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Original Article

Obesity Paradox and Long-term Cardiovascular Outcomes in Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention

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Abstract

Objectives: Obesity is a known modifiable risk factor for cardiovascular disease, and guidelines recommend maintaining a body mass index of 20–25 kg/m^2 to reduce cardiovascular risk. However, several studies have reported that obese patients with acute coronary syndrome (ACS) may have better short- and long-term survival than their normal-weight counterparts-a phenomenon known as the "obesity paradox." The effect of this paradox in ACS patients treated exclusively with percutaneous coronary intervention (PCI) remains unclear. This retrospective, two-center cohort study aimed to evaluate the impact of BMI on long-term mortality in ACS patients undergoing PCI, with a 3 years follow-up period.

Methods: A total of 1,198 patients were categorized according to BMI as normal weight (n=321), overweight (n=753), and obese (n=124). Survival across BMI categories was evaluated using Kaplan-Meier analysis with log-rank testing. Univariate and multivariable Cox proportional hazards regression models (backward likelihood ratio method) were performed to identify predictors of long-term mortality.

Results: In-hospital mortality was lowest among obese patients (2.2%) compared to overweight (2.8%) and normal-weight patients (7.4%) (χ^2=12.448; p=0.002). Over a 3 years follow-up, mortality remained significantly lower in the obese (6.5%) and overweight (8.5%) groups compared with the normal-weight group (20.1%) (χ^2=30.662; p<0.001). In multivariable analysis, smoking increased mortality risk by approximately 50% (Hazard ratio [HR]: 1.508; 95% confidence interval [CI]: 1.030–2.208; p=0.035), whereas obesity (HR: 0.297; 95% CI: 0.128–0.688; p=0.005) and overweight (HR: 0.404; 95% CI: 0.282–0.578; p<0.001) significantly reduced mortality risk.

Conclusion: Although obesity remains a significant risk factor for the development of cardiovascular disease, the present findings support the existence of an obesity paradox among ACS patients treated with PCI. Obese and overweight patients demonstrated a 60–70% lower risk of long-term mortality compared with normal-weight patients. These results highlight the need for a more nuanced approach to risk stratification and treatment planning in this patient group.

Keywords: Acute coronary syndrome, body mass index, obesity paradox, survival.

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Obesity is a well-recognized and modifiable risk factor for cardiovascular diseases, and it is commonly assessed through anthropometric measures, such as body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR). The presence of excess adipose tissue has been consistently linked with an increased

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risk of various metabolic and cardiovascular conditions, including type 2 diabetes mellitus, systemic hypertension, heart failure, and atrial fibrillation. Present international guidelines recommend maintaining a BMI between 20 and 25 kg/m^2 to lower cardiovascular risk and support long-term health.[1–3]

Interestingly, recent findings have described a counterintuitive observation known as the "obesity paradox," where individuals with obesity may experience more favorable clinical outcomes compared to their normal-weight counterparts after developing certain cardiovascular conditions. While the biological mechanisms behind this paradox are not yet fully understood, several hypotheses have been proposed. These include the protective role of higher lean body mass, reduced prevalence of malnutrition and cachexia, lower incidence of sarcopenia, and potentially greater metabolic and nutritional reserves in obese patients. In addition, differences in inflammatory and neurohormonal responses, as well as the potential effects of certain adipokines, have been suggested as contributing factors. It has been observed across different clinical groups, including patients with acute coronary syndrome – such as ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina – as well as individuals with heart failure and certain malignancies.[4–7] In these cases, obese patients have often shown lower short-term and long-term mortality rates than non-obese individuals.

Standard treatment strategies for ACS involve pharmacological therapy, percutaneous coronary intervention, and coronary artery bypass grafting (CABG). Although previous studies on the obesity paradox have included patients receiving a combination of these treatments, only a limited number have focused specifically on those treated solely with PCI. Considering the widespread use of PCI as a primary revascularization approach, investigating the role of obesity in this patient population holds significant clinical relevance. This study aims to explore the existence and prognostic impact of the obesity paradox in patients diagnosed with ACS who underwent PCI during their initial hospitalization and were followed for up to 3 years.[1–8]

Materials and Methods

We designed a retrospective cohort study by reviewing medical records from two high-volume secondary and tertiary care centers to identify patients who underwent percutaneous coronary intervention for acute coronary syndrome between 2015 and 2018.

