Original Article

No correlation between body mass index and 30-day prognostic outcome in Asians with acute ST-elevation myocardial infarction undergoing primary coronary intervention

Po-Jui Wu a,1, Hui-Ting Wanga,1, Pei-Hsun Sung a, Meng-Shen Tonga, Cheng-Hsu Yang a, Chien-Jen Chen a, Cheng-Jei Lin a, Shu-Kai Hsueha, Sheng-Ying Chungen a, Wen-Jung Chungen a, Chi-Ling Hang a, Chiung-Jen Wua,⁎⁎, Hon-Kan Yipa,b,c,e,f,⁎

a Division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan
b Center for Translational Research in Biomedical Sciences, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan
c Institute of Shock Wave Medicine and Tissue Engineering, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan
d Department of Emergency Medicine, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan
e Department of Medical Research, China Medical University Hospital, China Medical University, Taichung, Taiwan
f Department of Nursing, Asia University, Taichung, Taiwan

Article history:
Received 15 June 2016
Accepted 27 December 2016
Available online 29 May 2017

Keywords:
Acute ST-segment elevation myocardial infarction
Overweight
Obesity

Abstract

Background: This study investigated whether body mass index (BMI) was a risk factor predictive of 30-day prognostic outcome in Asians with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI).

Material and methods: Data regarding the impact of BMI on the prognostic outcome in Asian populations after acute STEMI is scarce. A number of 925 STEMI patients were divided into three groups according to the BMI: normal weight (<25 kg/m²), overweight (≥25.0 to <30.0 kg/m²) and obese (≥30.0 kg/m²).

Results: The obese group was significantly younger with significantly higher incidences of smoking and diabetes mellitus. The incidences of multi-vessel disease, final thrombolysis in myocardial infarction (TIMI)-3 flow, advanced Killip score, advance congestive heart failure, 30-day mortality and combined 30-day major adverse clinical outcome (MACO) did not differ between the three groups.

* Corresponding author. Division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, 123, Dapei Rd., Niaosong, Kaohsiung 833, Taiwan.
** Corresponding author. Division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, 123, Dapei Rd., Niaosong, Kaohsiung 833, Taiwan.
E-mail addresses: cvcjwu@mail.cgmh.org.tw (C.-J. Wu), han.gung@msa.hinet.net (H.-K. Yip).
Peer review under responsibility of Chang Gung University.
1 Equal contributors.

http://dx.doi.org/10.1016/j.bj.2016.12.002
2319-4170/© 2017 Chang Gung University. Publishing services by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
Obesity is well-recognized as an important risk factor for the development of coronary heart disease [1], stroke, heart failure, hypertension, and diabetes [1,2]. Additionally, epidemiologic studies have provided evidence that obesity is associated with increased rates of myocardial infarction (MI) and death from cardiovascular diseases [3,4]. Increasingly, studies have also revealed a link between obesity and higher rates of malignancy and overall mortality [5,6]. Likely as a result of studies such as these [1–4], the American Heart Association and American College of Cardiology guidelines for secondary prevention in coronary artery disease (CAD) list obesity as a major modifiable cardiovascular risk factor [7]. Additionally, obesity has been identified to increase insulin resistance, augment free fatty acid turnover, increase basal sympathetic tone, upregulate a hypercoagulable state, and promote systemic inflammation, all of which can contribute to the development and propagation of CAD [8].

Contrarily, an increasing number of epidemiological studies have revealed that obesity may offer protection in some common disease settings [9–15] such as end-stage renal disease, heart failure, atrial fibrillation and sudden death, leading to the proposal of the “obesity paradox” [16,17]. Further, clinical observational studies, including one meta-analysis, have demonstrated an “obesity paradox” after percutaneous coronary intervention (PCI), whereby overweight and obese patients seem to have better outcomes compared with normal weight individuals [15,17–21]. Consistently, the “obesity paradox” has also been demonstrated after coronary artery bypass graft (CABG) surgery [22]. Furthermore, additional investigations have reported better outcomes among obese patients with acute coronary syndromes [16,23–25], including obese patients with STEMI undergoing primary PCI [26–28]. Other studies did not support these data [29,30]. These inconsistent findings [15–30] suggest the need for more comprehensive investigation to understand the real impact of obesity on clinical outcome in patients with CAD undergoing PCI, especially considering that AMI remains the leading cause of death in patients hospitalized for CAD disease.

Additionally, when we looked at previous publications [15–26,28–30], we found that the majority of data originate from Western countries, with very few reports from Asia [27]. It is well established that the eating habits and the incidence of obesity are quite different in Western and Asian countries. Thus, it is of interest to clarify whether obesity is a risk factor for 30-day major adverse clinical outcome (MACO) in Asians with STEMI undergoing primary PCI. We, therefore, examined the database from the Kaohsiung Chang Gung Memorial Hospital, Taiwan to determine the effect of BMI on 30-day prognosis of patients with STEMI treated with primary PCI.

