

Original article

Association of Echocardiographic Parameters with Nitric Oxide Levels in Overweight Patients with Coronary Artery Disease after Coronary Artery Bypass Grafting

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Abstract: *Introduction* — Overweight and obesity are recognized as significant risk factors for adverse outcomes after coronary artery bypass grafting (CABG). One of the key mechanisms linking obesity to the progression of cardiovascular pathology is endothelial dysfunction, marked by reduced nitric oxide (NO) bioavailability.

Objective — To evaluate the association between epicardial fat thickness (EFT), NO levels, and echocardiographic parameters in overweight patients during cardiac rehabilitation after CABG.

Methods — We enrolled 155 post-CABG patients stratified by body mass index (BMI). Clinical and demographic characteristics, plasma NO levels, and echocardiographic parameters were assessed. Regression analysis was used to identify predictors.

Results — Overweight patients exhibited a statistically significant increase in EFT and a decrease in plasma NO levels compared to normal-weight patients ($p < 0.05$). EFT correlated positively with left ventricular myocardial mass and diastolic dysfunction. Regression analysis identified BMI as an independent predictor of adverse changes (odds ratio [OR]=1.917; 95% confidence interval [CI]: 1.204–3.052; $p = 0.006$). BMI remained an independent predictor of these changes and demonstrated diagnostic utility in predicting the risk of endothelial dysfunction, as indicated by NO levels.

Conclusion — In post-CABG patients, overweight is associated with increased EFT, reduced NO levels, and impaired diastolic function of the myocardium. These findings underscore obesity's role as a significant predictor of endothelial dysfunction and adverse cardiac remodeling postoperatively.

Keywords: coronary artery bypass grafting, obesity, epicardial fat, endothelial dysfunction, nitric oxide, echocardiography.

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Introduction

The metabolic syndrome – of which overweight and abdominal obesity are core components – is recognized as a major driver of cardiovascular disease (CVD) development and has been termed one of the defining noncommunicable “epidemics” of the 21st century [1, 2].

Overweight is an established predictor of adverse outcomes following CABG [3, 4].

Epicardial adipose tissue (EAT) is an important contributor to the development of atherosclerotic changes in the coronary arteries not only through its paracrine and vasoactive secretions but also via its anatomical proximity to the epicardial coronary arteries [5, 6].

Increased EFT is associated with biventricular hypertrophy, elevated myocardial workload, and left ventricular remodeling [7, 8]. Higher BMI values are associated with the development of atherosclerosis in grafts [9, 10].

Overweight patients following coronary interventions more frequently require prolonged hospitalization during rehabilitation and face an elevated risk of restenosis, associated with activation of inflammatory processes [11, 12]. Thus, obesity is regarded not only as a risk factor for the development of coronary artery disease (CAD) but also as a significant predictor of adverse outcomes after CABG.

Endothelial dysfunction is a key contributor to impaired vascular tone and atherosclerotic damage. Endothelial injury increases vascular permeability and activates the inflammatory cascade. The endothelium plays a central role in regulating vascular dilation and constriction, platelet adhesion, and smooth muscle cell proliferation [13]. Nitric oxide, the primary mediator of endothelium-dependent vasodilation, is central to this process. Reduced NO production or bioavailability is considered a sensitive biomarker of endothelial dysfunction and a predictor of atherosclerotic progression [14, 15].

Overweight patients who have undergone CABG represent a clinically relevant subgroup. Currently, data on the relationship between EFT, NO levels, and echocardiographic (ECHO) parameters in this population remain limited.

Objective: To investigate the relationship between echocardiographic parameters and NO levels in overweight patients during rehabilitation after CABG.

Material and Methods

Study Design

A prospective cohort study was conducted in accordance with Good Clinical Practice (GCP) standards and the principles of the Declaration of Helsinki from February 2021 to June 2022. The study protocol was approved by the Ethical Committee of Karaganda Medical University (Karaganda, Republic of Kazakhstan; Protocol No. 16, dated March 15, 2021).