A total of 2,563 patients were screened, of whom 1,198 met the eligibility criteria and were included in the final analysis. Inclusion criteria were diagnosis of ACS (STEMI, NSTEMI, or unstable angina) and treatment with PCI during the index hospitalization. Exclusion criteria were ACS patients who did not receive PCI, alternative diagnoses, such as myocarditis, Takotsubo cardiomyopathy, pulmonary embolism, or myocardial infarction with non-obstructive coronary arteries, referral for coronary artery bypass grafting, severe comorbid illness predicting a life expectancy under 1 year, missing BMI data, or BMI values ≤18 or ≥40 at the time of admission (Fig. 1).

After applying these criteria, patients were grouped based on their BMI, which was calculated using the Quetelet formula: Weight (kg) divided by height squared (m^2). According to the World Health Organization classification, patients were categorized as normal (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obese (≥30.0 kg/m^2).

All patients included were followed for a period of 3 years. The primary outcome was all-cause mortality. Secondary outcomes included recurrent myocardial infarction, development of atrial fibrillation, and occurrence of stroke.

The study protocol was reviewed and approved by the Manisa Celal Bayar University Ethics Committee (Protocol No: V01, 03.11.2021-E.182270, date: 03.11.2021). All procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Statistical Analysis

All statistical analyses were carried out using IBM Statistical Package for the Social Sciences Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY; Released 2017).

Descriptive data for continuous variables were summarized as means±standard deviations or medians with interquartile ranges (25th–75th percentile), depending on distribution.

For group comparisons, Student's t-test or the Mann-Whitney U test was applied, depending on the normality of the data. When comparing more than two independent groups, one-way Analysis of Variance or the Kruskal-Wallis test was used as appropriate, followed by Bonferroni or Dunn's test for post hoc comparisons.

Three-year survival outcomes were assessed using Kaplan-Meier analysis, stratified by BMI categories (normal weight, overweight, obese). The log-rank test was used to compare survival curves between groups. Univariate and multivariable Cox proportional hazard regression analyses were performed to identify predictors of long-term mortality. In the multivariable analysis, the backward

