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ORIGINAL ARTICLE

Do overweight patients have a better five years prognosis after an acute myocardial infarction treated with coronary intervention?

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Abstract

Background: Obesity and overweight alone may confer a survival benefit after myocardial infarction, independent of age, medical care or therapy.

Aim: To evaluate the impact of body mass index (BMI) on long-term mortality in ST-segment elevation acute myocardial infarction (STEMI) patients treated by primary angioplasty (PCI).

Methods: We prospectively studied a homogenous group of 131 patients who had suffered STEMI, and subsequently exhibited a TIMI 3 flow after primary PCI. The patients (41 women, 90 men, mean age 58.3 ± 10.8 years) were analysed in two groups: Group 1 — 30 (23%) patients with BMI < 25 kg/m^2 and Group 2 — 101 (77%) patients with BMI ≥ 25 kg/m^2.

Results: Altogether, 19 (14.5%) patients died during the five-year follow-up period — nine out of 30 (30%) were patients with BMI < 25 kg/m^2, and ten out of 101 (10%) were patients with BMI ≥ 25 kg/m^2 ($p < 0.001$). Individuals with BMI ≥ 25 kg/m^2 had lower five-year mortality, and this was independent of other potentially confounding variables. Area under the receiver-operating characteristic (ROC) curves for death with respect to weight on ROC analysis was significantly different than for a random model ($p < 0.05$). There were no significant differences in 30-day mortality and one-year mortality ($p = 0.6517$ and $p = 0.3573$, respectively).

Conclusions: Patients after primary angioplasty for STEMI with BMI < 25 kg/m^2 and patients with BMI ≥ 25 kg/m^2 have no difference in 30-day or one-year mortality, but individuals with BMI ≥ 25 kg/m^2 have a better five year prognosis, and this is independent of other potentially confounding variables.

Key words: acute myocardial infarction, mortality, obesity, percutaneous coronary intervention

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INTRODUCTION

Obesity is an important risk factor for the development of diabetes, hypertension, coronary artery disease and left ventricular dysfunction [1]. But in acute adverse cardiovascular events, for example in acute myocardial infarction (AMI), obese patients may have a survival benefit [2]. Previous studies in patients undergoing elective coronary angioplasty have shown a reduction in hospital and long-term mortality in obese patients. This phenomenon is called 'the obesity paradox' [1]. The significance of body mass index (BMI) has been less studied in the context of primary percutaneous coronary intervention (PCI). The statement that obesity alone may confer a survival benefit independent of age, medical care, or therapy is a matter of discussion [2, 3].

Obesity in the context of an AMI has been examined in relatively short follow-up scenarios. There is only limited data

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regarding the correlation between BMI and late-occurring mortality.

The aim of our study was to determine the association between BMI and 30-day, one-year and five-year mortality in patients successfully treated with primary PCI, and to assess which clinical and angiographic variables were most strongly predictive for death.

METHODS

Using a prospective cohort design, we studied 131 consecutive patients with AMI with ST-segment elevation (STEMI), who were referred to the catheterisation laboratory of our hospital for emergency primary PCI between January 2000 and December 2001. The study inclusion criteria were: 1) confirmed MI with ST-segment elevation; 2) successful primary PCI (defined as Thrombolysis in Myocardial Infarction [TIMI] flow grade 3 and residual stenosis < 30%) within 12 h of the onset of symptoms; and 3) informed consent obtained from each patient. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki, and was approved by the local ethics committee.

The exclusion criteria were: 1) cardiogenic shock on admission; 2) unsuccessful primary PCI; and 3) life-limiting non-cardiac disease. The upper age limit of the study was 75 due to the planned long-term follow-up. The primary end-point was all-cause mortality on five-year follow-up.

The patients were analysed as two groups: Group 1 — patients with BMI < 25 kg/m^2 and Group 2 — patients with BMI ≥ 25 kg/m^2.

Patients were treated with heparin and aspirin in the emergency department and transferred promptly to the catheterisation laboratory for mechanical reperfusion. Clopidogrel was continued for one month in patients with stent implantation.

12-lead standard ECG (with paper speed set at 25 mm/s) was performed on admission and immediately after finishing primary PCI.

All studies were performed using the Philips Ultrasound System Sonos 5500, equipped for harmonic imaging with a 3.6 MHz transducer. Left ventricular ejection fraction (LVEF) was calculated according to the modified Simpson's rule from orthogonal apical long-axis projections.