### Method

**Patient population, inclusion and exclusion criteria**

All patients with acute STEMI are considered eligible for primary PCI at our institute. Informed consent was obtained from each study subject to receive primary PCI. This was an observational study. The Institutional Review Committee on Human Research at our institution approved this observational study protocol.

Between October 2009 and December 2014, a total number of 925 patients presenting with STEMI of <12 h duration undergoing primary PCI were consecutively recruited into the present study.

**Definition of body mass index**

Body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in meters. Patients were classified as normal weight (<25 kg/m²), overweight (≥25.0 to < 30.0 kg/m²) and obese (≥30.0 kg/m²) in line with the World Health Organization classification system [45]. Additionally, height and weight values were measured at the time of admission.

**Procedure, protocol, and medication for study patients undergoing primary PCI**

Primary PCI was performed according to a previously described protocol [31–33]. A transradial artery approach utilizing a 6-French arterial sheath is a routine procedure for acute STEMI at our institute unless Allen’s test is positive on both sides. A 6-French Kimny guiding catheter (Boston Scientific, Scimed, Maple Grove, MN) was used for both the diagnosis of coronary artery occlusion and primary PCI. Intracoronary balloon pump (IABP) support was performed via a right or left femoral arterial approach in patients experiencing
acute pulmonary edema associated with unstable condition or hemodynamic instability. All patients received a loading dose of clopidogrel (300 mg, orally) in the emergency room, followed by a maintenance dose (75 mg/day, orally) for at least 12 months after primary PCI. Aspirin (100 mg/day, orally) was given indefinitely to each patient. Other commonly prescribed medications also included angiotensin-converting enzyme inhibitors (ACEIs), angiotensin II type I inhibitors (ARBs), statins, beta-blockers, isonitrate, and diuretics.

A loading dose of tirofiban (30 μg/kg of body weight) was administered to patients upon presentation at the emergency room, followed by a maintenance infusion of 0.15 μg/kg/min for 18–24 h at the beginning of this study. However, tirofiban therapy was subsequently withheld for routine administration to STEMI patients except for patients with high-burden thrombus formation [31] observed in infarct-related artery (IRA) at angiography study because it failed to provide any additional benefit to STEMI patients who underwent primary PCI [32]. Therefore, only 287 patients (31.0%) received tirofiban therapy in this study.

PercuSurge GuardWire was utilized when angiographic morphologic features of high-burden thrombus formation were noted in the infarct-related artery. The indications and procedures were based on our previous reports [33,34].

**Criteria for extracorporeal membrane oxygenation installation**

Extracorporeal membrane oxygenation (ECMO) was implemented in the catheterization room for patients whose systolic blood pressure could not be maintained above 75 mmHg after IABP support and intravenous administration of dopamine > 20 μg/kg/min. The procedure and protocol for ECMO support was described in our previous report [35].

**Functional assessment by echocardiography**

Left ventricular (LV) function was assessed using transthoracic echocardiography. With the patients in a supine position, left ventricular internal dimensions [i.e., end-systolic diameter (ESD) and end-diastolic diameter (EDD)] were measured according to the American Society of Echocardiography leading-edge method using at least 3 consecutive cardiac cycles. The LV ejection fraction (LVEF) was calculated as: LVEF (%) = [(LVEDD³ − LVEDS³)/LVEDD³] × 100.

**Definitions**

The definitions of STEMI and procedural success have been reported in our previous studies [31–34]. In detail, STEMI was defined as: (1) typical chest pain lasting for more than 30 min with ST-segment elevation >1 mm in two consecutive precordial or inferior leads and; (2) typical chest pain lasting for more than 30 min with a new-onset complete left bundle branch block. Procedural success was defined as a reduction to residual stenosis of <20% by balloon angioplasty or successful stent deployment at the desired position with a residual stenosis <10% followed by thrombolysis in myocardial infarction (TIMI) grade 3 flow in the IRA. Multi-vessel disease was defined as stenoses of ≥50% in ≥2 major pericardial coronary arteries. Advanced Killip score was defined as ≥Killip score 3 upon presentation. Advance congestive heart failure (CHF) was defined as New York Heart Association Functional Class III, and combined 30-day MACO was defined as advanced CHF, advanced Killip score, or 30-day mortality.

The utilizations of ACEI/ARB, stain and beta-blocker were recorded as the relevant parameters only when they were utilized more than two days in the current study.