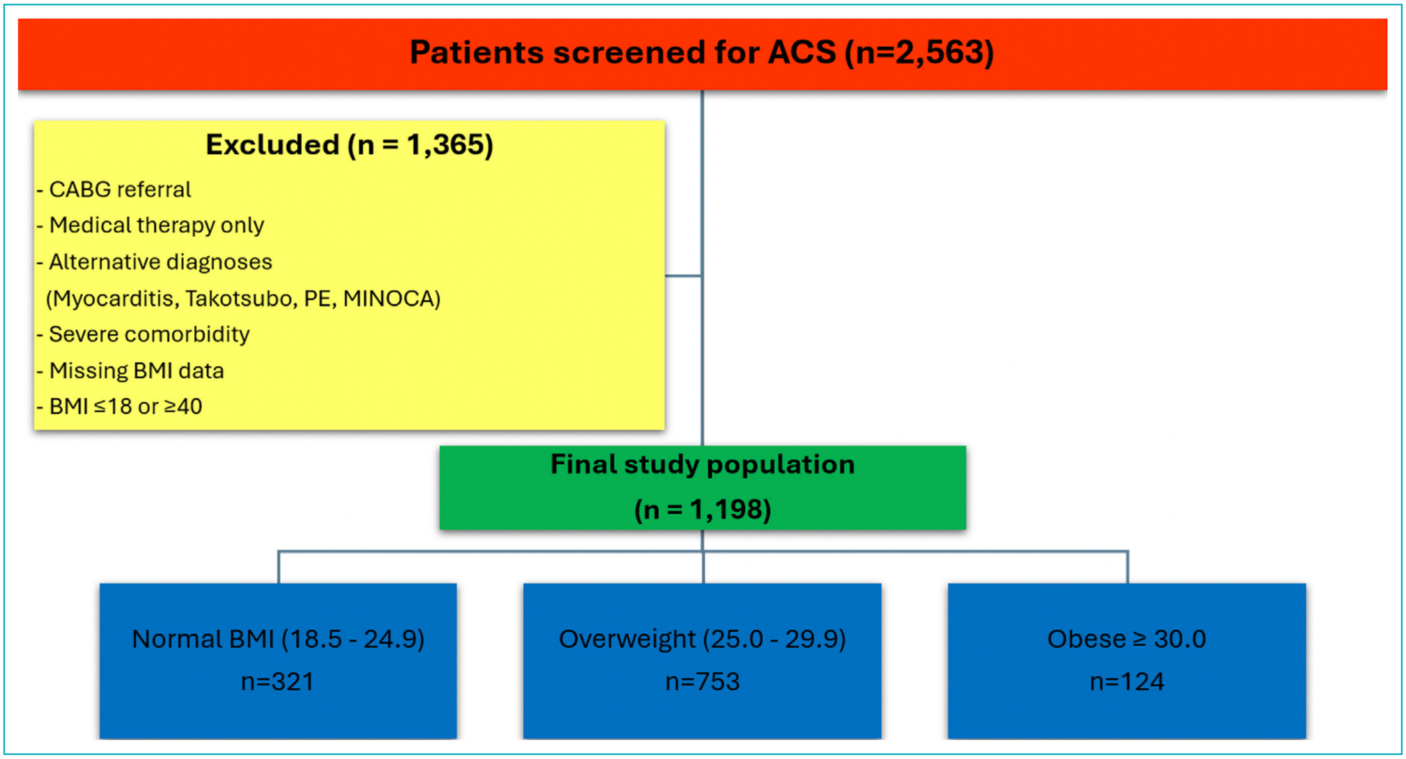

Figure 1. Flowchart of patient selection. A total of 2,563 patients with acute coronary syndrome were screened between 2015 and 2018. Of these, 1,365 were excluded due to referral for coronary artery bypass grafting, receipt of medical therapy only, alternative diagnoses (myocarditis, takotsubo cardiomyopathy, pulmonary embolism, myocardial infarction with non-obstructive coronary arteries), severe comorbid illness with life expectancy <1 year, missing body mass index [BMI] data, or BMI ≤18 or ≥40. The final study population included 1,198 patients treated with percutaneous coronary intervention, who were stratified by BMI categories into normal (n=321), overweight (n=753), and obese (n=124) groups.

likelihood ratio (LR) method was employed to construct the final model. A P-value below 0.05 was considered statistically significant throughout all analyses.

Results

A total of 2,563 patients diagnosed with acute coronary syndrome were screened. Among these, 1,365 patients were excluded due to reasons, such as referral for CABG, receiving conservative medical management, alternative diagnoses, missing BMI information, lack of adequate follow-up, or other clinical exclusions. The final study population consisted of 1,198 patients: 321 with normal BMI, 753 classified as overweight, and 124 identified as obese. Baseline demographic and clinical characteristics across BMI categories are presented in Table 1 (values reported as n [%] unless otherwise specified).

Compared to those with normal BMI, patients in the overweight and obese groups tended to be younger. The obese group also demonstrated a higher frequency of positive family history for cardiovascular risk factors. In terms of presentation, ST-elevation myocardial infarction was more commonly observed among obese patients. Laboratory parameters were largely similar between the groups, although levels of high-density lipoprotein cholesterol (HDL-C), high-sensitivity C-reactive protein (hs-CRP), and glomerular filtration rate (GFR) were significantly higher in the obese group (post hoc analyses are provided for each significant comparison in Table 1).

When comparing in-hospital mortality, the lowest rate was observed in the obese group (n=2; 2.2%), followed by the overweight group (n=19; 2.8%), and the highest rate was seen in the normal BMI group (n=24; 7.4%) (χ^2=12.448; p=0.002). All cell counts (n) are reported in Table 2.