Inclusion and Exclusion Criteria

Eligible participants met the following inclusion and exclusion criteria:

Inclusion criteria:

- Age 35-65 years
- Diagnosis of single-vessel coronary artery disease (CAD)
- Post-CABG status
- Provision of written informed consent

Exclusion criteria:

- Acute myocardial infarction
- Chronic heart failure (NYHA Class III-IV)
- Decompensated diabetes mellitus
- Class I-III obesity
- Acute cerebrovascular accident
- Severe comorbidities limiting participation

Group Formation

We enrolled 155 patients:

- Group 1 ($n=85$): Post-CABG, overweight (BMI: 28.0 ± 0.9 kg/m²; 48 men, 37 women);
- Group 2 ($n=70$): Post-CABG, normal weight (BMI: 23.3 ± 1.1 kg/m²; 39 men, 31 women);
- Control group ($n=30$): Healthy, age-matched volunteers without acute or chronic disease (BMI: 20.4 ± 0.7 kg/m²).

Rehabilitation Program

Cardiac rehabilitation was conducted in accordance with Orders No. 116 and No. 65 of the Ministry of Healthcare of the Republic of Kazakhstan and comprised three stages:

- Stage 1: 1 to 3 months post-CABG;
- Stage 2: 3 to 6 months;
- Stage 3: more than 6 months after surgery.

All patients received standard medication therapy (antiplatelet, cardioprotective, antianginal, antihypertensive, and hypolipidemic). In addition, exercise therapy, massage, breathing exercises, and physiotherapy were applied.

Research Methods

Nitric Oxide. The concentration of NO metabolites was quantified spectrophotometrically using the Griess reagent ($\lambda=370$ nm), with nitrite anion (NO₂⁻) levels measured as stable metabolites of NO. Prior to analysis, serum proteins were precipitated using a 10% zinc sulfate solution, and the samples were pelleted by centrifugation at 3000 rpm for 10 minutes. Optical density was measured against a standard curve generated from sodium nitrite (NaNO₂) solutions [16].

Table 1. Baseline Demographic and Clinical Characteristics of Patients by Group

Parameter	Group 1 (BMI+ CABG+) ($n=85$)	Group 2 (BMI- CABG+) ($n=70$)	<i>p</i> -value
Age, years (mean±SD)	57.4±6.3	56.1±7.1	0.34
Male, <i>n</i> (%)	48 (56.4%)	39 (55.7%)	0.97
Female, <i>n</i> (%)	37 (43.6%)	31 (44.3%)	0.95
BMI, kg/m ² (mean±SD)	28.0±0.9	23.3±1.1	<0.001*
Arterial Hypertension, <i>n</i> (%)	61 (71.8%)	48 (68.6%)	0.67
Type 2 Diabetes Mellitus, <i>n</i> (%)	29 (34.1%)	22 (31.4%)	0.78
Smokers, <i>n</i> (%)	33 (38.8%)	28 (40.0%)	0.88
ACE Inhibitors, <i>n</i> (%)	76 (89.4%)	64 (91.4%)	0.70
Beta-Blockers, <i>n</i> (%)	81 (95.3%)	67 (95.7%)	0.89
Statins, <i>n</i> (%)	80 (94.1%)	66 (94.3%)	0.95
Antiplatelet Agents, <i>n</i> (%)	85 (100%)	70 (100%)	1.00

* Statistically significant ($p < 0.05$). SD, Standard Deviation; BMI+, Overweight; BMI-, Normal Weight; CABG+, Post-CABG status.