**Data collection**

The primary PCI program started at our institute in May 1993. For the purpose of this study, all patients undergoing primary PCI were prospectively recruited. Detailed in-hospital and follow-up data including age, gender, coronary risk factors, Killip score on admission, peak level of creatine phosphokinase (CK)-MB arrival time, duration from puncture to first balloon inflation, reperfusion time, duration of procedure, both pre- and post-PCI TIMI flow grades, angiographic results, number of diseased vessels, in-hospital adverse events, and 30-day mortality were obtained. These data were collected prospectively and entered into a digital database.

This study was based on the Declaration of Helsinki (revised 2013). Written informed consent was obtained from all study participants. And this study was approved by the Institutional Review Committee on Human Research at Kaohsiung Chang Gung Memorial Hospital (IRB number: 104-705B).

**Statistics**

Data were expressed as mean ± SD. Categorical data were analyzed by χ² test. Continuous variables among three groups were compared using one-way ANOVA followed by Bonferroni multiple comparison procedure. Multivariable logistic regression analysis and was utilized to determine correlations between independent parameters and 30-day mortality. Hazard ratio (HR) for long-term mortality was assessed using multiple Cox-regression analysis. Statistical analysis was performed using SPSS statistical software for Windows version 13 (SPSS for Windows, version 17; SPSS, IL, U.S.A.). A p-value of less than 0.05 was considered statistically significant.

**Results**

**Baseline characteristics of 925 study patients**

Table 1 showed the baseline of variables among the normal weight, overweight and obese patients. Normal weight patients were significantly older than overweight and obese patients, and overweight patients significantly older than obese patients. Additionally, the incidence of smoking showed an identical pattern to age among the three groups. On the other hand, male gender was significantly lower in normal weight patients than in overweight and obese patients, but there was no statistical difference between genders in the overweight patients and obese patients. Furthermore, the incidence of diabetes mellitus (DM) was significantly lower...
### Table 1 Baseline characteristics of 925 study patients.

| Variables                      | Normal weight (n = 449) | Overweight (n = 391) | Obese (n = 85) | p-value*          |
|--------------------------------|-------------------------|----------------------|---------------|------------------|
| Age (yrs)                      | 63.6 ± 12.9a            | 59.5 ± 12.4b         | 53.1 ± 12.2c  | <0.001           |
| Male gender                    | 79.3% (359)a            | 86.2% (337)b         | 87.1% (74)b   | 0.017            |
| Smoking                        | 45.4% (204)a            | 205 (52.4%)b         | 53 (62.4%)c   | 0.007            |
| Hypertension                   | 50.6% (256)             | 61.4% (240)          | 63.5% (54)    | 0.317            |
| Diabetes mellitus              | 30.7% (138)a            | 32.0% (125)b         | 44.7% (38)b   | 0.040            |
| Old myocardial infarction      | 7.1% (31)               | 8.2% (32)            | 10.6% (9)     | 0.472            |
| Previous stroke                | 6.9% (31)               | 6.9% (27)            | 4.7% (4)      | 0.742            |
| End-stage renal disease        | 4.7% (21)               | 3.8% (15)            | 3.5% (3)      | 0.789            |
| Creatinine level (mg/dL)       | 1.5 ± 1.6               | 1.6 ± 1.9            | 1.5 ± 1.8     | 0.076            |
| WBC count (<10^3/μL)           | 11.1 ± 4.40             | 11.7 ± 4.63          | 12.2 ± 4.66   | 0.078            |
| Total cholesterol              | 169.5 ± 55.7            | 173.5 ± 49.5         | 180.9 ± 54.3  | 0.157            |
| LDL                            | 105.6 ± 43.1            | 111.3 ± 40.2         | 113.3 ± 46.2  | 0.087            |
| Troponin-I (ng/ml)             | 9.2 ± 30.5a             | 3.8 ± 12.4b          | 7.6 ± 33.5ab  | 0.008            |
| Hemoglobin (g/dl)              | 13.7 ± 2.2a             | 14.8 ± 5.5b          | 14.1 ± 2.1b   | <0.001           |
| Platelets (<10^3/μl)           | 227.6 ± 70.5a           | 215.9 ± 60.7ab       | 225.0 ± 68.9ab| 0.036            |
| Anterior wall MI               | 58.4% (262)a            | 49.6% (194)b         | 51.8% (44)b   | 0.037            |
| Advanced Killip-score         | 23.6% (106)             | 23.0% (90)           | 22.4% (19)    | 0.960            |
| Advanced CHF                  | 12.0% (54)              | 8.2% (32)            | 10.6% (9)     | 0.186            |
| SBP upon presentation          | 132.1 ± 34.8            | 137.1 ± 33.3         | 131.6 ± 27.2  | 0.079            |
| DBP upon presentation          | 65.8 ± 25.3             | 63.4 ± 27.8          | 66.3 ± 27.1   | 0.371            |
| LVEF (%)                       | 56.0 ± 13.9             | 57.6 ± 13.8          | 57.6 ± 12.8   | 0.232            |
| ACEI/ARB use                  | 86.6% (389)             | 89.0% (348)          | 91.8% (78)    | 0.315            |
| Statin use                     | 72.8% (327)             | 79.5% (311)          | 80.0% (68)    | 0.052            |
| Beta-blocker use              | 72.6% (326)             | 74.4% (293)          | 78.8% (67)    | 0.624            |
| Ibr/fila inhibitor use         | 28.7% (129)             | 34.3% (134)          | 28.2% (24)    | 0.188            |

