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The Effects of the Obesity Paradox and In-Hospital and One-Year Outcomes in Patients With ST Elevation Myocardial Infarction (STEMI): Results From a STEMI Registry

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Original Article

Abstract

BACKGROUND: Obesity is strongly associated with increased cardiovascular diseases (CVD) and cardiovascular risk factors, such as diabetes mellitus, hypertension, and dyslipidemia. However, numerous studies have suggested the existence of an "obesity paradox" in which overweight and mildly obese patients often exhibit a better outcome than their leaner counterparts. Therefore, this study aimed to characterize the association between BMI and in-hospital and one-year outcomes.

METHOD: This hospital-based research was conducted as a part of the Kermanshah STEMI Registry. Following the application of inclusion criteria, a total of 2,397 STEMI patients were evaluated. The data were collected using a standardized case report developed by the European Observational Registry Program (EORP). Body mass index (BMI) (kg/m^2) was classified into underweight (<18.5), normal weight (18.5–24.9), overweight (25–29.9), class I/mild obese (30–34.9), and class II/extreme obese (≥35) categories. The independent predictors of the in-hospital and one-year outcomes were assessed using multivariable logistic regression models.

RESULTS: Out of the 2397 patients, 43 (1.79%) were underweight, 934 (38.97%) were normal, 1038 (43.30%) were overweight, 322 (13.43%) were class I obese, and 60 (2.50%) were class II obese. The results of the crude analysis showed that class I obesity was protective against CV death (OR 0.50; 95% CI 0.30-0.84), MACE3 (MI, stroke, and death) (OR 0.47; 95% CI 0.29-0.76), and MACE5 (MACE3 plus unstable angina and heart failure) (OR 0.59; 95% CI 0.44-0.79).

CONCLUSIONS: Multivariate adjustment eliminated the protective effect of class I obesity against death and MACE events. Therefore, it is possible that this protective effect does not exist and instead reflects the impact of confounding variables such as age.

Keywords: Myocardial infarction, Registry, BMI, Iran, Obesity

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Introduction

Acute myocardial infarction (AMI) is an expensive condition and the leading worldwide cause of mortality and morbidity. ST-segment elevation myocardial infarction (STEMI) is the most deadly sub-class of MI (over 35%) [1]. Obesity is increasingly related to cardiovascular (CV) risk factors such as diabetes mellitus, hypertension, and hyperlipidemia. Furthermore, obese patients have a greater prevalence of acute coronary syndromes (ACS) [2, 3].

Despite the association between obesity and CV disease incidence, a paradoxical advantage of obesity on CV outcomes after MI has been

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documented [4-6]. The relationship between the interaction between obesity and CV outcomes and public health is substantial. As stated, an obesity paradox exists among MI patients; those with a normal BMI have a worse prognosis than overweight or obese patients [7, 8]. Therefore, the current study aimed to determine whether an obesity paradox exists over a one-year follow-up period among STEMI patients and to characterize further the association between BMI and in-hospital and one-year outcomes.

Material and Methods

Study Design and Population

This hospital-based study was part of the Kermanshah STEMI Registry in Imam Ali Cardiovascular Center, Kermanshah University of Medical Sciences (KUMS), western Iran. Imam Ali Hospital, the principal cardiovascular center in western Iran, annually serves more than two million patients, most of whom are Kurds. All participants who met the inclusion criteria between 1 July 2017 and 1 May 2019 were chosen to participate in the study. The design and foundations of the STEMI registry study are detailed in this article [9].

Inclusion and Exclusion Criteria

Inclusion criteria were a definite diagnosis by STEMI, patients ≥18, and patients who formally signed consent forms to participate in and complete the study. The STEMI diagnosis was based on the following criteria: 1) chest pain for more than 20 minutes within the previous 24 hours before admission; and 2) electrocardiographic changes per new ST-elevations or left bundle branch block, according to the third universal definition of MI described by the European Society of Cardiology/ACCF/AHA/World Heart Federation Task Force for the Universal Definition of MI [10].

In this study, patients with life-threatening diseases (such as cancer, severe kidney failure, and liver cirrhosis), and those with incomplete information, were excluded to eliminate confounding variables. Finally, 2397 patients were enrolled in the present study.

