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Original Article CARDIOLOGY

Body fat percentage and infarct size in patients with non-ST segment elevation myocardial infarction

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ABSTRACT

OBJECTIVE: Obesity is a global health problem that increases the risk of coronary artery disease (CAD). However in studies, it has been observed that when the disease develops, obese patients have a more favorable prognosis than leaner patients. This is called the "obesity paradox." This study aims to evaluate the effect of obesity assessed with body fat percentage (BFP) and relative fat mass (RFM) besides body mass index (BMI) on infarct size (IS) estimated from peak creatine kinase-MB (CK-MB) levels in patients with non-ST-segment elevation myocardial infarction (NSTEMI).

METHODS: Patients with a diagnosis of NSTEMI who underwent coronary angiography between January 2017 and January 2022 were retrospectively evaluated. Patients without available anthropometric data to calculate BMI, BFP, and RFM and serial CK-MB measurements were excluded from the study. BMI was calculated using weight(kg)/(height[m])^2 formula. Patients were dichotomized as obese (BMI≥30 kg/m^2) and non-obese (BMI<30 kg/m^2) to compare baseline characteristics. BFP and RFM were calculated from anthropometric data. Linear regression analysis was performed to define predictors of IS.

RESULTS: Final study population consisted of 748 NSTEMI patients (mean age was 59.3±11.2 years, 76.3% were men, 36.1% of the patients were obese). Obese patients were more likely to be female, hypertensive, and diabetic. Smoking was less frequently observed in obese patients. Peak CK-MB levels were similar among groups. Obese patients had higher in-hospital left ventricular ejection fraction, and less severe CAD was observed in coronary angiographies of these patients. Multivariable regression analysis identified diabetes mellitus, systolic blood pressure, white blood cell count, hemoglobin, and BFP (β=-4.8, 95% CI=-8.7; -0.3, p=0.03) as independent predictors of IS.

CONCLUSION: Higher BFP is associated with smaller IS in NSTEMI patients. These findings support the obesity paradox in this patient group, but further, randomized controlled studies are required.

Keywords: Body fat percentage; infarct size; non-ST-segment elevation myocardial infarction; obesity; peak creatine kinase-MB.

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Obesity is a growing global health issue with increased mortality in the general population [1]. The World Health Organization reported that, in 2016, over 650 million adults (13% of the adult population) were obese [2]. Obesity, abnormal accumulation of fat in the body, is defined as body mass index (BMI) ≥30 kg/m^2. BMI is calculated from weight and height measurements and is widely used as an indicator of obesity, although it does not consider body composition or fat distribution. Therefore, a need to consider other indices in addition

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to BMI with a greater ability to differentiate fat mass from lean mass and reflect central obesity has emerged. Although several techniques for accurate assessment of body fat percentage (BFP), such as dual-energy x-ray absorptiometry, magnetic resonance imaging (MRI), and computed tomography exist, they are not available in all clinics and their cost makes them unsuitable for routine clinical use. For this reason, some equations that estimate BFP and have a correlation with these sophisticated techniques were developed. U.S. Navy equation of BFP based on waist, neck, hip circumferences, and height has been used since 1984 for this purpose [3]. Recently, the relative fat mass (RFM) index calculated from the height-to-waist ratio has been proposed [4].

Obesity predisposes people to cardiovascular diseases and therefore is regarded as a preventable risk factor. However, literature suggests that after the development of the disease, obesity is related to better prognosis which is defined as "obesity paradox." Obesity paradox was also reported in acute coronary syndromes (ACS) and non-ST-segment elevation myocardial infarction (NSTEMI) [5, 6].

NSTEMI, accounting for approximately 60–70% of ACS, is a major cause of morbidity and mortality worldwide [7]. Infarct size (IS) and left ventricular remodeling are major determinants of left ventricular dysfunction and mortality after ACS [8, 9]. Although several methods can be used, cardiac biomarkers routinely used in clinical practice are easy and inexpensive methods to estimate IS. Evidence suggests that peak creatine kinase-MB (CK-MB) correlates significantly with IS assessed with single-photon emission computed tomography and cardiac MRI in both NSTEMI and ST-segment elevation myocardial infarction (STEMI) [10–12].

There are conflicting results about the effect of obesity on IS in ACS. Therefore, we aimed to evaluate the effect of obesity assessed with several indices (BMI, BFP, and RFM) on IS estimated from peak CK-MB levels in patients with NSTEMI.