Data were expressed as mean ± SD or % (n).  
Abbreviations: WBC: white blood cell; CHF: congestive heart failure; SBP: systolic blood pressure (mmHg); DBP: diastolic blood pressure (mmHg); LVEF: left ventricular ejection fraction; ACEI/ARB: angiotensin converting enzyme inhibitor/angiotension II type I receptor blocker.  
Letters (a,b,c) indicate significant difference (at 0.05 level) by Bonferroni multiple comparison procedure.  
a By one-way ANOVA test or chi-square test, when it is appropriate.  
b Defined as thromboembolization in distal infarct related artery.  
c Defined as > New York Heart Association Functional Class III.  
d Combined 30-day MACO was defined as advanced CHF, advanced Killip score, or 30-day mortality.

### Table 2 Reperfusion time, angiographic results and 30-day clinical outcome.

| Variables                      | Normal weight (n = 449) | Overweight (n = 391) | Obese (n = 85) | p-value*          |
|--------------------------------|-------------------------|----------------------|---------------|------------------|
| Chest pain to ER (min)         | 185 ± 162               | 180 ± 157            | 199 ± 181     | 0.632            |
| Door to balloon (min)          | 77 ± 60                 | 82 ± 96              | 71 ± 60       | 0.495            |
| Reperfusion time (min)         | 18 ± 11                 | 19 ± 12              | 19 ± 11       | 0.383            |
| Procedural time (min)          | 40 ± 23                 | 39 ± 24              | 42 ± 27       | 0.609            |
| Pre-PCI stenosis (%)           | 93.8 ± 9.9a             | 95.4 ± 8.4b          | 95.4 ± 8.5ab  | 0.028            |
| Post-PCI stenosis (%)          | 14.6 ± 8.1a             | 14.7 ± 8.1a          | 17.4 ± 12.1b  | 0.018            |
| Pre-TIMI flow                  | 0.8 ± 1.1               | 0.6 ± 1.0            | 0.6 ± 1.0     | 0.062            |
| Post-TIMI flow                 | 2.9 ± 0.4               | 2.9 ± 0.4            | 2.9 ± 0.3     | 0.786            |
| Multi-vessel diseaseb          | 62.8% (282)             | 68.0% (266)          | 60.0% (51)    | 0.562            |
| Thrombuser use                 | 63.0% (283)             | 66.5% (260)          | 65.9% (56)    | 0.562            |
| Percusurge use                 | 19.6% (88)              | 19.7% (77)           | 17.6% (15)    | 0.390            |
| Distal embolization            | 2.4% (11)               | 2.6% (10)            | 1.2% (1)      | 0.743            |
| IABP use                       | 18.7% (84)              | 17.4% (68)           | 16.5% (14)    | 0.825            |
| ECMO use                       | 4.2% (19)               | 2.6% (10)            | 2.4% (2)      | 0.351            |
| 30-day mortality               | 7.1% (32)               | 7.7% (30)            | 5.9% (5)      | 0.839            |
| Combined 30-day MACOd          | 29.6% (133)             | 27.4% (107)          | 25.9% (22)    | 0.671            |

Data were expressed as mean ± SD or % (n).  
Abbreviations: ER: emergency room; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction; IABP: intra-aortic balloon pump; ECMO: extracorporeal membrane oxygenator; MACO: major adverse clinical outcome.  
Letters (a,b) indicate significant difference (at 0.05 level) by Bonferroni multiple comparison procedure.  
a By one-way ANOVA test or chi-square test, when it is appropriate.  
b Defined as >2 vessels with >50% of stenosis.  
c Defined as thromboembolization in distal infarct related artery.  
d Combined 30-day MACO was defined as advanced CHF, advanced Killip score, or 30-day mortality.
in normal weight and overweight patients than in obese patients, but this parameter did not differ between the former two groups [Table 1].

The incidences of hypertension, old MI, previous stroke and end-stage renal disease with the requirement of hemodialysis were similar among the three groups. Additionally, the levels of total cholesterol, low-density of lipoprotein, creatinine and white blood cell (WBC) count did not statistically differ among the three groups. The troponin level upon presentation was significantly higher in normal weight patients than in overweight patients, but it showed no difference between normal weight and obese patients or between overweight and obese patients. Furthermore, the incidence of anterior wall STEMI was significantly higher in normal weight patients than in those of overweight and obese patients, but it exhibited no difference between the latter two groups of patients.