Table 2. Comparative Analysis of Echocardiographic Parameters and Plasma NO Levels in the Study Groups and the Control Group (Median [Q25; Q75])

Parameter	Groups				
	Group 1 ($n=85$)	Group 2 ($n=70$)	<i>p</i> -value*	Control Group ($n=30$)	<i>p</i> -value**
LVEF, %	47.0 (42.5-53.0)	53.0 (50.7-58.0)		62.0 (58.0-64.0)	
EFT, mm	4.23 (3.65-4.63)	2.43 (1.98-3.06)	0.0001	1.47 (1.06-2.02)	0.0001
LVMMI, g/m ²	110.0 (103.5-119.5)	82.0 (71.7-93.0)		68.0 (63.0-74.0)	
Plasma NO, μmol/L	3.30 (2.35-3.96)	4.21 (3.02-4.67)		5.13 (4.32-6.96)	

Data are presented as median (interquartile range: Q25-Q75). LVEF, Left Ventricular Ejection Fraction; EFT, Epicardial Fat Thickness; LVMMI, Left Ventricular Myocardial Mass Index; NO, Nitric Oxide. *p*-value*: Comparison between Group 1 and Group 2. *p*-value**: Comparison between Group 1 and the Control Group (healthy individuals).

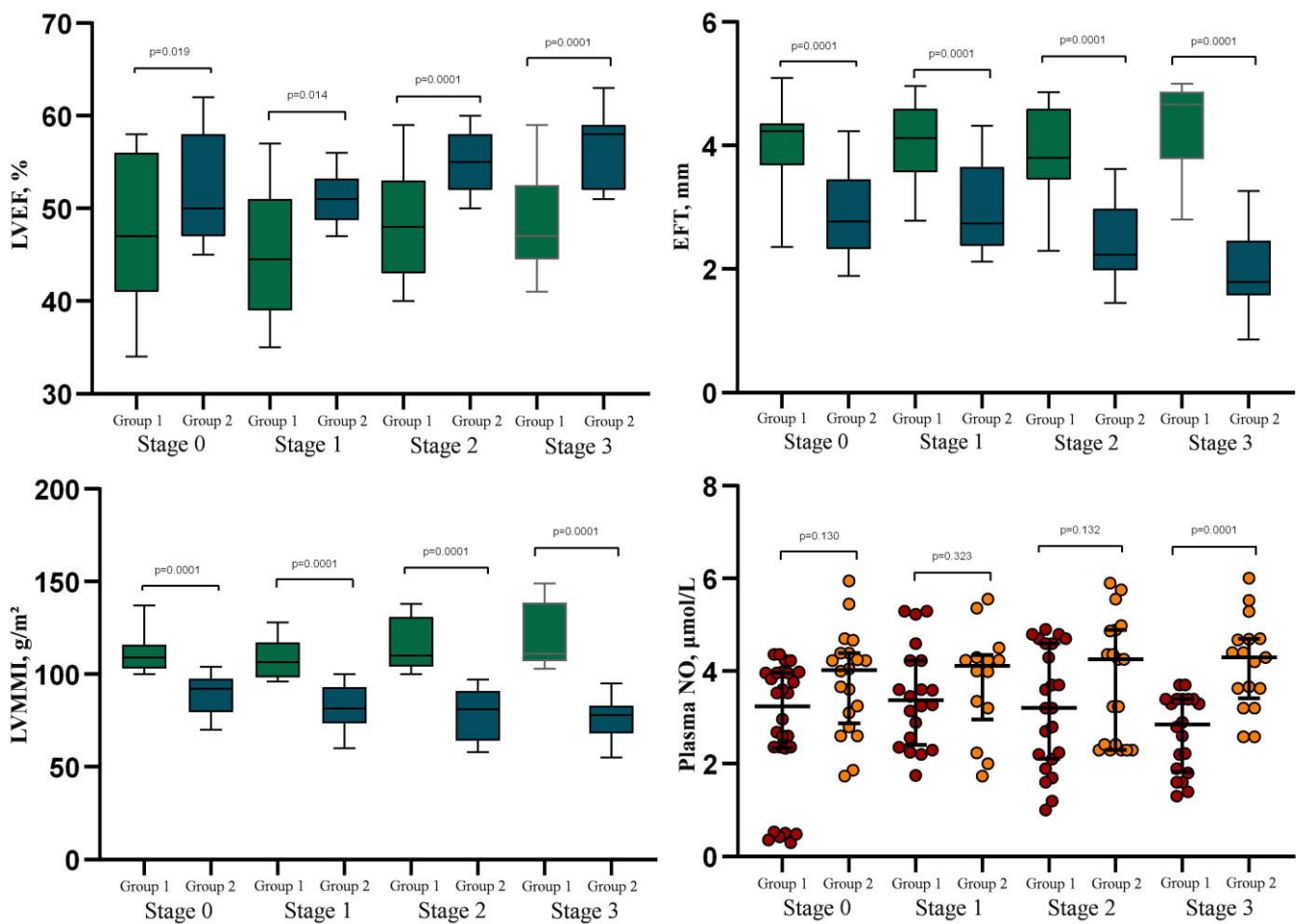


Figure 1. Comparative Analysis of Echocardiographic Parameters and NO Levels Across Rehabilitation Stages in Study Groups (Median [Q25–Q75]).

Echocardiography. All EFT measurements were performed by a single ultrasound specialist using a *Vivid 8* echocardiography system (GE Healthcare, USA) in B- and M-modes. Standardization was achieved using the parasternal long-axis view at end-diastole, with values averaged over three cardiac cycles. Intra-observer reproducibility was assessed in 20 randomly selected patients, with repeat measurements performed 7-10 days later; the intraclass correlation coefficient (ICC) was 0.92 (95% CI: 0.87-0.96), indicating high consistency. Calculated parameters included left ventricular ejection fraction (LVEF, by the Teichholz formula), left ventricular myocardial mass (by the ASE method), and EFT, measured on the free wall of the right ventricle from the parasternal long-axis view at end-diastole and averaged over three cardiac cycles.

Ethical Aspects

All participants provided voluntary informed consent.

Statistical Analysis