Survival analysis over the 3 years follow-up period revealed that patients in the overweight and obese categories had significantly higher survival rates compared to those in the normal BMI group (mortality rates: 6.5%, 8.5%, and 20.1%, respectively; χ^2=30.662; p<0.001). The mean survival time was significantly longer in the overweight and obese groups compared to the normal BMI, with results expressed in months (Table 3). Kaplan–Meier survival curves (Fig. 2) further illustrate this difference, showing a clear separation of survival trajectories by BMI category, with overweight and obese patients demonstrating better outcomes.

Table 1. Baseline characteristics stratified by BMI categories

Number of patients	Normal weight n=321		Overweight n=753		Obese n=124		Test statistics; p	Post hoc comparisons
	n	%	n	%	n	%		
Age med [Q1–Q3]	63.5 [53.0–74.0]		62.0 [50.0–71.5]		60.0 [52.0–72.0]		**KW=6.813; 0.033**	**0–2: 0.035**
Sex male	214	28.6	466	62.4	67	9.0	χ^2=1.885	0.393
Sex female	110	32.5	202	59.8	26	7.7		
Family history, yes	57	47.1	58	47.9	6	5.0	**χ^2=19.749 <0.001**	
Diagnosis								
Non-ST MI	109	33.6	281	42.1	28	30.1	**χ^2=26.741 <0.001**	
ST elevation MI	90	27.8	203	30.4	45	48.4		
UAP	125	38.6	184	27.5	20	21.5		
Risk factors								
Smoking, yes	194	59.9	355	53.1	51	54.8	χ^2=4.010	0.135
Diabetes mellitus, yes	104	32.1	202	30.2	26	28.0	χ^2=0.690	0.708
Hypertension, yes	178	54.9	407	60.9	48	51.6	χ^2=5.115	0.077
Hyperlipidemia, yes	170	52.5	320	47.9	51	54.8	χ^2=2.826	0.243
CAD, yes	188	58.0	336	50.3	49	52.7	χ^2=5.225	0.074
CABG, yes	37	11.4	51	7.6	7	7.5	χ^2=4.105	0.128
hs-CRP med [Q1–Q3]	5.5 [3.1–8.1]		7.2 [4.5–9.6]		6 [4.6–7.9]		**KW=13.5; 0.001**	0–1; 0.020
Total cholesterol med [Q1–Q3]	197 [178.8–215]		208 [186–221.5]		200 [187.3–218]		KW=4.54; 0.103	
Triglyceride med [Q1–Q3]	168 [129–211.3]		186 [134.5–231]		183.5 [147–195]		KW=3.19; 0.202	
Laboratory								
Fasting glucose med [Q1–Q3]	131.5 [113.8–159]		129 [103.5–162]		120 [113–189.8]		KW=0.237; 0.888	
HDL med [Q1–Q3]	40 [35–51.3]		39 [35–47]		42 [40–47.3]		**KW=8.77; 0.012**	1–2: 0.014
LDL med [Q1–Q3]	114 [90.8–141]		125 [100.5–146]		126 [95–142.3]		KW=3.13; 0.209	
HbA1c med [Q1–Q3]	6.0 [5.5–6.5]		5.9 [5.6–6.7]		6.1 [5.3–7.3]		KW=3.64; 0.162	
GFr med [Q1–Q3]	68.6 [48.4–97.2]		89.0 [52.3–115]		94.4 [65.9–112.6]		**KW=28.0; <0.001**	0–1: <0.001 0–2: <0.001
Urea med [Q1–Q3]	38.5 [32–45.3]		32 [25–43]		37 [26.8–40]		**KW=13.4; 0.001**	0–1: <0.001
Creatinine med [Q1–Q3]	0.93 [0.8–1.1]		0.91 [0.8–1.2]		0.98 [0.8–1.1]		KW=0.925; 0.630	
EKG								
EKG AF	36	11.4	74	10.0	13	10.6	χ^2=0.450; 0.798	
EKG SR	281	88.6	667	90.0	110	89.4		
Drugs after discharge								
Acetyl salicylic acid	309	95.4	659	98.7	86	92.5	**χ^2=16.461; 0.129**	
Clopidogrel	166	51.2	429	64.2	65	69.9	**χ^2=18.950; <0.001**	
Ticagrelor	85	43.8	126	43.6	18	40.0	χ^2=0.230; 0.895	
Prasugrel	35	10.8	49	7.3	4	4.3	χ^2=5.500; 0.064	
Beta-blocker	142	43.8	374	56.0	62	66.7	**χ^2=20.293; <0.001**	
Ace inh/ARB	156	48.1	373	55.8	48	51.6	χ^2=5.282; 0.071	
Statin	278	85.8	499	74.7	85	91.4	**χ^2=25.36; <0.001**	
Coumadin	16	5.0	22	3.4	3	3.3	χ^2=1.614; 0.446	
NOAC	16	4.9	26	3.9	7	7.5	χ^2=2.692; 0.260	