Data Collection and Quality Control

Case report forms developed by the European Observational Research Program were employed to collect data from a nurse and a research assistant trained in the study protocol. Before the final analysis, a general practitioner examined and verified all completed questionnaires for errors. In addition, data were adjudicated according to the European Observational Research Program's (EORP) standards.

Body mass index (BMI) (kg/m^2) was classified into underweight (<18.5), normal weight (18.5–24.9), overweight (25–29.9), class I/mild obese (30–34.9), and class II/extreme obese (≥35) categories.

All patients were invited for examinations after the year. In-hospital and one-year outcomes were evaluated, including in-hospital MI, in-hospital stroke, in-hospital death, percutaneous coronary intervention (PCI) after primary reperfusion, coronary artery bypass grafting (CABG) after primary reperfusion, and MI, stroke, heart failure, unstable angina, and cardiovascular death at one-year follow-up.

Statistical Analysis

Data were analyzed using descriptive statistics, including mean ± standard deviation (SD), median, frequencies, and percentages wherever applicable. One-way analysis of variance (ANOVA) for continuous and normally distributed variables and chi-square (or Fisher exact tests) for categorical variables were used to evaluate differences between subgroups. Multiple logistic regression models were used to determine the independent predictors of in-hospital and one-year outcomes. We calculated odds ratios (ORs) and 95% confidence intervals (CIs). A P-value<0.05 was considered statistically significant. All analyses were performed using Stata (V. 14.1, Stata Corp, College Station, TX, USA).

Ethical Approval

The Research Ethics Committee at KUMS approved the study protocol (Ethics No.

KUMS.REC.1400.125). In addition, patients signed a consent form after being informed about the study and consent to participate. Patient data were kept confidential, with access limited to two researchers and the quality control physician.

Results

Over 24 months, 2,397 patients met the study's inclusion criteria. From the total number of patients, 43 (1.79%) had a BMI<18.5, 934 (38.97%) had 18.5≤BMI<25, 1038 (43.30%) had 25≤BMI<30, 322 (13.43%) had 30≤BMI<35, and 60 (2.50%) had a BMI≥35. Patients with a BMI<18.5 were significantly more likely to be older, male, and smokers and to take thrombolytic therapy, angiotensin-converting enzyme inhibitors/ angiotensin receptor blockers (ACEI/ARBs), and beta-blocker (P= 0.001). Patients with 30≤BMI<35 were more likely to have diabetes (P = 0.003), hyperlipidemia (P = 0.001), and elevated triglycerides (P = 0.001). Similarly, patients with a BMI≥35 were more likely to be hypertensive (P = 0.001) and to have elevated low density lipoprotein (LDL-c) (P = 0.001), high density lipoprotein (HDL-c) (P = 0.043), hemoglobin (P = 0.001), and ejection fraction (EF) (P = 0.001). In addition, patients with an 18.5≤BMI<25 were more likely to take statins (P = 0.033). Table 1 outlines baseline characteristics.

Table 2 shows cardiovascular events and complications. Patients with 30≤BMI<35 experienced a lower rate of MACE3 (P=0.001) and MACE5 (P=0.011) at follow-up. However, patients with 30≤BMI<35 and BMI≥35 experienced a higher rate of PCI after primary reperfusion (P=0.014) at follow-up.

The OR results with a 95% CI for CV complications according to BMI categories are presented in Table 3. Class I obesity was protective against CV death (OR 0.50; 95% CI 0.30-0.84), MACE3 (MI, stroke, and death) (OR 0.47; 95% CI: 0.29 to 0.76), and MACE5 (MACE3 plus unstable angina and heart failure) (OR 0.59; 95% CI 0.44-0.79).

Discussion

According to the findings of this study, the prevalence of obesity in STEMI patients was 15.93%, and the combined prevalence of overweight and obesity was 59.23%. Sandeep et al. found that three-quarters of STEMI patients were overweight or obese [11]. Kaneko et al. studied 1205 PCI patients and discovered that approximately 39% were overweight or obese [12]. Lazzeri et al. studied 1268 STEMI patients who received primary PCI therapy and observed that 51.7% were overweight and 13.6% were obese [13]. Mobeirek et al. examined 3469 patients admitted with ACS and evidenced that 72% were either overweight or obese [14].