Analyses were conducted using SPSS version 27.0 and MedCalc version 22. Normality of data distribution was assessed using the Kolmogorov-Smirnov test. Non-normally distributed variables are reported as median and interquartile range (IQR). Between-group comparisons were performed using the Mann-Whitney *U* test and

Kruskal-Wallis *H* test, as appropriate. Within-group changes over time were evaluated using the Friedman test.

Receiver operating characteristic (ROC) analysis was employed to assess the diagnostic accuracy of BMI, with the area under the curve (AUC) and 95% confidence intervals (CI) reported.

Regression analysis

Multivariable logistic regression was performed to identify independent predictors of reduced NO levels and increased EFT, incorporating clinical and instrumental parameters as covariates. Statistical significance was defined as $p < 0.05$.

Results

Table 1 presents the baseline demographic and clinical characteristics of the study groups, along with pharmacotherapy received during the rehabilitation period.

Comparison of baseline characteristics between the two groups revealed no significant differences in demographic or clinical parameters. Mean age was 57.4 ± 6.3 years in the overweight group ($BMI \geq 25 \text{ kg/m}^2$) and 56.1 ± 7.1 years in the normal-weight group ($BMI 18.5-24.9 \text{ kg/m}^2$; $p=0.34$). The proportion of male participants was comparable (55.3% vs. 55.7% ; $p=0.96$). Prevalence of comorbidities – including arterial

hypertension (71.8% vs. 68.6%; $p=0.67$), type 2 diabetes mellitus (34.1% vs. 31.4%; $p=0.78$), and smoking (38.8% vs. 40.0%; $p=0.88$) – did not differ significantly between groups. Both groups received standard pharmacotherapy, including angiotensin-converting enzyme (ACE) inhibitors (89.4% vs. 91.4%; $p=0.70$), beta-blockers (95.3% vs. 95.7%; $p=0.89$), statins (94.1% vs. 94.3%; $p=0.95$), and antiplatelet agents (100% in both groups). The only parameter showing a statistically significant difference was BMI: 28.0 ± 0.9 kg/m² in the overweight group compared to 23.3 ± 1.1 kg/m² in the normal-weight group ($p < 0.001$).