χ^2: Chi-square test, KW: Kruskal Wallis test, BMI: Body mass index, MI: Myocardial infarction, ST elevation MI: ST-Segment elevation myocardial infarction, UAP: Unstable angina pectoris, CAD: Coronary artery disease, CABG: Coronary artery bypass grafting, hs-CRP: High-sensitivity C-reactive protein, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HbA1c: Hemoglobin A1c, GFR: Glomerular filtration rate, EKG: Electrocardiogram, AF: Atrial fibrillation, SR: Sinus rhythm, ACE inh: Angiotensin-converting enzyme inhibitor, ARB: Angiotensin II receptor blocker, NOAC: Non-Vitamin K antagonist oral anticoagulant, CVE: Cerebrovascular event, "Code: 0=Normal weight, 1=Overweight, 2=Obese", p<0.05 significance level.

Table 2. Endpoints according to BMI in the hospital and long-term

	Normal weight		Overweight		Obese		Test statistics; p
	n	%	n	%	n	%	
Death (overall)	65	20.1	57	8.5	6	6.5	χ^2=30.662; <0.001
Survived (overall)	256	79.9	696	91.5	118	93.5	
Death in Hospital	24	7.4	19	2.8	2	2.2	χ^2=12.448; 0.002
Alive at discharge	297	92.6	734	97.2	122	97.8	
Seconder endpoint							
MI_{have}	11	3.4	25	3.7	2	2.2	χ^2=0.628; 0.731
MI_{not}	310	96.6	728	96.3	122	97.8	
CVE_{have}	32	9.9	43	6.4	3	3.2	χ^2=6.263; 0.054
CVE_{not}	289	90.1	710	93.6	121	96.8	
AF development	33	10.2	72	10.8	3	3.2	χ^2=5.223; 0.073
AF_{not}	288	89.8	681	89.2	121	96.8	

χ^2: Chi-square test. BMI: Body mass index, AF: Atrial fibrillation, MI: Myocardial infarction, CVE: Cerebrovascular event, p<0.05 significance level.

Table 3. Kaplan Meier survival analysis of all-cause mortality in normal weight, overweight, and obese patients at 3-years follow-up

BMI (kg/m²)	Means for survival time (months)				Log rank (Mantel-Cox)		
	Estimate	SE	95% CI		Normal weight	Overweight	Obese
			Lower bound	Upper bound	Test statistics; p	Test statistics; p	Test statistics; p
Normal weight	31.892	0.613	30.690	33.094		**χ^2=27.427; <0.001**	**χ^2=9.053; 0.003**
Overweight	34.882	0.289	34.315	35.449	**χ^2=27.427; <0.001**		χ^2=0.454; 0.501
Obese	35.323	0.692	33.966	36.679	χ^2=9.053; 0.003	χ^2=0.454; 0.501	
Overall	34.027	0.266	33.506	34.547			

χ^2: Chi-square test, p<0.05 Significance level. BMI: Body mass index, SE: Standard error, CI: Confidence interval.

There were no statistically significant differences between BMI groups in secondary outcomes, such as recurrent myocardial infarction (χ^2=0.628; p=0.731), atrial fibrillation (χ^2=5.223; p=0.073), or stroke (χ^2=6.063; p=0.054), as displayed in Table 2.