The incidences of advanced CHF and advanced Killip were similar among the three groups. Additionally, systolic and diastolic blood pressure upon presentation, the left ventricular ejection fraction, and the incidences of ACEI/ARB, statin, beta-blocker and IIb/IIa inhibitor use did not differ among the three groups.

Reperfusion time, angiographic results and 30-Day clinical outcome among three groups

There were no statistical differences in terms of chest pain onset to emergency room, door to balloon time, reperfusion time, procedure time, pre-TIMI flow and post TIMI flow among the three groups. Additionally, the incidences of multi-vessel disease, distal embolization and the utilizations of thrombuster, PercuSurge protective device, IABP and ECMO were similar among the three groups of patients. Moreover, the incidence of 30-Day mortality or 30-Day MACO did not differ among the three groups [Table 2].

The pre-PCI stenosis was significantly lower in normal weight patients than in overweight and obese patients, but it displayed no difference between overweight and obese patients. Additionally, post-PCI residual stenosis was significantly higher in obese patients than in normal weight and overweight patients, but it exhibited no difference between the latter two groups.

Comparison of baseline variables between patients with and without 30-Day death after STEMI

Table 3 showed the baseline characteristics among patients with and without 30-Day mortality. Age, mean of body mass index, and incidences of male gender, hypertension, diabetes mellitus, smoking, old MI and end-stage renal disease with the requirement of hemodialysis did not differ among the three groups of patients. Additionally, the levels of CK-MB and troponin-I did not differ between the two groups. However, the levels of creatinine and WBC count were significantly higher in dead patients than in surviving patients, whereas the levels of total cholesterol, LDL showed a reversed pattern of the CK-MB between the two groups [Table 3].

The systolic and diastolic blood pressure and the LVEF were significantly lower, whereas, the incidences of advanced CHF and advanced Killip were significantly higher in normal weight patients than in surviving patients. Additionally, the incidences of ACEI/ARB, beta-blocker, stain and IIb/IIa inhibitor use were significantly lower in dead patients than in surviving patients. This difference could be explained either as due to
death occurring very quickly after primary PCI or as those dead patients who had hemodynamic compromise were not candidates for the ACEI/ARB or beta-blocker use, or had contraindications for IIB/IIA inhibitor therapy.

The chest pain onset to emergency room and the door to balloon time were similar in the two groups of the patients. However, the reperfusion time and the procedure time were significantly higher in the dead group than in the survival group. Additionally, the pre-PCI TIMI flow, post-PCI TIMI flow and final TIMI-3 flow were significantly lower, whereas the pre-PCI restenosis rate was significantly higher in dead patients than in surviving patients. On the other hand, the post-PCI residual stenosis and the incidences of anterior wall myocardial infarction and thrombuser use did not differ between these two groups of patients.

The incidences of multi-vessel disease, distal embolization and the requirement of IABP and ECMO use were significantly higher in dead patients than in surviving patients.

Univariate analysis of predictors of 30-day mortality

Univariate analysis showed that old age, previous stroke, higher WBC count and creatinine level, higher incidences of advanced Killip score, advanced CHF and distal embolization, longer reperfusion time and procedure time, and lower systolic blood pressure, hemoglobin, pre-TIMI flow and LVEF were significantly most strongly associated with 30-day mortality. Additionally, diabetes mellitus, multi-vessel disease, lower diastolic blood pressure and pre-PCI TIMI flow, as well as without utilizations of IIB/IIA inhibitor and PercuSurge distal protection device were also significantly associated with 30-day mortality [Table 4]. When categorized variables of BMI were utilized into univariate analysis, the results showed that BMI was not a predictor of 30-day mortality (all p > 0.6). However, when BMI was considered as continuous variable to be univariate analysis, the results showed that BMI was significantly predictive of 30-day mortality (p < 0.001).

Multivariable logistic regression analysis of predictors of 30-day mortality

The results of multivariate logistic regression analysis showed that old age, advanced Killip score and lower LVEF were most strongly and independently predictive of 30-day mortality. Additionally, longer reperfusion time, higher creatinine level and WBC count, and lower pre- and post-TIMI flow were also significantly and independently predictive of 30-day mortality, whereas utilization of PercuSurge was significantly and independently predictive of free from 30-day mortality [Table 5]. Importantly, the continuous variable of BMI was no longer an independent predictor of 30-day mortality. Further analysis exhibited that BMI and age had a very high negative correlation (r = -0.157, p < 0.001). Moreover, the result of receiver operating characteristic curve (ROC) curve [Fig. 1] showed that the area under curve (AUC) = 0.477 (p = 0.539), i.e., no discrimination ability. These findings could explain why continuous variable of BMI was not an independent predictor of 30-day mortality in STEMI patients undergoing primary PCI.

