2009;95:895-9. doi: 10.1136/hrt.2008.155804.
12. Poulter N. Global risk of cardiovascular disease. *Heart* 2003;89 Suppl 2:ii2-5. doi: 10.1136/heart.89.suppl_2.ii2.
13. Zhang B, Jiang DM, Zhou XC, Liu J, Zhang W, Sun YJ, *et al.* Prospective multi-center study of female patients with ST-elevation myocardial infarction in Liaoning Province, China. *Chin Med J* 2012;125:1915-9. doi: 10.37274/sp.j.1263.2011.00258.
14. Cutlip DE, Windecker S, Mehran R, Boam A, Cohen DJ, van Es GA, *et al.* Clinical end points in coronary stent trials: A case for standardized definitions. *Circulation* 2007;115:2344-51. doi: 10.1161/CIRCULATIONAHA.106.685313.
15. Zheng X, Dreyer RP, Hu S, Spatz ES, Masoudi FA, Spertus JA, *et al.* Age-specific gender differences in early mortality following ST-segment elevation myocardial infarction in China. *Heart* 2015;101:349-55. doi: 10.1136/heartjnl-2014-306456.
16. Second Chinese Cardiac Study (CCS-2) Collaborative Group. Rationale, design and organization of the Second Chinese Cardiac Study (CCS-2): A randomized trial of clopidogrel plus aspirin, and of metoprolol, among patients with suspected acute myocardial infarction. Second Chinese Cardiac Study (CCS-2) Collaborative Group. *J Cardiovasc Risk* 2000;7:435-41.

doi: 10.1177/204748730000700608.

17. Chen ZM, Pan HC, Chen YP, Peto R, Collins R, Jiang LX, *et al.* Early intravenous then oral metoprolol in 45,852 patients with acute myocardial infarction: Randomised placebo-controlled trial. *Lancet* 2005;366:1622-32. doi: 10.1016/S0140-6736(05)67661-1.
18. Chandrasekhar J, Baber U, Sartori S, Faggioni M, Aquino M, Kini A, *et al.* Sex-related differences in outcomes among men and women under 55 years of age with acute coronary syndrome undergoing percutaneous coronary intervention: Results from the PROMETHEUS study. *Catheter Cardiovasc Interv* 2017;89:629-37. doi: 10.1002/ccd.26606.
19. D'Onofrio G, Safdar B, Lichtman JH, Strait KM, Dreyer RP, Geda M, *et al.* Sex differences in reperfusion in young patients with ST-segment-elevation myocardial infarction: Results from the VIRGO study. *Circulation* 2015;131:1324-32. doi: 10.1161/CIRCULATIONAHA.114.012293.
20. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. *N Engl J Med* 1999;340:1801-11. doi: 10.1056/NEJM199906103402306.
21. Lidegaard Ø. Hormonal contraception, thrombosis and age. *Expert Opin Drug Saf* 2014;13:1353-60. doi: 10.1517/14740338.2014.950654.
22. Jackson EA, Moscucci M, Smith DE, Share D, Dixon S, Greenbaum A, *et al.* The association of sex with outcomes among patients undergoing primary percutaneous coronary intervention for ST elevation myocardial infarction in the contemporary era: Insights from the Blue Cross Blue Shield of Michigan Cardiovascular Consortium (BMC2). *Am Heart J* 2011;161:106-120. doi: 10.1016/j.ahj.2010.09.030.
23. Berger JS, Elliott L, Gallup D, Roe M, Granger CB, Armstrong PW, *et al.* Sex differences in mortality following acute coronary syndromes. *JAMA* 2009;302:874-82. doi: 10.1001/jama.2009.1227.
24. Biava LM, Scacciarella P, Calcagnile C, Dalmaso P, Conrotto F, Fanelli AL, *et al.* Sex-related differences in patients with ST-elevation myocardial infarction undergoing primary PCI: A long-term mortality study. *Cardiovasc Revasc Med* 2015;16:135-40. doi: 10.1016/j.carrev.2015.02.001.
25. Lawesson SS, Alfredsson J, Fredrikson M, Swahn E. A gender perspective on short- and long term mortality in ST-elevation myocardial infarction – A report from the SWEDEHEART register. *Int J Cardiol* 2013;168:1041-7. doi: 10.1016/j.ijcard.2012.10.028.
26. Grundtvig M, Hagen TP, German M, Reikvam A. Sex-based differences in premature first myocardial infarction caused by smoking: Twice as many years lost by women as by men. *Eur J Cardiovasc Prev Rehabil* 2009;16:174-9. doi: 10.1097/HJR.0b013e328325d7f0.
27. Shapiro S. Oral contraceptives and the risk of myocardial infarction. *N Engl J Med* 2002;346:1826-9.
28. Anand SS, Islam S, Rosengren A, Franzosi MG, Steyn K, Yusufali AH, *et al.* Risk factors for myocardial infarction in women and men: Insights from the INTERHEART study. *Eur Heart J* 2008;29:932-40. doi: 10.1093/eurheartj/ehn018.
29. Kenchaiah S, Pfeffer MA, St. John Sutton M, Plappert T, Rouleau JL, Lamas GA, *et al.* Effect of antecedent systemic hypertension on subsequent left ventricular dilation after acute myocardial infarction (from the Survival and Ventricular Enlargement trial). *Am J Cardiol* 2004;94:1-8. doi: 10.1016/j.amjcard.2004.03.020.
30. Kalyani RR, Lazo M, Ouyang P, Turkbey E, Chevalier K, Brancati F, *et al.* Sex differences in diabetes and risk of incident coronary artery disease in healthy young and middle-aged adults. *Diabetes Care* 2014;37:830-8. doi: 10.2337/dc13-1755.
31. Frink RJ. Gender gap, inflammation and acute coronary disease: Are women resistant to atheroma growth? Observations at autopsy. *J Invasive Cardiol* 2009;21:270-7.
32. Kramer MC, Rittersma SZ, de Winter RJ, Ladich ER, Fowler DR, Liang YH, *et al.* Relationship of thrombus healing to underlying plaque morphology in sudden coronary death. *J Am Coll Cardiol* 2010;55:122-32. doi: 10.1016/j.jacc.2009.09.007.
33. Kruk M, Pregowski J, Mintz GS, Maehara A, Tyczynski P, Witkowski A, *et al.* Intravascular ultrasonic study of gender differences in ruptured coronary plaque morphology and its associated clinical presentation. *Am J Cardiol* 2007;100:185-9. doi: 10.1016/j.amjcard.2007.02.084.
34. Reiner AP, Schwartz SM, Kumar PN, Rosendaal FR, Pearce RM, Aramaki KM, *et al.* Platelet glycoprotein IIb polymorphism, traditional risk factors and non-fatal myocardial infarction in young women. *Br J Haematol* 2001;112:632-6. doi: 10.1046/j.1365-2141.2001.02609.x.
35. De Luca G, Suryapranata H, Ottervanger JP, Antman EM. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: Every minute of delay counts. *Circulation* 2004;109:1223-5. doi: 10.1161/01.CIR.0000121424.76486.20.