In all patients, the coronary angiography and angioplasty procedures were performed by an experienced invasive cardiologist within 12 h of the onset of symptoms. Arterial access was gained via the right femoral artery. Selective coronary arteriography and left ventriculography were performed using standard techniques. The infarct-related artery (IRA) was identified based on coronary anatomy, regional left ventricular dysfunction and ECG changes. The angioplasty procedure was considered successful when the residual stenosis was < 30%, and occurred in the absence of dissection and/or thrombosis. Only bare metal stents were used. Contrast flow through the epicardial vessel was graded using the standard TIMI trial flow scale of 0 to 3 [4]. Time to reperfusion was defined as the time from the onset of symptoms until balloon inflation. Pharmacological treatment in the catheterisation laboratory included intravenous heparin in doses related to weight.

For all patients, mortality data were obtained from the Department of Civil Affairs and Foreigners in Podlasie Voivodship Office, at a mean follow-up period of 5.0 years (1,779 ± 416 days).

Statistical analysis

Distribution of every variable was assessed with the Kolmogorov-Smirnov test. Additionally, ANOVA and the Student's t test, or alternatively the Mann-Whitney U test, were used for statistical analysis where applicable. Further analysis of correlations between non-categorical variables was performed using Pearson and/or Spearman tests where applicable. Survival rates were displayed with Kaplan-Meier curves. Receiver operating characteristic (ROC) curves were used to assess the value of weight in prediction of death in the long term observation. Multivariate logistic regression was used to test associations between variables and outcomes. When distribution for variables is normal, final data is expressed as means and standard deviations (SD). Categorical variables are presented as the relative frequencies, with the actual number of patients given in brackets. These variables were assessed using the χ^2 test. A p value of less than 0.05 was considered as statistically significant. Statistical analyses were performed with Statistica 9.0 for Windows (StatSoft, Inc. Tulsa, OK, USA, 2009).

RESULTS

A total of 131 patients (41 women, 90 men, mean age 58.3 ± ± 10.8 years) were enrolled into the study. The patients were analysed as two groups: Group 1 — 30 (23%) patients with BMI < 25 kg/m^2 and Group 2 — 101 (77%) patients with BMI ≥ 25 kg/m^2. The mean value of BMI in the group with BMI < 25 kg/m^2 was 22.59 ± 1.72, while the mean value in the group with BMI ≥ 25 kg/m^2 was 29.25 ± 3.24 ($p < < 0.0001$). In our whole population, there were four underweight patients. Demographic, laboratory data and haemodynamic parameters are set out in Table 1. Patients with BMI ≥ 25 kg/m^2 were younger than those with BMI < 25 kg/m^2. Mean sex distribution, heart rate and blood pressure, Killip-Kimball class, LVEF on echocardiography, and CK-MB levels were similar in both groups. There were no significant differences in the prevalence of anterior MI (Table 1). Mean BMI was 22.6 ± 1.7 in the Group 1 and 29.3 ± 3.2 in the Group 2 ($p < 0.001$). There were 36 obese individuals in the group with BMI ≥ 25 kg/m^2, which is 27.6% of the whole population and 35.6% of the subgroup.

There were no significant differences in the use of aspirin, clopidogrel, ACE-inhibitors, beta-blockers and statins in either of the two groups (Table 1). We did not find any significant differences in drug dosing. Furthermore, the mean 'pain-to-balloon' time was similar in both groups (244 ± 91 vs.

Table 1. Clinical and laboratory characteristics of the population according to body mass index (BMI)