Highlight key points

- Obesity is a common entity in non-ST-segment elevation myocardial infarction.
- Increased body fat percentage but not body mass index is related with reduced infarct size in this patient group which supports obesity paradox defined in the literature.
- Although obesity is shown to be associated with more favorable outcomes after acute coronary syndrome, it should be evaluated with more accurate parameters than body mass index and treated as it is a modifiable risk factor for cardiovascular diseases.

MATERIALS AND METHODS

We analyzed the data of consecutive patients diagnosed with NSTEMI and underwent coronary angiography (CAG) between January 1, 2017, and January 1, 2022 in this single-center retrospective observational study. NSTEMI was diagnosed in patients with typical chest pain with no-persistent ST-segment elevation and elevated cardiac biomarkers [7, 13]. Patients with malignancies, end-stage renal disease (estimated glomerular filtration rate <30 mL/min/1.73 m^2), missing anthropometric data and patients without serial cardiac biomarker measurements to determine peak CK-MB were excluded from the study. The study was approved by the Haydarpasa Numune Training and Research Hospital Clinical Ethics Committee (approval date/number: June 05, 2023/ HNEAH-KAEK 2023/KK/103) and conducted in accordance with the Declaration of Helsinki.

A total of 748 patients fulfilled the inclusion criteria. Demographic data, cardiovascular risk factors and medical history (smoking status, hypertension, diabetes mellitus, hyperlipidemia, renal and pulmonary diseases, medication use), vital signs, and Killip class at admission were collected from medical records.

Anthropometric Measurements and Calculation of Obesity Indices

Weight, height, waist, hip, and neck circumferences of the patients were obtained after admission to the hospital using standard methods. Measurements were done with a nonstrechable standard tape. BMI was calculated as weight in kilograms divided by the square of the height in meters. Study participants were grouped according to the presence of obesity according to BMI (BMI <30 kg/m^2, BMI ≥30 kg/m^2) to compare baseline characteristics.

Waist circumference was obtained horizontally at the navel level for men and at the smallest width for women. Hip circumference was obtained horizontally at the level of the largest extension. Neck circumference was obtained just below the larynx.

BFP was calculated with the following U.S. Navy formula: $BFP(for men)=495/(1.0324-0.19077\times\log_{10}[waist-neck]+0.15456\times\log_{10}[height])-450$; $BFP\ (for\ women)=495/(1.29579-0.35004\times\log_{10}[waist+hip-neck]+0.22100\times\log_{10}[height])-450$ [3].

RFM index was calculated with the following formula: 64-(20×height/waist)+(12×sex), in which sex=0 for men and 1 for women [4].

Laboratory Parameters

Venous blood samples were obtained at admission (complete blood count, glucose, creatinine, C-reactive protein, troponin, CK-MB) and after overnight fasting for lipid parameters. Cardiac biomarkers were re-evaluated every 12 h according to routine clinical practice. Maximum CK-MB level was noted as peak CK-MB and used as a measure of IS.

Angiographic and Transthoracic Echocardiographic Examination

All patients received guideline-directed optimal medical therapy and underwent CAG during index hospitalization according to current guidelines using standard techniques. Angiographic images were recorded for offline analysis. Images were analyzed by two experienced interventional cardiologists blinded to patients' clinical data. Number of coronary arteries with ≥50% luminal stenosis were identified, and CAG results were categorized as non-obstructive coronary artery disease (CAD), single-vessel CAD, two-vessel CAD, and three-vessel CAD. Decisions related to treatment strategies were left to the discretion of the attending interventional cardiologists.

Transthoracic echocardiographic examination was performed before discharge to all patients according to current guidelines. Left ventricular ejection fraction (LVEF) was calculated using the two-dimensional biplane method of disks [14].

The primary objective of the study was to evaluate the effect of obesity assessed with different indices (BMI, RFM, and BFP) on IS evaluated with peak CK-MB.

Statistical Analysis

Normality was tested using the Kolmogorov–Smirnov test. Categorical variables are presented as numbers (percentages) and compared using the Chi-square test or Fisher's exact test. Continuous variables are presented as mean±standard deviation or median (interquartile range) and were compared using Student's t-test or Mann–Whitney U test, as appropriate. The associations between obesity indicators (BMI, RFM, and BFP) and peak CK-MB (as a measure of IS) were assessed using multiple linear regression. Variables with a $p<0.2$ in univariable analysis were included in the multivariable model. Goodness-of-fit of the linear regression models was assessed with R^2. Statistical significance was set at two-tailed $p<0.05$. Statistical analysis was performed using R 4.01 software (Vienna, Austria: R Foundation for Statistical Computing) with "ipw," "ggplot," "rms" packages.