In multivariable Cox regression analysis, factors, such as advanced age, NSTEMI presentation, and smoking status were associated with increased mortality risk. Conversely, both overweight and obesity were identified as protective factors. Specifically, smoking was associated with a ~50% increased mortality risk (Hazard ratio [HR]: 1.508; 95% confidence interval [CI]: 1.030–2.208; p=0.035), whereas overweight and obesity were associated with substantial reductions in mortality risk (overweight: HR 0.404; 95% CI 0.282–0.578; p<0.001, corresponding to ~60% risk reduction; obese: HR 0.297; 95% CI 0.128–0.688; p=0.005, corresponding to ~70% risk reduction). These findings, summarized in Table 4, remained significant after adjustment for potential confounders, such as age and comorbidities.

Discussion

This study investigated the impact of BMI on long-term outcomes in patients with acute coronary syndrome undergoing percutaneous coronary intervention. The analysis demonstrated that overweight and obese patients experienced significantly lower in-hospital and 3 years mortality compared with normal-weight individuals,

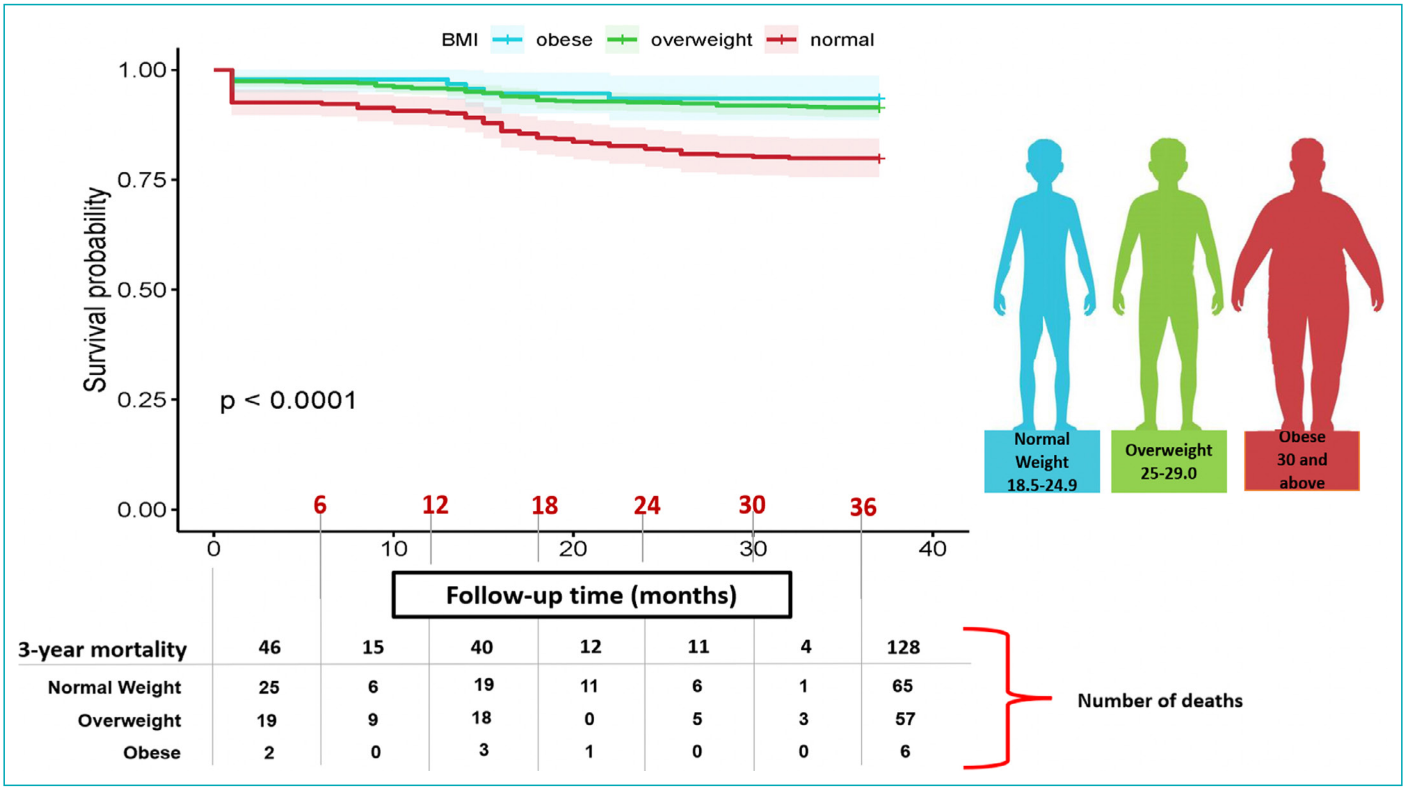

Figure 2. Survival analysis for all-cause mortality in normal weight, overweight, and obese patients at 3-year follow-up.

BMI: Body mass index.

Table 4. Univariate and multivariable predictors of long-term mortality

	Univariate analysis				Multivariable analysis			
	p	HR	95%CI for HR		p	HR	95%CI for HR	
			Lower bound	Upper bound			Lower bound	Upper bound
Age	**<0.001**	0.975	0.961	0.988	**0.001**	**0.976**	0.963	0.990
Diagnosis	**0.043**		**0.022**					
ST elevation MIUAP	0.088	0.692	0.453	1.057	0.718	1.086	0.693	1.702
Non-ST MI UAP	**0.016**	0.602	0.398	0.911	**0.011**	**1.726**	1.135	2.624
Diabetes, no	0.378	0.840	0.569	1.239				
Hypertension, no	0.740	1.061	0.746	1.510				
Hyperlipidemia, no	0.137	1.303	0.919	1.848				
Family history, no	0.749	0.914	0.515	1.622				