	BMI < 25 (n = 30)			BMI ≥ 25 (n = 101)			P
	Mean	%	SD	Mean	%	SD	
Male sex		73	–		67	–	0.5369
Age [years]	62.2		9.4	57.5		11.1	0.0366
Body weight on admission [kg]	64.2		9.0	82.1		11.6	0.0000
Height [cm]	168.2		8.4	167.4		8.0	0.6435
Killip class on admission	1.30		0.47	1.50		0.66	0.1141
History of hypertension		20	–		55	–	0.0005
History of diabetes mellitus type 2		10	–		24	–	0.1033
History of hypercholesterolaemia		43	–		36	–	0.4485
Smoking		77	–		57	–	0.0574
Family history of cardiovascular events		17	–		29	–	0.1890
'Pain-to-balloon' time [min]	244		91	253		134	0.7320
Stent implantation during hospitalisation		47	–		49	–	0.8601
Heart rate on admission	74.2		8.9	76.1		14.6	0.5218
LVEF on admission		44.5	8.3		46.6	8.2	0.2382
SBP on admission [mm Hg]	142.6		23.4	141.6		27.3	0.3655
DBP on admission [mm Hg]	90.2		14.4	88.9		18.0	0.7164
Previous MI		6	–		7	–	0.1384
Previous anterior MI		30	–		35	–	0.6387
Previous inferior MI		60	–		63	–	0.7403
Previous lateral MI		7	–		19	–	0.1130
Previous posterior MI		7	–		3	–	0.3573
Acetylsalicylic acid on discharge		100	–		100	–	–
Beta-blockers on discharge		97	–		90	–	0.2581
ACE-inhibitors on discharge		70	–		65	–	0.6149
Statins on discharge		47	–		49	–	0.7250
Clopidogrel on discharge		77	–		72	–	0.1088
Creatinine [mg/dL]	1.1		0.4	1.0		0.2	0.0878
Glomerular filtration rate [mL/min/1.73 m^2]	78.2		23.3	83.1		25.4	0.0897
Phosphocreatine (CK) [IU]	319.1		253.0	422.5		880.9	0.5348
Cardiac fraction of phosphocreatine (CK-MB) [IU]	48.3		33.9	51.6		85.8	0.8397
Urea [mg/dL]	39.1		13.4	38.3		12.0	0.7460
Glycaemia on admission [mg/dL]	128.8		40.5	149.6		58.1	0.0915
Total cholesterol [mg/dL]	197.9		46.6	204.5		35.6	0.4396
LDL-cholesterol [mg/dL]	128.7		42.5	130.0		31.2	0.8729
HDL-cholesterol [mg/dL]	44.6		11.9	43.7		15.3	0.8066
Triglycerides [mg/dL]	126.9		62.3	151.8		76.8	0.1297

LVEF — left ventricular ejection fraction; SBP — systolic blood pressure; DBP — diastolic blood pressure; MI — myocardial infarction; ACE — angiotensin converting enzyme; LDL — low-density lipoprotein; HDL — high-density lipoprotein

253 ± 134 min, p = 0.732). The angiographic extent of coronary artery disease was also similar. There were no significant differences in the number of stents implanted per patient. Moreover, the TIMI 0 flow in IRA before PCI was almost the same in both groups, as shown in Table 2.

Altogether, 19 (14.5 %) patients died during the five-year follow-up period — nine out of 30 (30%) were patients with BMI < 25 kg/m^2, and ten out of 101 (10%) were patients with BMI ≥ 25 kg/m^2 (p < 0.001), as illustrated in Figure 1. Mortality in patients with BMI ≥ 30 kg/m^2 was 11%. There were no significant differences between the groups when it came to 30-day mortality and one-year mortality (p = 0.6517 and p = 0.3573, respectively). Individuals with BMI ≥ 25 kg/m^2 had lower five-year mortality compared to the patients with BMI < 25 kg/m^2, and this was independent of other potentially confounding variables (Table 3, Fig. 1). Kaplan-Meier

Table 2. Angiographic characteristics of the population according to body mass index (BMI)

	BMI < 25 (n = 30)			BMI ≥ 25 (n = 101)			P
	Mean	%	SD	Mean	%	SD	
LAD as IRA		33			40		0.5141
Cx/MA as IRA		17			17		0.9897
RCA as IRA		50			44		0.6447
% of stenosis of IRA before PCI	98.52		2.46	97.86		9.96	0.7264
% of stenosis of IRA after PCI	5.17		10.22	3.61		6.79	0.3366
TIMI before PCI	0.97		1.21	0.76		1.14	0.4061
Proximal location of stenosis		44			56		0.2095
Medial location of stenosis		36			35		0.8947
Distal location of stenosis		20			9		0.0608
Diameter of balloon	3.11		0.35	3.24		0.33	0.0706
Length of balloon	19.54		1.65	20.65		2.31	0.0237
Max. pressure in balloon	7.50		1.27	7.94		2.36	0.3650
Use of stent		47			49		0.8601
Number of stents	1.14		0.36	1.02		0.14	0.0592
Diameter of stent	3.32		0.37	3.19		0.35	0.2411
Length of stent	17.57		4.82	17.54		4.07	0.9817
Max. pressure in stent	14.31		1.55	15.38		2.47	0.1455
LAD stenosis apart from IRA		27			28		0.7474
Cx/MA stenosis apart from IRA		20			30		0.3161
RCA stenosis apart from IRA		13			18		0.3089
Number of vessels with stenosis apart from IRA	0.56		0.58	0.85		0.72	0.0545

LAD — left anterior descending coronary artery; Cx — circumflex coronary artery; MA — marginal artery; RCA — right coronary artery; IRA — infarct-related coronary artery; PCI — percutaneous coronary intervention

curve displaying survival according to the BMI is shown in Figure 2, with BMI used as a continuous value.