RESULTS

Among 748 consecutive patients with NSTEMI, the mean age was 59.3±11.2 years and 76.3% were men. The mean BMI of the study group was 28.86±5.69 kg/m^2. 17% (n=127) of the patients had normal BMI, 46.5% (n=348) of the patients were overweight and 36.1% (n=270) of the patients were obese. Only 3 patients (0.4% of the group) were underweight. The mean BFP and RFM of the group were 29.97±10.28% and 32.98±7.46, respectively.

Table 1 presents the baseline characteristics of the study group divided into two as obese and non-obese according to BMI. The mean age was similar among groups. There were significantly more female patients in the obese group. Non-obese patients were more often smokers. Hypertension and diabetes mellitus were more commonly observed in obese patients, whereas no significant difference was observed in the prevalence of hyperlipidemia, previous CAD, chronic kidney disease, and lung diseases. Killip class and vital signs at admission were similar except for higher heart rate in obese patients.

Complete blood count parameters, admission blood glucose, creatinine, and C-reactive protein levels were comparable between the two groups. Regarding lipid parameters, obese patients had significantly higher triglyceride and lower high-density lipoprotein cholesterol levels. Total cholesterol and low-density lipoprotein cholesterol levels were similar among groups. Furthermore, peak CK-MB levels of the groups stratified according to BMI did not differ significantly. Obese patients had higher in-hospital LVEF than non-obese patients.

In CAG, three-vessel CAD was more frequent in non-obese patients (18.6% vs. 11.9%, p=0.016), whereas non-obstructive CAD was more frequent in obese patients (20.7% vs. 13.6%, p=0.011). As a result, revascularization by either percutaneous coronary intervention or coronary artery bypass grafting was performed more frequently in non-obese patients (68.8% vs. 60.7%, p=0.03).

All anthropometric measurements varied significantly among groups. Obese patients had significantly

Table 1. Baseline characteristics of the study group according to the presence of obesity

	Non-obese (BMI<30 kg/m^2, n=478)	Obese (BMI≥30 kg/m^2, n=270)	p
Age (years)	59.7±11.6	58.8±10.3	0.29
Male (%)	82.4	65.6	<**0.001**
Hypertension (%)	49.4	65.2	<**0.001**
Diabetes mellitus (%)	34.5	48.9	<**0.001**
Hyperlipidemia (%)	40.2	38.5	0.65
Current smoker (%)	48	40.4	**0.04**
COPD/OSAS (%)	6.9	7.7	0.46
Chronic kidney disease (%)	5.5	5.9	0.52
Previous CAD (%)	23.6	25.2	0.63
Systolic blood pressure (mmHg)	148±27	149±28	0.81
Diastolic blood pressure (mmHg)	84±16	84.3±16	0.98
Heart rate (beats/min)	81±20	86±24	**0.005**
Killip class at admission (%)			0.37
I	97.5	95.9	
II	1	1.1	
III	1.5	3	
Left ventricular ejection fraction (%)	50.9±9.7	53.5±8.9	<**0.001**
Anthropometric parameters			
Height (meters)	1.69± 0.08	1.65 ± 0.09	<**0.001**
Body weight (kilograms)	75±10.6	92±13.5	<**0.001**
Neck circumference (cm)	39.2±3	41.7±3	<**0.001**
Waist circumference (cm)	95±8.5	109±9	<**0.001**
Hip circumference (cm)	95±5.5	106±9	<**0.001**
Body mass index (kg/m^2)	26.4±2.5	34±4.5	<**0.001**
Relative fat mass	30.2±5.8	37±6.7	<**0.001**
Body fat percentage	25.7±6.7	36.9±10.4	<**0.001**
Laboratory parameters			
White blood cell (x10^9/L)	9.8±3.7	10.1±3	0.18
Hemoglobin (g/dL)	14.2±4.4	14.1±1.9	0.47
Platelet (x10^9/L)	230±64	242±60	0.07
Admission blood glucose (mg/dL)	141±78	151±76	0.09
Creatinine (mg/dL)	1.03±0.76	0.94±0.3	0.06
Total cholesterol (mg/dL)	194±49	192±44	0.47
Triglyceride (mg/dL)	184±143	228±162	<**0.001**
HDL cholesterol (mg/dL)	36.3±9.2	34±7.8	<**0.001**
LDL cholesterol (mg/dL)	124±61	120±40	0.21
Peak CK-MB (IU/L)	143 (79–354)	132 (85–256)	0.59
C-reactive protein (mg/dL)	0.6 (0.2–1.4)	0.7 (0.3–1.7)	0.10
Angiographic parameters			
Number of diseased coronary vessels (%)			**0.01**
No obstructive CAD	13.6	20.7	
Single-vessel CAD	45.8	44.1	
Two-vessel CAD	22	23.3	
Three-vessel CAD	18.6	11.9	
LMCA disease (%)	5.3	2.6	0.08
Treatment strategy (%)			**0.03**
Medical	31.3	39.3	
PCI	53.1	50	
CABG	15.7	10.7	