In this study of the association between BMI and CV outcomes in 2397 patients with STEMI, we observed an obesity paradox in which patients with class I obesity (30≤BMI<35) had a lower risk of CV death and composite MACE event at one-year follow-up compared to those of normal weight. Although the possibility of inverse causality cannot be ruled out, our analyses indicate that the decreased OR for death and composite MACE events among patients with class I obesity (compared with normal BMI) persists even after excluding patients with preexisting cancer, severe renal failure, and liver cirrhosis.

The fact that the lower unadjusted death rates and composite MACE events in the class I obesity subgroup, compared to the normal BMI subgroup, disappeared after multivariable adjustment is of great interest.

This finding strongly suggests that the unadjusted association of lower death and MACE events with class I obesity is explained by the effect of confounding factors, such as younger age or known or unknown serious medical conditions. In this study, class I obese patients were seven years younger than normal-weight patients on average. Thus, "protective" effects that have been attributed to mild obesity in patients with STEMI may not actually exist and may be the result of unmeasured confounding.

Table 1. Baseline characteristics of patients

Variable	Total (n=2397)	BMI<18.5 (n=43)	18.5≤BMI<25 (n=934)	25≤BMI<30 (n=1038)	30≤BMI<35 (n=322)	BMI≥35 (n=60)	P-value
Age	60.0±11.4	64.9±10.0	63.6±12.3	58.9±11.9	57.0±11.5	55.6±11.4	0.001***
Male	1860(77.6)	35(81.4)	746(79.8)	817(78.7)	224(67.4)	38(63.3)	0.001*
Smoker	1168(48.7)	31(72.1)	482(51.6)	490(47.2)	141(43.7)	22(36.6)	0.001*
Diabetic	481(20.1)	4(9.30)	158(16.9)	226(21.7)	81(25.1)	12(20.0)	0.003*
Hypertensive	1000(41.7)	9(20.9)	350(37.4)	454(43.7)	154(47.8)	33(55.0)	0.001**
Hyperlipidemic	553(23.1)	0(0)	164(17.5)	260(25.0)	109(33.8)	19(31.6)	0.001**
Prior MI	283(11.8)	7(16.3)	100(10.7)	128(12.3)	36(11.1)	10(16.6)	0.43*
Prior stroke	119(4.96)	1(2.32)	50(5.35)	58(5.5)	8(2.48)	2(3.33)	0.17*
Prior PCI/CABG	217(9.05)	4(9.30)	65(6.95)	110(10.5)	31(9.62)	7(11.6)	0.07*
Thrombolytic therapy	633(26.4)	25(58.1)	247(26.4)	257(24.7)	89(27.6)	15(25.0)	0.001*
Primary PCI	1375(57.4)	12(27.9)	519(55.5)	621(59.8)	187(58.0)	36(60.0)	
CABG	209(15.5)	3(6.97)	76(8.13)	96(9.24)	31(9.62)	3(5.0)	
LDL	103.7±86.0	87.6±29.1	100.4±29.0	107.2±30.8	110.9±34.3	113.3±41.0	0.001***
HDL	41.6±9.36	39.4±8.79	41.4±9.66	41.1±8.90	41.5±9.37	44.4±10.0	0.043***
Triglycerides	142.5±76.0	92.7±40.7	126.7±95.5	152.2±93.7	170.7±102.7	168.0±97.4	0.001***
Hemoglobin	14.6±1.77	13.9±1.73	14.5±1.80	14.8±1.79	14.9±1.72	15.0±1.82	0.001***
GFR	68.4±17.4	66.6±18.2	67.7±17.9	68.8±17.2	68.3±18.4	70.5±19.1	0.49***
EF	38.2±10.1	35.3±11.1	36.9±9.91	38.0±9.88	39.4±8.84	41.0±8.49	0.001***
DAPT	1353(56.4)	20(46.5)	509(54.5)	610(58.7)	182(56.5)	32(53.3)	0.22*
Statin	1901(79.3)	35(81.4)	771(82.5)	802(77.2)	246(76.3)	47(78.3)	0.03*
ACEI/ARB	1160(48.4)	35(81.39)	475(50.8)	489(47.1)	136(42.2)	25(41.6)	0.001*
Beta-blocker	1376(58.3)	33(76.7)	581(62.2)	576(55.4)	161(50.0)	25(41.6)	0.001*