A number of predictive variables such as age ($p = 0.002$), Killip class ($p = 0.003$), LVEF ($p = 0.001$), and BMI ($p = 0.029$) were found to strongly correlate with all-cause five-year mortality when a single value analysis was carried out. After multivariate logistic regression analysis, adjusted for confounding variables, only age, Killip class, LVEF, and BMI ≥ 25 kg/m^2 were independently associated with death, as presented in Table 4.

Area under the ROC curves for death with respect to the weight on ROC analysis was significantly different than for a random model ($p < 0.05$; Fig. 3).

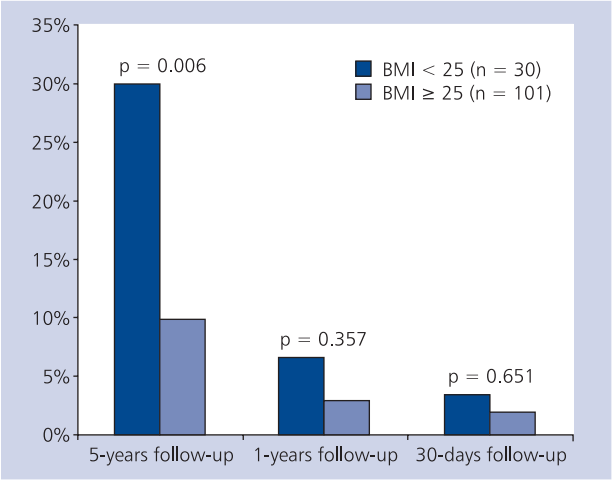

Figure 1. Body mass index (BMI) and mortality during observation

DISCUSSION

Recent years have witnessed lively discussion in the literature as to the influence of BMI on mortality after MI. Our study aims to shed new light on the fact that actual higher weight is associated with significantly lower mortality in the context of an extended follow-up. The longest follow-up in the literature focused on BMI is 3.8 years after a primary PCI, but in that instance TIMI flow < 3 was not an exclusion criterion. We believe that our findings are made all the more interesting through the use of a homogenous group of patients with TIMI 3 flow after primary PCI. In a few studies, the authors have emphasised that obese patients are more frequently treated invasively, and that this is the real explanation of the better prognosis. This factor was eliminated in our study, as well as

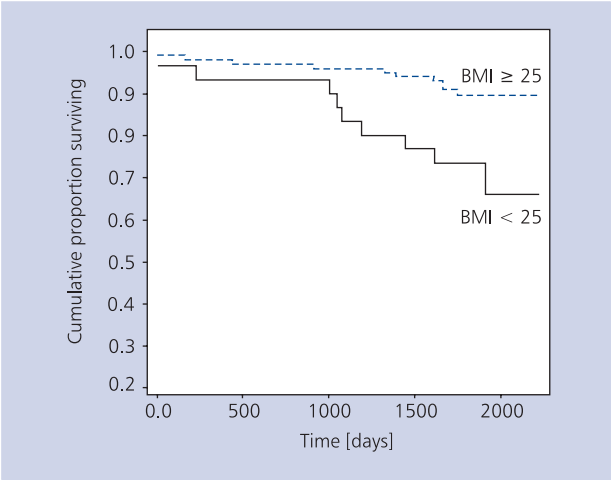

Figure 2. Kaplan-Meier curves displaying cumulative proportion of survival according to body mass index (BMI)

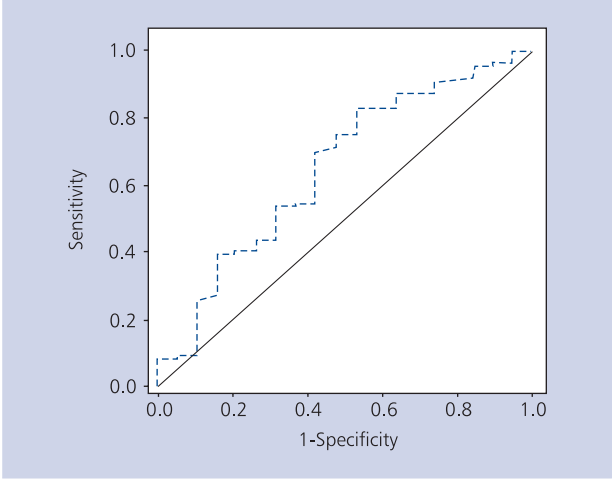

Figure 3. Area under the receiver-operating characteristic curves for death with respect to weight. Area under the curve is significantly different to a random model ($p < 0.05$)