Smoking, no	**0.005**	1.698	1.174	2.456	**0.035**	**1.508**	1.030	2.208
CAD, no	0.122	1.321	0.929	1.880				
CABG, no	0.056	1.645	0.988	2.741				
BMI	<0.001		<0.001					
BMI obese	**0.005**	0.303	0.131	0.698	**0.005**	**0.297**	0.128	0.688
BMI overweight	**<0.001**	0.402	0.282	0.574	**<0.001**	**0.404**	0.282	0.578

Age: Continuous variable, Diagnosis: Acute MI: 0, UAP: 1, NSTEMI: 2, Diabetes: No: 0, Yes: 1, Hypertension: No: 0, Yes: 1, Hyperlipidemia: No: 0, Yes: 1, BMI: 25 and upper: 0, 18.5–24.9:1. HR: Hazard ratio, CI: Confidence interval, MI: Myocardial infarction, UAP: Unstable angina pectoris, NSTEMI: Non-ST Segment elevation myocardial infarction, CAD: Coronary artery disease, CABG: Coronary artery bypass grafting, BMI: Body mass index.

supporting the presence of an obesity paradox in this population. Specifically, overweight and obesity were associated with approximately 60% (HR 0.404; 95% CI 0.282–0.578) and 70% (HR 0.297; 95% CI 0.128–0.688) reductions in mortality risk, respectively, relative to normal BMI ($p<0.01$).

Low-grade chronic inflammation represents a biological link between excess adiposity and cardiovascular disease. Elevated high-sensitivity C-reactive protein levels in the overweight and obese groups in this study are consistent with large epidemiologic cohorts, such as NHANES III,[9] which also reported increased hs-CRP with rising BMI after controlling for confounders. Similarly, Festa et al.[10] and Pou et al.[11] demonstrated strong correlations between adiposity indices (BMI, waist circumference [WC]) and inflammatory biomarkers (CRP, interleukin-6), emphasizing the contribution of visceral fat to systemic inflammation. The persistence of elevated hs-CRP among overweight and obese patients in the present study may partly explain the earlier onset of ACS observed in these subgroups, as chronic inflammation accelerates atherosclerotic plaque formation.[12]