In [Table 2](#), NO levels are presented in the study groups and the control group.

As shown in [Table 2](#), all parameters in Group 1 differed statistically significantly from those in Group 2 and the control group ($p < 0.05$). Compared with the control group, Group 1 exhibited a 24% reduction in left ventricular ejection fraction (LVEF), a 187% increase in epicardial fat thickness (EFT), a 62% increase in left ventricular myocardial mass index (LVMMI), and a 36% decrease in plasma NO levels. Compared with Group 2, Group 1 had a 12% lower LVEF, 22% lower plasma NO, 43% higher EFT, and 25% higher LVMMI.

Table 3. Correlation Analysis of NO and Echocardiographic Parameters Across Rehabilitation Stages in Study Groups

Echocardiographic Parameter	Statistic	NO			
		Stage 0	Stage 1	Stage 2	Stage 3
LVEF, %	Correlation	-0.492*	0.341	0.495*	0.908**
	<i>p</i> -value	0.020	0.141	0.016	0.000
EFT, mm	Correlation	-0.207	-0.035	-0.657**	-0.807**
	<i>p</i> -value	0.367	0.883	0.001	0.000
LVMMI, g/m ²	Correlation	0.297	-0.487*	-0.665**	-0.947**
	<i>p</i> -value	0.180	0.029	0.001	0.000

Table 4. Regression Model (Rehabilitation Stage 2)

Parameter	OR (95% CI)	<i>p</i> -value
BMI	1.797 (1.038-3.110)	0.036

OR, Odds Ratio; CI, Confidence Interval.

Table 5. Characteristics of the ROC Curves (Rehabilitation Stage 2)

Variable	Cut-off	Youden Index	AUC (95% CI)	Sensitivity, % (95% CI)	Specificity, % (95% CI)	<i>p</i> -value
BMI	>27.34	0.5152	0.734 (0.575-0.858)	66.67 (29.9-92.5)	84.85 (68.1-94.9)	0.057

AUC, Area Under the Curve; CI, Confidence Interval; Youden Index, Sensitivity + Specificity – 1.

Table 6. Regression Model (Rehabilitation Stage 3)

Parameter	OR (95% CI)	<i>p</i> -value
BMI	1.917 (1.204-3.052)	0.006

OR, Odds Ratio; CI, Confidence Interval.

Table 7. Characteristics of the ROC Curves (Rehabilitation Stage 3)

Variable	Cut-off	Youden Index	AUC (95% CI)	Sensitivity, % (95% CI)	Specificity, % (95% CI)	<i>p</i> -value
BMI	>28.08	0.8333	0.867 (0.714-0.956)	83.33 (51.6-97.9)	100 (86.3-100.0)	0.0001

AUC, Area Under the Curve; CI, Confidence Interval; Youden Index, Sensitivity + Specificity – 1.

Further comparison of echocardiographic parameters and plasma NO levels in Group 1 and Group 2 across rehabilitation stages is presented in [Figure 1](#).

According to our data, echocardiographic parameters in Group 1 were statistically significantly higher than those in Group 2 across all rehabilitation stages. Notably, in Group 2, all parameters demonstrated progressive improvement throughout the rehabilitation period: left ventricular ejection fraction (LVEF) showed a strong positive trend, while EFT and LVMMI decreased. In contrast, Group 1 exhibited a more complex pattern: although LVMMI and EFT initially decreased during early rehabilitation stages, both parameters demonstrated a statistically significant increase by the third rehabilitation stage. Concurrently, a statistically significant decline in LVEF was observed in Group 1 at the third stage.

A statistically significant difference in NO levels was observed between Group 1 and Group 2 only at the third rehabilitation stage.

To evaluate the relationship between NO levels and echocardiographic parameters across rehabilitation stages in overweight patients who had undergone reperfusion therapy, a correlation analysis was performed ([Table 3](#)).