Table 3. Body mass index (BMI) and mortality during five-year observation

	BMI < 25 (n = 30)	BMI ≥ 25 (n = 101)	P
Five-year follow-up	9 (30%)	10 (10%)	0.0058
One-year follow-up	2 (7%)	3 (3%)	0.3573
30-day follow-up	1 (3%)	2 (2%)	0.6517

Table 4. Risk factors of death on five-year follow-up in multivariate analysis

	Odds ratio	CI –95.00%	CI +95.00%	P
Age [years]	0.01	0.00001	0.01048	0.0496
Killip class	0.09	–0.00142	0.19138	0.0534
Left ventricular ejection fraction	–0.01	–0.01718	–0.00336	0.0039
Body mass index ≥ 25	–0.19	–0.32526	–0.05615	0.0058

the influence of pharmacological treatment, which was almost identical in both analysed subgroups.

Some authors have shown that overweight and obesity is associated with classical risk factors but BMI does not influence in-hospital, six-month and one-year mortality after MI [5, 6]. According to Hadi et al. [7], in-hospital mortality is comparable in normal weight and obese individuals with STEMI; moreover, patients with obesity are more likely to have recurrent ischaemia and major bleeding complications.

Other authors have shown that overweight patients have a better prognosis after primary PCI, but this is dependent on various potentially confounding variables [3]. A Japanese group indicated that the paradoxical survival associated with obesity was largely explained by the younger age of obese patients [8]. Janssen [9] even suggested that a BMI cut-off point of 25 kg/m^2 might be too restrictive for the elderly.

Nigam et al. [10] showed that overweight and obese individuals are protected from in-hospital death. 'The obesity paradox' could also be explained by fewernon-cardiovascular comorbidities. In a Korean population, the investigators underlined better use of medical treatment and haemodynamic stability [11]. Lower mortality could also be attributable to better renal function and fewer anterior infarctions [12].

Interestingly, although obese patients have a higher incidence of hypertension, diabetes, and hyperlipidaemia, the angiographic extent of coronary artery disease is similar among the BMI groups [13]. Lower long-term mortality in obese patients remains significant after adjusting for confounding prognostic factors including coronary status and left ventricular function [13]. Similar results were also shown according to different kinds of acute coronary syndromes [14, 15], even after adjustment for age, left ventricular dimensions, contractile efficiency or number of akinetic segments [16, 17].

It seems that the relationship between BMI and mortality is U-shaped, with the highest mortality in underweight and obese class II (BMI > 40 kg/m^2), but the lowest in the other BMI classes [18, 19]. Romero-Corral et al. [20] in a systematic review based on 40 studies with over 250,000 patients with 3.8 years follow-up found that obese patients had no increased risk for total mortality or cardiovascular mortality, probably due to the lack of discriminatory power of BMI to differentiate between body fat and lean mass.

In our study, we concentrated on all-cause mortality because our data was drawn from the Polish population registry at the Ministry of the Interior and Administration Affairs, and as such was not specific for cause of death. In the baseline patients' characteristics there were no significant differences concerning the risk of cancer. Thus the risk of death from non-cardiac reasons such as cancer and accidents was comparable according to statistical methods. All-cause mortality is also a widely used clinical parameter in the assessment of the late results of PCI [21].

What then is the exact explanation of lower mortality in overweight AMI patients? The findings of the MADIT-II trial suggest an independent inverse association between BMI values and the risk of sudden cardiac death, which is very frequent in people after AMI especially with left ventricular dysfunction [22].

In addition, overweight and obese patients with AMI or unstable angina are more likely to receive aspirin, beta-blockers, inhibitors of the rennin–angiotensin system, and lipid-lowering agents, and are also more likely to undergo cardiac catheterisation, PCI, and coronary artery bypass graft (CABG) [23]. Neurohormonal blockade which may attenuate the relationship between high BMI and increased mortality risk can also partly explain 'the obesity paradox' [24].