MI: Myocardial Infarction.
PCI:percutaneous
coronary intervention.
CABG:coronary artery bypass grafting.
LDL-c: Low density lipoprotein.
HDl-c:High density lipoprotein.
GFR: Glomerular Filtration Rate.
EF: Ejection fraction.
DAPT: Dual antiplatelet therapy
ACEI/ARBs: angiotensin-converting enzyme inhibitors/angiotensin receptor blockers

Table 2. Cardiovascular events and complications

Variable	Total (n=2397)	BMI<18.5 (n=43)	18.5≤BMI<25 (n=934)	25≤BMI<30 (n=1038)	30≤BMI<35 (n=322)	BMI≥35 (n=60)	P-value
In-hospital MI	3(0.12)	0(0)	2(0.2)	1(0.09)	0(0)	0(0)	0.88*
In-hospital stroke	14(0.58)	0(0)	5(0.53)	9(0.86)	0(0)	0(0)	0.41*
In-hospital death	102(4.25)	3(6.97)	39(4.17)	50(4.81)	9(2.79)	1(1.66)	0.37*
In-hospital MACE3	112(4.67)	3(6.97)	46(4.92)	53(5.10)	9(2.79)	1(1.66)	0.30*
PCI after primary Reperfusion	486(20.3)	8(18.6)	158(16.9)	226(21.8)	79(24.5)	15(25.0)	0.014*
CABG after primary Reperfusion	314(13.1)	5(11.6)	120(12.8)	138(13.3)	44(13.7)	7(11.7)	0.98*
MI at follow-up	26(1.08)	0(0)	10(1.07)	12(1.15)	4(1.24)	0(0)	0.868*
Stroke at follow-up	33(1.37)	0(0)	15(1.60)	18(1.7)	0(0)	0(0)	0.122*
Unstable angina at follow-up	307(12.8)	5(11.6)	115(12.3)	142(13.68)	36(11.2)	9(15.0)	0.737*
Heart failure at follow-up	248(10.3)	7(16.3)	109(11.7)	99(9.53)	27(8.38)	6(10.0)	0.242*
Cardiovascular death	210(8.76)	5(11.6)	98(10.5)	85(8.19)	18(5.59)	4(6.66)	0.079*
MACE3 at follow-up	256(10.7)	4(9.30)	120(12.8)	106(10.2)	21(6.52)	5(8.33)	0.001*
MACE5 at follow-up	753(31.4)	15(34.9)	322(34.5)	321(30.9)	77(23.9)	18(30.0)	0.011*

MACE3 comprised MI, stroke, and death.
MACE5 comprised MACE3 plus unstable angina and heart failure.
* Chi-square

Table 3. OR results with 95% CI of CV complications according to BMI categories

Variable	In-hospital CV death		In-hospital MACE3		CV death at follow-up		MACE3 at follow-up		MACE5 at follow-up	
	Crude	Adjusted	Crude	Adjusted	Crude	Adjusted	Crude	Adjusted	Crude	Adjusted
<25	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
25≤BMI<30	1.16(0.75, 1.78)	1(0.55,1.78)	1.03(0.69,1.55)	0.90(0.52,1.54)	0.76(0.56,1.03)	0.80(0.54,1.19)	0.77(0.58,1.01)	0.85(0.60,1.22)	0.85(0.70,1.02)	1.02(0.81,1.28)
P-value	0.126	0.592	0.515	0.648	0.715	0.187	0.475	0.752	0.624	0.156
30≤BMI<35	0.65(0.31,1.37)	1.15(0.48,2.76)	0.55(0.26,1.14)	0.92(0.39,2.13)	**0.50(0.30,0.84)**	0.70(0.36,1.35)	**0.47(0.29,0.76)**	0.62(0.34,1.13)	**0.59(0.44,0.79)**	0.70(0.49,1.01)
P-value	0.895	0.125	0.789	0.681	**0.001**	0.648	**0.001**	0.842	**0.001**	0.588
BMI≥35	0.38(0.05,2.88)	0.67(0.06,6.69)	0.32(0.04,2.41)	0.63(0.07,5.34)	0.60(0.21,1.71)	1.49(0.47,4.71)	0.48(0.17,1.36)	1.10(0.36,3.35)	0.81(0.46,1.43)	1.46(0.77,2.77)
P-value	0.158	0.178	0.489	0.268	0.156	0.762	0.159	0.125	0.791	0.153