Correlation analysis revealed both positive and negative associations between NO levels and echocardiographic parameters at Stage 0 and during the first and second rehabilitation stages. By the third rehabilitation stage, strong and consistent correlations emerged between all echocardiographic parameters and NO levels.

A regression model was constructed to assess the influence of BMI on echocardiographic parameters and NO levels.

Receiver operating characteristic (ROC) analysis was conducted to evaluate the diagnostic utility of BMI in predicting combined alterations in NO levels and echocardiographic parameters ([Tables 4-7](#)).

Summarizing the data, at Rehabilitation Stage 2, BMI was identified as an independent predictor of reduced NO levels and adverse echocardiographic changes (OR=1.797; 95% CI: 1.038-3.110; $p=0.036$).

[Figure 2](#) presents the ROC curve for Rehabilitation Stage 2 (AUC=0.734; 95% CI: 0.575-0.858). At Rehabilitation Stage 3 ([Figure 3](#)), the diagnostic value of BMI was higher (AUC=0.867; 95% CI: 0.714-0.956; $p < 0.001$). The optimal BMI threshold was 28.08 kg/m², at which sensitivity reached 83.3% and specificity was 100%.

Thus, the analysis demonstrated that overweight in patients after CABG is associated with reduced NO levels, increased EFT, elevated left ventricular myocardial mass, and decreased cardiac contractile function.

BMI serves as an independent predictor of these changes and demonstrates diagnostic utility in predicting the risk of endothelial dysfunction, as reflected by NO levels.

Discussion

Our study demonstrates that overweight patients after CABG exhibit significantly increased EFT, reflecting impaired lipid metabolism in the epicardium. EFT was higher in the overweight cohort compared to normal-weight controls ($p < 0.05$), reinforcing

that obesity promotes not only visceral adiposity but also pathological fat deposition in the epicardium.

The American Heart Association recognizes obesity as an independent risk factor for coronary artery disease (CAD) [17, 18]. Large-scale studies further corroborate the association between excess body weight and adverse clinical outcomes after CABG [3, 4].

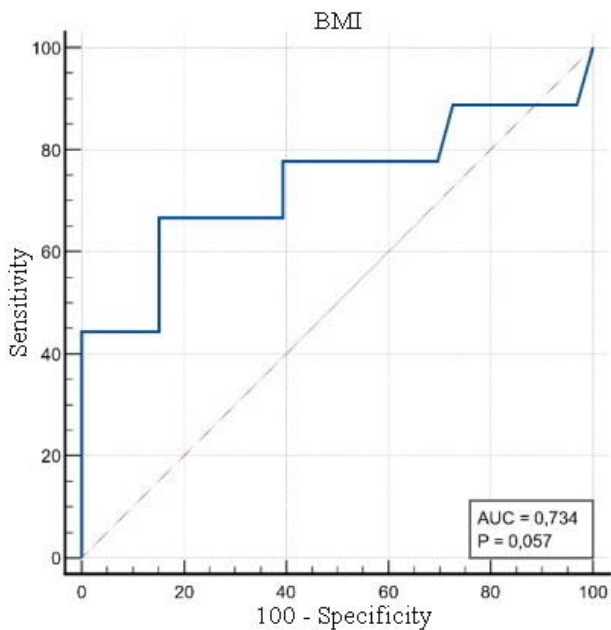


Figure 2. ROC Curve for Predicting the Development of Combined Adverse Changes in NO Levels and Echocardiographic Parameters Among Study Patients at Rehabilitation Stage 2. AUC and p-values for statistically significant predictors are indicated on the graph.