Discussion

This study investigated the impact of obesity on 30-day clinical outcome in patients with STEMI undergoing primary PCI. The results had several clinical implications. First, the results of the present study did not show a correlation between obesity and the incidences of advanced Killip score, advanced CHF, multiple vessel disease and final TIMI-3 flow. Second, the BMIs (normal weight, overweight and obesity) were not significant predictors of 30-day MACO. Third, only traditional

| Variables | OR (95% CI) | p-value |
|-----------|------------|---------|
| Age (yrs) | 1.045 (1.024–1.066) | <0.001 |
| Diabetes mellitus | 1.756 (1.062–2.903) | 0.028 |
| Previous stroke | 3.526 (1.774–7.006) | <0.001 |
| White blood cell count | 1.098 (1.051–1.147) | <0.001 |
| Hemoglobin | 0.813 (0.733–0.901) | <0.001 |
| Creatinine level | 1.182 (1.080–1.294) | <0.001 |
| Systolic blood pressure | 0.981 (0.974–0.988) | <0.001 |
| Diastolic blood pressure | 0.990 (0.981–0.998) | 0.017 |
| Advanced Killip score | 8.278 (4.839–14.160) | <0.001 |
| Advanced congestive heart failure | 4.073 (2.278–7.282) | <0.001 |
| Left ventricular ejection fraction | 0.934 (0.914–0.955) | <0.001 |
| IIB/IIA inhibitor use | 0.463 (0.244–0.878) | 0.018 |
| Reperfusion time | 1.036 (1.020–1.052) | <0.001 |
| Procedure time | 1.029 (1.021–1.037) | <0.001 |
| Pre-PCI TIMI flow | 0.661 (0.483–0.904) | 0.010 |
| Post-PCI TIMI flow | 0.271 (0.186–0.396) | <0.001 |
| Multi-vessel disease | 2.646 (1.395–5.016) | 0.003 |
| Percusurge use | 0.057 (0.008–0.417) | 0.005 |
| Distal embolization | 6.557 (2.575–16.694) | <0.001 |
| BMI (continuous variable) | 0.932 (0.893–0.973) | <0.001 |
| BMI ≥ 25/BMI < 25 | 1.034 (0.629–1.701) | 0.895 |
| BMI ≥ 30/BMI < 30 | 0.784 (0.306–2.007) | 0.612 |

Abbreviations: OR: Odds ratio; CI: confidence interval; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction; BMI: body mass index.

- Defined as the Killip score ≥ score 3 upon presentation.
- Defined as ≥ New York Heart Association Functional Class III.
- Defined as ≥2 vessels with >50% of stenosis.

| Variables | OR (95% CI) | p-value |
|-----------|------------|---------|
| Age (yrs) | 1.053 (1.024–1.082) | <0.001 |
| Advance Killip score | 3.672 (1.859–7.254) | <0.001 |
| Left ventricular ejection fraction | 0.956 (0.932–0.980) | <0.001 |
| Post-PCI TIMI flow | 0.396 (0.227–0.692) | 0.001 |
| Creatinine (mg/dl) | 1.175 (1.032–1.338) | 0.015 |
| Percusurge use | 0.077 (0.009–0.631) | 0.017 |
| White blood cell count (x10^3/mL) | 1.091 (1.014–1.095) | <0.001 |
| Reperfusion time | 1.025 (1.004–1.047) | 0.021 |
| Pre-PCI TIMI flow | 0.634 (0.429–0.936) | 0.022 |
| BMI | 0.995 (0.959–1.033) | 0.804 |

Abbreviations: OR: Odds ratio; CI: confidence interval; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction.

- Defined as the Killip score ≥ score 3 upon presentation.
disease was similar among the three groups of patients. This finding highlights obesity as not being a risk factor for diffuse CAD/multi-vessel disease.

In contrast to the findings, that BMI did not provide clinically relevant prognostic information, the traditional factors, including age, advanced Killip score upon presentation, lower LEVF, increased WBC count and creatinine level, unsuccessful reperfusion, and prolonged reperfusion time were found to be independent predictors of 30-day mortality in STEMI patients undergoing primary PCI. Consistently, these parameters have also been identified to be significant and independent predictors of untoward clinical outcomes in AMI patients undergoing primary PCI [31–35]. Accordingly, our findings corroborated those of previous studies [31–35]. On the other hand, the utilization of the PercuSurge distal protection device for patients with high-burden thrombus formation was found to be significantly and independently predictive of free from 30-day mortality. Intriguingly, our previous studies have shown that the PercuSurge distal protection device offered additional benefit in preserving integrity and microcirculation and reducing inhospital mortality in AMI patients undergoing primary PCI [33,34]. In this way, the results of the present study corroborated the findings of our previous studies [33,34].