Categorical variables are expressed as percentages. Continuous variables are expressed as mean±standard deviation or median (interquartile range), as appropriate. CABG: Coronary artery bypass grafting; CAD: Coronary artery disease; CK-MB: Creatine kinase MB isoenzyme; COPD: Chronic obstructive pulmonary disease; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; LMCA: Left main coronary artery; OSAS: Obstructive sleep apnea syndrome; PCI: Percutaneous coronary intervention. Bold p values indicate statistical significance.

Table 2. Univariate and multivariate linear regression analysis for the prediction of infarct size

	Univariate analysis		Multivariate analysis	
	β coefficient (95% CI)	p	β coefficient (95% CI)	p
Age (years)	-1.16 (-6.47;4.16)	0.55		
Hypertension	-23.1 (-139;92.6)	0.69	–	–
Diabetes mellitus	-87 (-210;36.4)	0.16	-145 (-275;15)	0.03
Previous CAD	-65.6 (-202;70.4)	0.34	–	–
Current smoker	86.2 (-28.9;201)	0.14	-52 (-176;71.9)	0.41
Systolic blood pressure (mmHg)	2.89 (1.78;4.98)	0.01	3.1 (1.1–5.1)	0.002
Heart rate (beats/min)	0.90 (-1.66;3.47)	0.49	–	–
Three-vessel CAD	180 (23;337)	0.02	62.9 (-97;223)	0.44
White blood cell, (x10^9/L)	55.6 (39.7;71.6)	<0.001	59.2 (41.8;76.6)	<0.001
Hemoglobin (g/dL)	24 (-7.9;56)	0.14	51.8 (16.8;87)	0.004
Creatinine (mg/dL)	19.5 (-98;137)	0.75	–	–
Total cholesterol (mg/dL)	-0.55 (-1.87;0.76)	0.41	–	–
C-reactive protein (mg/dL)	18.5 (-4.6;41.6)	0.11	7.4 (-32;17.2)	0.55
Body mass index (kg/m^2)	-9.15 (-19.8;1.49)	0.09		
Relative fat mass	-8.7 (-16.4; -1.1)	0.02		
Body fat percentage (%)	-6.3 (-11.7; -1)	0.02		

CAD: Coronary artery disease; CI: Confidence interval.

Table 3. Multivariate linear regression models for infarct size comparing different obesity indices

	β coefficient (95% CI)	p	R^2
BMI model	-8.9 (-20.5; 2.7)	0.13	0.188
RFM model	-5.9 (-14.4; 2.5)	0.17	0.187
BFP model	-4.8 (-8.7; -0.3)	0.03	0.192

All three models were adjusted for white blood cell, diabetes mellitus, systolic blood pressure, smoking, C-reactive protein, hemoglobin and three-vessel coronary artery disease. BFP: Body fat percentage; BMI: Body mass index; CI: Confidence interval; RFM: Relative fat mass.