Endogenous cannabinoids, potent vasodilators which are upregulated in obese patients, might have important protective cardiovascular effects [25–27]. Endocannabinoid receptor agonists exert a cardioprotective effect in ischaemia — reperfusion models with a delay in the formation of necrotic zones and an improved cardiac resistance to malignant arrhythmias and sudden cardiac death [13, 26, 28]. Excess triglyceride content in heart tissue including areas of healed MI might also influence the vulnerability to ventricular arrhythmias in obese compared to normal weight patients [29].

It has also been suggested that the protective effect of obesity may be due to larger overall vessel size [23, 30]. Interestingly, a significant decrease in the rate of transfusion, potentially a surrogate for procedural complications, has been noted as BMI increases [19]. Modern advances in technology such as the use of the radial access approach and the introduction of vascular closure devices explain the high rate of invasive procedures in obese patients [19].

Furthermore, underweight patients have the highest rate of post-PCI complications, including emergency CABG or vascular injury [31]. The increased rate of adverse events could also be due to overdosed anticoagulants, increased sheath-to-artery size ratio that can lead to increased vascular injury, smaller coronary diameter, and increased rates of severe non-cardiac systemic illness.

Our analysis, unlike many previous studies [32, 33], was adjusted for many important factors. After adjustment for confounders such as age, LVEF, the rate of anterior MI, and pharmacological treatment, we found that the patients with BMI ≥ 25 kg/m^2 after STEMI had lower mortality than normal weight patients on five-year follow-up.

No difference between the analysed groups in 30-day and one-year mortality, and yet a significant difference after five years, could be associated with a higher rate of heart failure after AMI on very long-term follow-ups. Patients with heart failure show elevated serum levels of TNF-alpha and leptin and its soluble receptor which may participate in the catabolic state leading to the development of cardiac cachexia in the course of heart failure [34].

Other possible underlying mechanisms of 'the obesity paradox' remain to be investigated.

Limitations of the study

The main limitation of our study was the relatively small group of investigated patients. Although obesity may have different prognostic implications than overweight, with such a sample size, formation of another group than a group with BMI < 25 kg/m^2 and BMI ≥ 25 kg/m^2 would make no sense.

We did not assess waist circumference, which is considered more reliable than BMI by some investigators [35]. We were unable to collect data about weight gain and weight loss during the five-year follow-up, although we are aware that weight loss of > 5% remains a significant risk factor for death [36]. The results of this study cannot be applied to patients with cardiogenic shock, who were excluded from the study. There was a low percentage of patients with stent implantation. This is a consequence of the very long follow-up, as the patients were treated according to the guidelines that were recommended at that time.

Unfortunately, we were not able to obtain the reasons for deaths in our patients. The registry at the Ministry of the Interior and Administration Affairs used as a source of information does not provide data regarding the reason for death.

Nevertheless, the limitations of our study are the price one must accept when investigating a long-term follow-up.

CONCLUSIONS

Patients after primary angioplasty for STEMI with BMI < 25 kg/m^2 and patients with BMI ≥ 25 kg/m^2 have no difference in hospital and one-year mortality, but individuals with BMI ≥ 25 kg/m^2 have a better five-year prognosis, and this finding is independent of other potentially confounding variables. The relation between obesity and long-term prognosis after STEMI should be further assessed in larger population-based cohort studies.