Study limitation

This study had limitations. First, this study was a retrospective analytical study rather than a prospective clinical observational study. Therefore, bias in the present study cannot be completely ruled out. However, all parameters in the present study were prospectively entered into the computer for data analysis, which reflected real world clinical practice. Second, although nearly 1000 patients were enrolled into the study for analysis, the sample size in the present cohort study was still relatively small. Therefore, in view that realizable statistical analysis may be distorted by a smaller sample size, bias of statistical analysis cannot be completely ruled out in this study. Third, the infarct site of the culprit artery did not be utilized as a variate for statistical analysis. Thus, this study did not provide information regarding the association between the infarct site and MACO in patients after STEMI undergoing primary PCI.

Conclusion

Obesity is not a predictor of 30-day prognostic outcome in Asians with STEMI undergoing primary PCI. Unlike those traditional risk factors, BMI did not provide clinically relevant prognostic information for STEMI undergoing primary PCI. Age, advanced Killip score, lower LEVF and unsuccessful reperfusion are statistically significantly related to 30-day mortality in patients with STEMI undergoing primary PCI, but BMI contributed little incremental prognostic information in such an AMI clinical setting.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.
Acknowledgement

This work was supported by grants from the Chang Gung Memorial Hospital Research Project, Taiwan (CMRPG8C1111 and CMRPG8C1112).

References

[1] Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983;67:968–77.

[2] Eckel RH. Obesity and heart disease: a statement for healthcare professionals from the Nutrition Committee, American Heart Association. Circulation 1997;96:3248–50.

[3] Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath Jr CW. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med 1999;341:1097–105.

[4] Wilson PW, D’Agostino RB, Sullivan I, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med 2002;162:1867–72.

[5] Adams KF, Schatzkin A, Harris TB, Kipnis V, Mouw T, Ballard-Barbash R, et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. N Engl J Med 2006;355:763–78.

[6] Key TJ, Allen NE, Spencer EA, Travis RC. The effect of diet on risk of cancer. Lancet 2002;360:861–8.

[7] Smith Jr SC, Blair SN, Bonow RO, Brass LM, Cerqueira MD, Dracup K, et al. AHA/ACC guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease: 2001 update. A statement for healthcare professionals from the American Heart Association and the American College of Cardiology. J Am Coll Cardiol 2001;38:1581–3.

[8] Pi-Sunyer FX. The obesity epidemic: pathophysiology and consequences of obesity. Obes Res 2002;10:975–104S.

[9] Degoulet P, Legrain M, Reach I, Aime F, Devries C, Rojas P, et al. Mortality risk factors in patients treated by chronic hemodialysis. Report of the Diaphane collaborative study. Nephron 1982;31:103–10.

[10] Gonzalez MC, Pastore CA, Orlandi SP, Heymsfield SB. Obesity paradox in cancer: new insights provided by body composition. Am J Clin Nutr 2008;16:442.

[11] Venkatachalam MA, Trusler BA, DeMets DL, Blackburn H, Leppo JA, Olefsky JM, et al. Impact of obesity on short- and long-term mortality postcoronary revascularization: a meta-analysis. Obesity (Silver Spring) 2008;16:442–50.

[12] Powell BW, Lennon RJ, Lerman A, Bell MR, Berger PB, Higano ST, et al. Association of body mass index with outcome after percutaneous coronary intervention. Am J Cardiol 2003;91:472–6.

[13] Gruberg L, Micareto N, Milo S, Boersma E, van Es GA, et al. Impact of body mass index on the outcome of patients with multivessel disease randomized to either coronary artery bypass grafting or stenting in the ARTS trial: the obesity paradox II? Am J Cardiol 2005;95:439–44.

[14] Eisenstein EL, McGuire DK, Bhakar MV, Krishnassan A, Hochman JS, Kong DF, et al. Elevated body mass index and intermediate-term clinical outcomes after acute coronary syndromes. Am J Med 2005;118:981–90.

[15] Wells B, Gentry M, Ruiz-Arango A, Dias J, Landolfo CK. Relation between body mass index and clinical outcome in acute myocardial infarction. Am J Cardiol 2006;98:474–7.

[16] Kojima S, Funahashi T, Sakamoto T, Miyamoto S, Soejima H, Hokamaki J, et al. The variation of plasma concentrations of a novel, adipocyte derived protein, adiponectin, in patients with acute myocardial infarction. Heart 2003;89:667.

[17] Nikolsky E, Stone GW, Grines CL, Cox DA, Garcia E, Tcheng JE, et al. Impact of body mass index on outcomes after primary angioplasty in acute myocardial infarction. Am Heart J 2006;151:168–75.

[18] Koseu M, Kimura K, Kojima S, Sakamoto T, Ishihara M, Asada Y, et al. Impact of body mass index on in-hospital outcomes after percutaneous coronary intervention for ST-segment elevation acute myocardial infarction. Circ J 2008;72:521–5.