In addition to inflammatory markers, the study revealed higher glomerular filtration rate and unexpectedly elevated HDL-C levels in overweight and obese patients. Although obesity is known to be a risk factor for long-term kidney damage, elevated GFR may reflect early compensatory changes or measurement bias related to body size. In addition, the study revealed unexpectedly higher HDL levels in obese patients. The higher HDL-C values, which diverge from classical dyslipidemic profiles of obesity, might be attributed to metabolic alterations during the acute phase of ACS or variability in fasting status at the time of sampling.[13–15]

Multivariable Cox regression analysis identified age above 65 years, NSTEMI presentation, and smoking as independent predictors of long-term mortality, consistent with established risk factors in ACS populations. Smoking was associated with approximately a 50% higher risk of death (HR 1.51; 95% CI 1.03–2.21; p=0.035). In contrast, both overweight and obesity were independently linked to improved survival, in agreement with findings from previous studies.[16,17] The Kaplan–Meier survival curves clearly demonstrated this pattern, showing the best survival outcomes among obese patients and confirming an inverse association between BMI and mortality over the 3 years follow-up period.

A large meta-analysis by Lamelas et al.,[18] including more than 81,000 ACS patients, similarly found that underweight individuals had higher mortality than those of normal or slightly elevated BMI, while overweight and mildly obese patients had the best survival, with the lowest mortality near 30.9 kg/m^2. Gruber et al.[19] also reported better long-term outcomes after PCI among patients with higher BMI, identifying age, diabetes, and left-ventricular function as key predictors of mortality.

Several mechanisms may underline the apparent protective effect of higher BMI in ACS patients treated with PCI. Patients of normal weight are often older and have more comorbidities, whereas those who are overweight or mildly obese may possess greater physiological reserves, such as higher lean muscle mass, lower prevalence of sarcopenia, and reduced likelihood of malnutrition. These features could enhance tolerance to acute illness and recovery after PCI. Furthermore, BMI does not differentiate between fat and muscle tissue; therefore, individuals categorized as normal weight may include patients with low muscle mass or frailty, conditions known to worsen outcomes.[20,21]

From a clinical perspective, these findings suggest that weight-management strategies should be individualized rather than uniformly aggressive in ACS patients undergoing PCI. While excessive adiposity remains an established risk factor for developing coronary artery disease, once ACS occurs, modest overweight or mild obesity may not translate into poorer prognosis and could even be associated with better long-term survival. Incorporating other anthropometric and metabolic indicators – such as WC, visceral fat, and body composition – into clinical risk assessment may help refine prognostic evaluation beyond BMI alone.

Despite these findings, this study has several limitations. First, we relied exclusively on BMI for categorization, without including additional anthropometric measures, such as WC, WHtR, or direct assessments of visceral adiposity, which might provide stronger prognostic value. Second, although the study included a relatively large sample size and a 3 years follow-up, it was retrospective in design, and residual confounding by unmeasured variables, such as nutritional status, physical activity, and socioeconomic factors cannot be excluded. Third, we assessed all-cause mortality but did not differentiate between cardiac and non-cardiac deaths. Evaluating cause-specific mortality in future studies could offer greater insight into the prognostic relevance of BMI in acute coronary syndrome patients treated with percutaneous coronary intervention. Finally, while our findings are consistent with the obesity paradox described in previous studies, they may not be generalizable to populations treated with coronary artery bypass grafting or medical therapy alone, as our cohort included only patients who underwent percutaneous coronary intervention.

Conclusion

Obesity and being overweight are well-recognized risk factors for the development of cardiovascular disease. In this cohort of patients with acute coronary syndrome treated with percutaneous coronary intervention, however, both overweight and obesity were associated with lower long-term mortality, supporting the presence of an obesity paradox. These findings emphasize the need for more refined risk stratification in acute coronary syndrome, as BMI alone may not fully capture the prognostic impact of body composition. The results also suggest that aggressive weight-reduction strategies may not be universally beneficial in this population and should be carefully individualized. Further studies incorporating additional anthropometric and body-composition measures are warranted to improve clinical decision-making and guide secondary prevention strategies.

Disclosures

Ethics Committee Approval: The study was approved by the Manisa Celal Bayar University Ethics Committee (no: V01, 03.11.2021-E.182270, date: 03/11/2021).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: All authors declared no conflict of interest.

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