中青年急性ST段抬高心肌梗死患者行急诊PCI的预后的性别差异研究

摘要

背景: 在中国, 急性ST段抬高型心肌梗死 (ST-elevation myocardial infarction, STEMI) 的女性住院率和短期死亡率高于男性, 这表明性别之间存在差异。目前, STEMI发病年龄提前, 存在年轻化的趋势。因此, 尚不明确性别对STEMI患者行急诊经皮冠状动脉介入治疗 (primary percutaneous coronary intervention, PPCI) 术后的远期疗效是否存在性别差异。本研究探讨中青年STEMI患者行急诊PCI治疗的临床特征及术后随访30天、1年、3年的净不良临床事件 (net adverse clinical event, NACE) 的发生情况, 旨在评价性别对中青年STEMI患者行急诊PCI的近期和远期预后的差异。

方法: 回顾性收集沈阳军区总医院从2006年1月至2012年12月年龄 ≤ 60 岁的STEMI患者行急诊PCI治疗的1920例, 按照性别分为两组, 采用倾向性评分校正不同性别患者年龄及合并症等基线资料差异后, 进一步分析性别对STEMI患者行PPCI术后的预后差异。主要研究终点为3年NACE发生情况, 并应用Kaplan-Meier曲线评价两组患者的预后不良事件的发生率, 应用log-rank P 检验。应用Cox比例风险模型观察性别对术后3年NACE发生风险的预后独立影响因素。

结果: 与男性患者比较, 女性平均年龄较男性大, 症状发作到入院时间延长, 合并患有 高血压、糖尿病、慢性肾脏病 ($P < 0.05$), 且吸烟史、Killip ≥ 3 级及三支血管病变比例较高 ($P < 0.05$)。女性低密度脂蛋白 [low density lipoprotein, LDL; 2.72(2.27, 3.29) vs. 2.53(2.12, 3.00), $P < 0.001$], 高密度脂蛋白 [high density lipoprotein, HDL; 1.43(1.23, 1.71) vs. 1.36(1.11, 1.63), $P = 0.003$], 胆固醇 (total cholesterol, TC; 4.98 \pm 1.10 vs. 4.70 \pm 1.15, $t = -3.508$, $P < 0.001$), 估计肾小球滤过率 (estimated glomerular filtration rate, eGFR; 103.12 \pm 22.22 vs. 87.55 \pm 18.03, $t = -11.834$, $P < 0.001$) 水平高于男性组, 采用倾向性评分校正不同性别患者的年龄及合并症等临床特征后, 不同性别的患者术后30天、1年NACE 和主要不良心脑血管事件 (major adverse cardiac or cerebral events, MACCE) 的发生率差异没有 统计学意义, 3年发生NACE和MACCE仍有统计学显著性差异。COX多因素分析显示, 女性[风险比 (HR): 2.557, 95% 可信区间 (CI): 1.415–4.620, $P = 0.002$], 高血压 (HR: 2.017, 95% CI: 1.138–3.576, $P = 0.016$), 冠心病家族史 (HR: 2.256, 95% CI: 1.115–4.566, $P = 0.024$) 是3年NACE预后独立危险因素。支架数目是 (HR: 0.625, 95% CI: 0.437–0.894, $P = 0.010$) 3年NACE 预后保护因素。

结论: 中青年女性STEMI患者行急诊PCI术后近期临床结局和男性相当, 远期预后较差, 因此 需要考虑到这个特定的危险群体, 需要长期关注中青年女性STEMI患者。