[19] Wienbergen H, Gitt AK, Juenger C, Schiele R, Heer T, Towae F, et al. Impact of the body mass index on occurrence and outcome of acute ST-elevation myocardial infarction. Clin Res Cardiol 2008;97:83–8.

[20] Iakobishvili Z, Danicek V, Porter A, Assali AR, Battler A, Hasdai D. Is increased body mass index associated with a cardioprotective effect after ST-segment-elevation myocardial infarction? Acute Card Care 2006;8:95–8.

[21] Mehta RH, Califf RM, Garg J, White HD, Van de Werf F, Armstrong PW, et al. The impact of anthropomorphic indices on clinical outcomes in patients with acute ST-elevation myocardial infarction. Eur Heart J 2007;28:415–24.

[22] Yip HK, Chen MC, Chang HW, Chang CL, Hsieh YK, Fang CY, et al. Angiographic morphologic features of infarct-related arteries and timely reperfusion in acute myocardial infarction: predictors of slow-flow and no-reflow phenomenon. Chest 2002;122:1322–32.
[32] Yip HK, Wu CJ, Chang HW, Hsieh YK, Fang CY, Chen SM, et al. Impact of tirofiban on angiographic morphologic features of high-burden thrombus formation during direct percutaneous coronary intervention and short-term outcomes. Chest 2003;124:962–8.

[33] Yip HK, Wu CJ, Chang HW, Fang CY, Yang CH, Chen SM, et al. Effect of the PercuSurge GuardWire device on the integrity of microvasculature and clinical outcomes during primary transradial coronary intervention in acute myocardial infarction. Am J Cardiol 2003;92:1331–5.

[34] Chen YH, Wu CJ, Chang HW, Fang CY, Chen CJ, Yu TH, et al. Effects and safety of intracoronary thrombectomy using transradial application of the PercuSurge distal balloon protection system in patients with early or recent myocardial infarction. Cardiology 2004;102:206–14.

[35] Sheu JJ, Tsai TH, Lee FY, Fang HY, Sun CK, Leu S, et al. Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock. Crit Care Med 2010;38:1810–7.

[36] Chen YL, Chang CL, Sun CK, Wu CJ, Tsai TH, Chung SY, et al. Impact of obesity control on circulating level of endothelial progenitor cells and angiogenesis in response to ischemic stimulation. J Transl Med 2012;10:86.

[37] Tsai TH, Chai HT, Sun CK, Yen CH, Leu S, Chen YL, et al. Obesity suppresses circulating level and function of endothelial progenitor cells and heart function. J Transl Med 2012;10:137.

[38] Wang HT, Liu CF, Tsai TH, Chen YL, Chang HW, Tsai CY, et al. Effect of obesity reduction on preservation of heart function and attenuation of left ventricular remodeling, oxidative stress and inflammation in obese mice. J Transl Med 2012;10:145.

[39] Ishihara M, Sato H, Tateishi H, Kawagoe T, Shimatani Y, Kurisu S, et al. Clinical implications of cigarette smoking in acute myocardial infarction: acute angiographic findings and long-term prognosis. Am Heart J 1997;134:955–60.

[40] Ruiz-Bailen M, de Hoyos EA, Reina-Toral A, Torres-Ruiz JM, Alvarez-Bueno M, Gomez Jimenez FJ, et al. Paradoxical effect of smoking in the Spanish population with acute myocardial infarction or unstable angina: results of the ARIAM register. Chest 2004;125:831–40.

[41] Shiraishi J, Kohno Y, Sawada T, Hashimoto S, Ito D, Kimura M, et al. Prognostic impact of systolic blood pressure at admission on in-hospital outcome after primary percutaneous coronary intervention for acute myocardial infarction. J Cardiol 2012;60:139–44.

[42] Shiraishi J, Kohno Y, Sawada T, Hashimoto S, Ito D, Kimura M, et al. Prognostic impact of pulse pressure at admission on in-hospital outcome after primary percutaneous coronary intervention for acute myocardial infarction. Heart Vessels 2013;28:434–41.

[43] Cigolini M, Targher G, Bergamo Andreis IA, Tonoli M, Agostino G, De Sandra G. Visceral fat accumulation and its relation to plasma hemostatic factors in healthy men. Arterioscler Thromb Vasc Biol 1996;16:368–74.

[44] Kozek E, Katra B, Malecki M, Sieradzki J. Visceral obesity and hemostatic profile in patients with type 2 diabetes: the effect of gender and metabolic compensation. Rev Diabet Stud 2004;1:122–8.

[45] World Health Organisation. Obesity and overweight. Fact sheet number 311. 2006. Available at: http://www.who.int/mediacentre/factsheets/fs311/en/index.html.