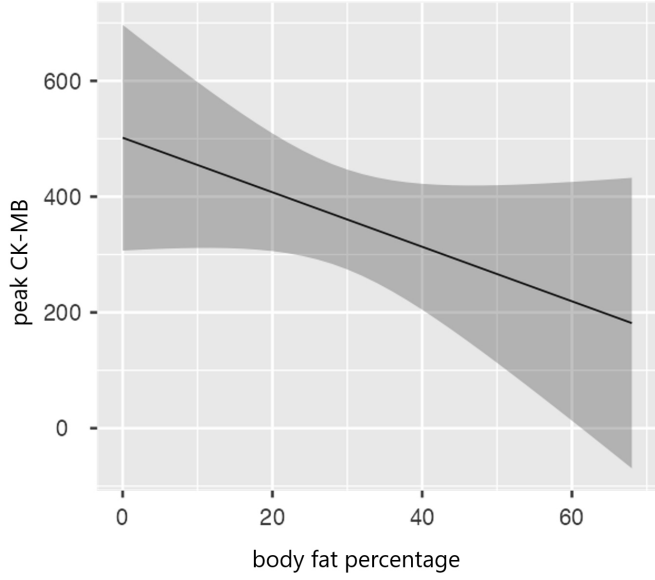

Figure 1. Marginal effect plot demonstrating association between peak CK-MB and BFP.

higher waist circumference (109±9 cm vs. 95±8.5 cm), RFM (37±6.7 vs. 30.2±5.8), and BFP (36.9±10.4% vs. 25.7±6.7%) than non-obese patients (p<0.001, for all).

BMI was not associated with IS in univariate and multivariate analysis.

Univariable regression analysis revealed RFM and BFP as predictors of IS among obesity indices. However, after adjustment, only BFP remained statistically significant. Multivariable regression model of IS identified diabetes mellitus, systolic blood pressure, white blood cell count, hemoglobin and BFP as independent predictors (Table 2, 3). The association of BFP with peak CK-MB, as a surrogate of IS, is also illustrated in Figure 1.

DISCUSSION

The main findings of the present study were: (1) Obesity and overweight were common in patients with NSTEMI. (2) Higher BFP is associated with smaller IS in NSTEMI patients.

Obesity is a global health burden and is regarded as a major preventable cause of death in the world. Obesity is common in patients with ACS with increasing prevalence both in NSTEMI and STEMI patients [15]. This is reflected also by the results of the present study in which 36.1% of the patients were obese and nearly half of the patients (46.5%) were overweight.

Adipose tissue acts as an endocrine organ and performs important tasks for regulating body metabolism and homeostasis. Obesity causes aberrant remodeling and thus dysfunctional adipose tissue, which in turn results in the dysregulation of various molecules secreted by adipocytes (lipids, inflammatory cytokines, and hormones like leptin, adiponectin, resistin that regulate food intake, insulin sensitivity, and immune responses) [16]. These obesity-related alterations have deleterious effects on the cardiovascular system. Obesity predisposes to dyslipidemia, hypertension, metabolic syndrome, type 2 diabetes mellitus (via insulin resistance), heart failure (via abnormal left ventricular geometry, left ventricular systolic and diastolic dysfunction), and CAD (via higher prevalence of risk factors, increased systemic inflammation, prothrombotic state and endothelial dysfunction) [17, 18].

Despite that, a phenomenon called "obesity paradox" is observed in studies suggesting that obese patients have better outcomes than leaner patients with the same cardiovascular diseases such as hypertension, atrial fibrillation, CAD, and heart failure. Obesity is a risk factor for CAD, but once CAD develops, obesity is associated with a better prognosis [19]. Obesity paradox has been described in NSTEMI [5]. Although not fully understood, some explanations have been proposed. These can be summarized as younger presentation of obese people with ACS, lower prevalence of smoking, more effective employment of secondary prevention measures, greater metabolic reserves, attenuated response to the renin-angiotensin-aldosterone system, lower natriuretic peptides, being able to tolerate medications with survival benefits such as beta-blockers, angiotensin-converting enzyme inhibitors and statins due to higher incidence of hypertension and dyslipidemia, better LVEF, less severe CAD, cardioprotective effects of adiopose-tissue derived hormones and poor diagnostic performance of BMI to identify obesity [18, 20]. Obesity is defined generally according to BMI, but BMI can not discriminate between fat mass and lean mass. Moreover, as known, central adipose tissue contributes more to the development of chronic diseases and BMI does not consider the localization of fat mass [21, 22]. In consistent with these concerns and contrary to the findings of studies using BMI and suggesting an obesity paradox, Lee et al. [23] found that central obesity assessed with waist-hip ratio was associated with poor prognosis in NSTEMI. Although dual-energy X-ray absorptiometry or imaging techniques such as MRI or computed tomography provide more accurate BFP estimation, availability and cost are the limiting factors to use them in routine clinical practice. Therefore, in this study, we evaluated body fat with different formulas (BFP and RFM) based on simple anthropometric measurements and gender that can be done in every clinic and correlate with these sophisticated methods [3, 4].