Conflict of interest: *none declared*

References

1. Amundson DE, Djurkovic S, Matwiyoff GN. The obesity paradox. Crit Care Clin, 2010; 26: 583–596.
2. Timóteo AT, Ramos R, Toste A et al. Impact of obesity on results after primary angioplasty in patients with ST segment elevation acute myocardial infarction. Rev Port Cardiol, 2010; 29: 999–1008.
3. Wu AH, Pitt B, Anker SD et al. Association of obesity and survival in systolic heart failure after acute myocardial infarction: potential confounding by age. Eur J Heart Fail, 2010; 12: 566–573.
4. TIMI Study Group. The Thrombolysis In Myocardial Infarction (TIMI) trial: phase 1 findings. N Engl J Med, 1985; 312: 932–936.
5. Fiol M, Amézaga R, Arós F et al. Body mass index in the prognosis of first myocardial infarction. Med Clin (Barc), 2010; 135: 631–636.
6. Ndrepepa G, Keta D, Byrne RA et al. Impact of body mass index on clinical outcome in patients with acute coronary syndromes treated with percutaneous coronary intervention. Heart Vessels, 2010; 25: 27–34.
7. Hadi HA, Zubaid M, Al Mahmeed W et al. The prevalence and outcome of excess body weight among Middle Eastern patients presenting with acute coronary syndrome. Angiology, 2010; 61: 456–464.
8. Kosuge M, Kimura K, Kojima S et al. Japanese Acute Coronary Syndrome Study (JACSS) Investigators. Impact of body mass index on in-hospital outcomes after percutaneous coronary intervention for ST segment elevation acute myocardial infarction. Circ J, 2008; 72: 521–525.
9. Janssen I. Morbidity and mortality risk associated with an overweight BMI in older men and women. Obesity (Silver Spring), 2007; 15: 1827–1840.
10. Nigam A, Wright RS, Allison TG et al. Excess weight at time of presentation of myocardial infarction is associated with lower initial mortality risks but higher long-term risks including recurrent re-infarction and cardiac death. Int J Cardiol, 2006; 110: 153–159.
11. Kang WY, Jeong MH, Ahn YK et al. Korea Acute Myocardial Infarction Registry Investigators. Obesity paradox in Korean patients undergoing primary percutaneous coronary intervention in ST-segment elevation myocardial infarction. J Cardiol, 2010; 55: 84–91.
12. Nikolsky E, Stone GW, Grines CL et al. Impact of body mass index on outcomes after primary angioplasty in acute myocardial infarction. Am Heart J, 2006; 151: 168–175.
13. Buettner HJ, Mueller C, Gick M et al. The impact of obesity on mortality in UA/non-ST-segment elevation myocardial infarction. Eur Heart J, 2007; 28: 1694–1701.
14. Mehta L, Devlin W, McCullough PA et al. Impact of body mass index on outcomes after percutaneous coronary intervention in patients with acute myocardial infarction. Am J Cardiol, 2007; 99: 906–910.
15. Eisenstein EL, McGuire DK, Bhapkar MV et al. Elevated body mass index and intermediate-term clinical outcomes after acute coronary syndromes. Am J Med, 2005; 118: 981-990.
16. Mahaffey KW, Tonev ST, Spinler SA et al. SYNERGY Trial Investigators. Obesity in patients with non-ST-segment elevation acute coronary syndromes: results from the SYNERGY trial. Int J Cardiol, 2010; 139: 123–133.
17. Nicoletti I, Cicoira M, Morando G et al. Impact of body mass index on short-term outcome after acute myocardial infarction: does excess body weight have a paradoxical protective role? Int J Cardiol, 2006; 107: 395–399.
18. Abdulla J, Kober L, Abildstrom SZ et al. Impact of obesity as a mortality predictor in high-risk patients with myocardial infarction or chronic heart failure: a pooled analysis of five registries. Eur Heart J, 2008; 29: 594–601.
19. Diercks DB, Roe MT, Mulgund J et al. The obesity paradox in non-ST-segment elevation acute coronary syndromes: results from the Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the American College of Cardiology/American Heart Association Guidelines Quality Improvement Initiative. Am Heart J, 2006; 152: 140–148.
20. Romero-Corral A, Montori VM, Somers VK et al. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. Lancet, 2006; 368: 666–678.
21. Boden WE, O'Rourke RA, Teo KK et al. COURAGE Trial Research Group. Optimal medical therapy with or without PCI for stable coronary disease. N Engl J Med, 2007; 356: 1503–1516.
22. Choy B, Hansen E, Moss AJ et al. Multicenter Automatic Defibrillator Implantation Trial-II Investigators. Relation of body mass index to sudden cardiac death and the benefit of implantable cardioverter-defibrillator in patients with left ventricular dysfunction after healing of myocardial infarction. Am J Cardiol, 2010; 105: 581–586.
23. Kennedy LM, Anker SD, Kjekshus J et al. Impact of neurohormonal blockade on association between body mass index and mortality. Int J Cardiol, 2007; 119: 33–40.
24. Hiley CR, Ford WR. Cannabinoid pharmacology in the cardiovascular system: potential protective mechanisms through lipid signalling. Biol Rev Camb Philos Soc, 2004; 79: 187–205.
25. Lepicier P, Bouchard JF, Lagneux C et al. Endocannabinoids protect the rat isolated heart against ischemia. Br J Pharmakol, 2003; 139: 805–815.
26. Steffens S, Veillard NR, Arnaud C, et al. Low dose oral cannabinoid therapy reduces progression of atherosclerosis in mice. Nature, 2005; 434: 782–786.
27. Fox KA, Poole-Wilson P, Clayton TC, et al. 5-year outcome of an interventional strategy in non-ST-elevation acute coronary syndrome: the British Heart Foundation RITA 3 randomised trial. Lancet, 2005; 366: 914–920.
28. Su L, Siegel JE, Fishbein MC. Adipose tissue in myocardial infarction. Cardiovasc Pathol, 2004; 13: 98–102.
29. Gurm HS, Whitlow PL, Kip KE; BARI Investigators. The impact of body mass index on short- and long-term outcomes inpatients undergoing coronary revascularization. Insights from the bypass angioplasty revascularization investigation (BARI). J Am Coll Cardiol, 2002; 39: 834–840.
30. Minutello RM, Chou ET, Hong MK et al. Impact of body mass index on in-hospital outcomes following percutaneous coronary intervention (report from the New York State Angioplasty Registry). Am J Cardiol, 2004; 93: 1229–1232.
31. Gurm HS, Brennan DM, Booth J et al. Impact of body mass index on outcome after percutaneous coronary intervention (the obesity paradox). Am J Cardiol, 2002; 90: 42–45.
32. Powell BD, Lennon RJ, Lerman A et al. Association of body mass index with outcome after percutaneous coronary intervention. Am J Cardiol, 2003; 91: 472–476.
33. Schulze PC, Kratzsch J, Linke A et al. Elevated serum levels of leptin and soluble leptin receptor in patients with advanced chronic heart failure. Eur J Heart Fail, 2003; 5: 33–40.
34. Zeller M, Steg PG, Ravisy J et al. RICO Survey Working Group. Relation between body mass index, waist circumference, and death after acute myocardial infarction. Circulation, 2008; 118: 482–490.
35. Lopez-Jimenez F, Wu CO, Tian X et al. Weight change after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease patients (ENRICHD) experience. Am Heart J, 2008; 155: 478–484.
36. Lancefield T, Clark DJ, Andrianopoulos N et al. Is there an obesity paradox after percutaneous coronary intervention in the contemporary era? An analysis from a multicenter Australian registry. MIG (Melbourne Interventional Group) Registry. JACC Cardiovasc Interv, 2010; 3: 660–668.