Adjusted by gender, smoking, diabetes mellitus, hypertension, hyperlipidemia, prior MI, prior Stroke, prior PCI, prior CABG, EF, LDL, HDL, Triglycerides, Hb, and GFR

Corroborating our results, Niedziela et al. conducted a meta-analysis of 218, 532 patients with MI in 2014. After 1–3 years of follow-up, the study found that obese patients had a 30–40% lower risk of death than patients with normal BMI [15]. At three years of follow-up, the Swedish Coronary Angiography and Angioplasty Registry revealed that overweight/obese patients had the lowest mortality rate, while underweight/normal BMI patients had the highest mortality rate [16].

Neeland et al. (2017) reported, based on the National Cardiovascular Data Registry (NCDR), that mild obesity is associated with a lower long-term risk in older STEMI patients, whereas normal weight and extreme obesity are associated with worse complications [17]. After PCI, Kaneko et al. demonstrated that overweight and obese patients had a lower long-term risk of all-cause mortality, cardiac death, heart failure admission, and MACE [12]. Firman et al. examined 400 STEMI patients who underwent PCI in 2021; they observed that obese patients had a lower incidence of MACE, particularly recurrent MI, at two-year follow-ups [18]. Conversely, Akin et al. examined 890 PCI patients and found no significant difference in MACE-free and target vessel revascularization-free survival based on BMI at one-year follow-up. They concluded that their patients exhibited no "obesity paradox" [19].

Our results showed that the unadjusted and adjusted odds of in-hospital death and in-hospital MACE were not significantly different for any BMI category. Conversely, Sandeep et al. observed that the adjusted odds of in-hospital mortality were lowest for patients with class I obesity, were not significantly different for patients with normal BMI, overweight, or class II obesity, but were significantly greater for patients with class III obesity compared with class I obese patients [11]. In contrast, Akin et al. reported that in-hospital deaths were significantly higher in patients with normal BMI compared to overweight and obese patients [19].

The increased risk of death (adjusted OR: 1.49), composite MACE3 events (adjusted OR: 1.10), and composite MACE5 events (adjusted OR: 1.46) observed with class II obesity suggests a 'threshold effect,' in which BMI ≥35kg/m^2 diminishes or reverses any protective effects caused by excess energy reservoirs in mild obesity. In line with this finding, Neeland et al. found that those with a BMI ≥40 kg/m^2 had a higher mortality rate than those who were mildly obese [17]. Extreme obesity may be associated with adverse effects on hemodynamics and cardiac anatomy and function, such that more blood volume is required to perfuse a growing adipose limb with increased cardiac output and workload [7].

Strengths and Limitations of the Study

Our research has several limitations. First, the study design (observational registry) may not be able to control for the effects of cofactors due to its non-randomized nature; however, the researchers measured and controlled for the effects of the main confounding factors. Second, post-discharge care and treatment differences may have affected the protective association observed with mild obesity. Thirdly, body composition and fat distribution are crucial predictors of CV outcomes [20]. Alternative metrics of adiposity, such as waist circumference and waist-to-hip ratio, were not obtained in this study, thereby limiting the ability to investigate variation in body composition or body fat distribution. Fourth, our data originated from a single-center registry; therefore, our findings may not apply to other racial/ethnic groups. No long-term follow-up data are available; the data presented here can only determine the association between BMI and in-hospital and one-year outcomes. This study has some strengths. For example, it is the first population-based registry with a large sample size in the west of Iran. In addition, patients were evaluated by trained and seasoned experts.

Conclusion

In conclusion, the obesity paradox for CV death and composite MACE event was observed

over a one-year follow-up in STEMI patients. However, it did not extend to extreme obesity. These lower unadjusted rates of death and MACE events in class I obesity disappeared following multivariable adjustment. Therefore, it is possible that this protective effect does not exist and is instead due to unmeasured confounding factors.

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Conflict of interest

The authors have no conflict of interest to declare.

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