IS, left ventricular remodeling and consequent left ventricular dysfunction are major determinants of outcome in patients after ACS. Heart failure is very common after myocardial infarction (diagnosed in nearly 20–30% of patients at 1 year after discharge for myocardial infarction) and the development of heart failure increases total and cardiovascular mortality risk three and four-fold, respectively [9]. Cardiac MRI using late gadolinium enhancement is currently regarded as the gold standard to quantify IS. A recent study in patients with revascularized NSTEMI reported that peak CK-MB was strongly associated with chronic scar size evaluated with cardiac MRI which lead us to use peak CK-MB, a cardiac biomarker routinely used in clinical practice, as a measure of IS in this study [12].

There are conflicting results about the effect of obesity on IS and whether smaller IS in obese patients might serve as an explanation to "obesity paradox" in patients with ACS. Most of these studies used BMI to define obesity and most of them examined patients with STEMI. To our knowledge, this is the first study to examine the effect of different indicators of obesity on IS in NSTEMI.

Most of the studies, including a patient-level analysis from six randomized trials, in STEMI patients, reported that BMI was not associated with IS assessed with different methods [24–26]. Contrary to these findings, it

was reported that BMI ≥25 kg/m^2 was independently associated with smaller IS assessed with cardiac MRI in a study with 193 STEMI patients [27]. In an animal study, it was shown that the effect of obesity on IS is gender-dependent, and obesity increased IS in males but not in females [28]. Studies conducted in patients with ACS also revealed different conclusions. In a study with 40 men with ACS, waist circumference was reported to be associated with greater myocardial necrosis size evaluated with total creatine kinase and CK-MB [29]. Pingitore et al. [30] evaluated the effect of obesity (BMI ≥30 kg/m^2) on IS assessed with cardiac MRI in patients with at least 3 months old first myocardial infarction (n=89, >83% STEMI, 15.7% obese). They found that IS was significantly smaller in obese than non-obese patients. A study of 102 ACS patients composed mostly of patients with NSTEMI (n=73) reported that obesity defined with BMI and waist circumference was associated with smaller IS evaluated with troponin and creatinine phosphokinase in STEMI patients while it was associated with greater IS in NSTEMI patients, contrary to our findings [31]. The present study included a greater number of patients with NSTEMI and parameters associated better with adiposity than BMI were utilized. We found that BFP, not BMI and RFM, was associated with smaller IS in obese patients that may help to explain obesity paradox in NSTEMI patients.

Although underlying mechanisms are not fully elucidated, there are some reports suggesting cardioprotective effects of obesity. Leptin, adiponectin and resistin (adipose tissue-derived hormones) may protect against ischemia/reperfusion injury [32–34]. Also endocannabinoids, that are up-regulated in obesity, were reported to reduce both the overflow of biomarkers of infarction and IS which might indicate that cannabinoids can protect the heart from ischemia-reperfusion injury [35]. High epicardial adipose tissue was found to be related to smaller IS in 193 patients with STEMI [36]. Epicardial adipose tissue in obese patients may secrete anti-inflammatory, anti-oxidant and vasodilatory products and may serve as source of free fatty acids to overcome the energy needs of myocardium [27]. Besides that, possible explanations for the reduced IS in obese patients found in our study may be a lower prevalence of smoking and less severe CAD in this group, consistent with the previous literature [6, 37]. These findings together with smaller IS in patients with higher BFP in the present study, may provide insight into obesity paradox in patients with NSTEMI.

We included all consecutive NSTEMI patients with anthropometric measurements, but our study had several limitations. First, it can not imply a causal relationship due to its observational nature. Second, BFP and IS were evaluated with formulas based on anthropometric measurements and CK-MB, respectively, instead of more accurate but sophisticated techniques that are not available in every clinic.

Conclusion

Obesity is common in NSTEMI patients as in the general population and higher BFP is associated with smaller IS in these patients. These findings support the obesity paradox in NSTEMI, but further, randomized controlled studies are required to enlighten this association, keeping in mind that obesity is associated with increased cardiovascular disease risk and should be treated with lifestyle modifications or other means necessary as it is regarded as a preventable cause of death worldwide.

Ethics Committee Approval: The Haydarpasa Numune Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 05.06.2023, number: HNEAH-KAEK 2023/KK/103).

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