Czy pacjenci z nadwagą po zawale serca leczonym inwazyjnie mają lepsze rokowanie w 5-letniej obserwacji?

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Streszczenie

Wstęp: Otyłość i nadwaga mogą wpływać na przeżycie pacjentów po zawale serca niezależnie od wieku i sposobu leczenia.

Cel: Celem pracy była ocena wpływu wskaźnika masy ciała (BMI) na długoterminowe przeżycie u pacjentów z zawałem serca z uniesieniem odcinka ST (STEMI) leczonych pierwotną plastyką wieńcową (PCI).

Metody: W pracy prospektywnej oceniono homogenną grupę złożoną ze 131 pacjentów, ze STEMI oraz przepływem TIMI 3 po PCI. Dane pacjentów (41 kobiet, 90 mężczyzn, średni wiek 58,3 ± 10,8 roku) analizowano w 2 grupach: Grupa 1 — 30 (23%) osób z BMI < 25 kg/m^2 oraz Grupa 2 — 101 (77%) osób z BMI ≥ 25 kg/m^2.

Wyniki: Łącznie w czasie 5-letniej obserwacji 19 (14,5%) pacjentów zmarło — 9 z 30 (30%) z grupy 1 oraz 10 z 101 (10%) z grupy 2 ($p < 0,001$). Chorzy z BMI ≥ 25 kg/m^2 charakteryzowali się mniejszą śmiertelnością 5-letnią i było to niezależne od innych czynników. Pole pod krzywą w analizie ROC dotyczące występowania zgonów w zależności od BMI było istotnie różne niż pole pod krzywą dla przypadkowego modelu ($p < 0,05$). Nie zaobserwowano istotnych statystycznie różnic w śmiertelności 30-dniowej i rocznej ($p = 0,6517$ i $p = 0,3573$, odpowiednio).

Wnioski: Nie stwierdzono różnic w śmiertelności 30-dniowej i rocznej między pacjentami ze STEMI leczonymi za pomocą PCI z BMI < 25 kg/m^2 oraz chorymi z BMI ≥ 25 kg/m^2. Natomiast osoby z BMI ≥ 25 kg/m^2 charakteryzowały się lepszym 5-letnim przeżyciem i było to niezależne od innych czynników.

Słowa kluczowe: zawał serca z uniesieniem odcinka ST, przezskórna interwencja wieńcowa, otyłość, śmiertelność

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