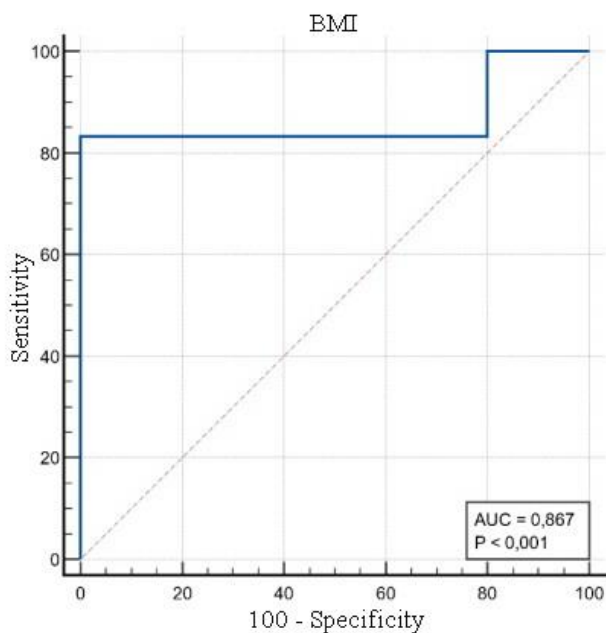


Figure 3. ROC Curve for Predicting the Development of Combined Adverse Changes in NO Levels and Echocardiographic Parameters Among Study Patients at Rehabilitation Stage 3. AUC and p-values for statistically significant predictors are indicated on the graph.

Notably, our study identified a significant further increase in EFT and left ventricular myocardial mass (LVMM) at the late (third) rehabilitation stage in patients with excess body weight. This likely reflects sustained metabolic and inflammatory dysregulation due to inadequate lifestyle modification, persistent dyslipidemia, and reduced physical activity after hospital discharge. Furthermore, decreased NO bioavailability and persistent endothelial dysfunction may contribute to myocardial remodeling and the paracrine activation of epicardial adipose tissue. These findings underscore the need for an individualized approach to patient management during late-stage cardiac rehabilitation, one that incorporates an understanding of the metabolic mechanisms underlying cardiac remodeling.

Analysis of the Society of Thoracic Surgeons (STS) database indicates that severe obesity in women after CABG does not elevate in-hospital mortality but is associated with a higher incidence of wound complications and rehospitalizations [19]. In contrast, several authors have identified obesity as an independent predictor of increased long-term mortality following myocardial revascularization [20-22].

According to the literature, EFT < 7 mm is associated with subclinical atherosclerosis, whereas EFT > 7 mm is reliably associated with an increased risk of coronary artery disease (CAD) [7, 5]. Corradi *et al.* demonstrated that EFT positively correlates with left ventricular myocardial mass, atrial size, and diastolic dysfunction; this parameter is significantly elevated in the setting of myocardial hypertrophy [23]. Similar findings have been reported by other investigators, who noted that a pronounced increase in EFT contributes to ventricular remodeling and hypertrophy of the left heart chambers [8].

In our study, a significant correlation between EFT and NO levels was observed, suggesting the development of an inflammatory process linked to endothelial dysfunction. In patients with atherosclerosis, reduced NO bioavailability – resulting from endothelial dysfunction – impairs coronary blood flow [15, 24]. This mechanism is largely attributable to tetrahydrobiopterin (BH₄) deficiency, a critical cofactor for endothelial nitric oxide synthase (eNOS), alongside elevated levels of asymmetric dimethylarginine (ADMA), an endogenous inhibitor of NO synthesis whose concentrations increase under the influence of oxidized low-density lipoproteins [14, 24].

Notably, during the late (third) rehabilitation stage, we observed very high correlation coefficients between NO levels and echocardiographic parameters ($r=0.908$ for LVEF and $r=-0.947$ for LVMMI). These findings indicate a strong pathophysiological link between endothelial function and myocardial remodeling: diminished NO bioavailability is accompanied by increased myocardial mass and reduced contractility. This suggests that, at this stage, endothelial dysfunction may be the primary driver of remodeling severity in overweight patients.

Our results illustrate that dynamic changes in echocardiographic parameters and NO levels in overweight patients reflect a complex interplay of metabolic and endothelial mechanisms. The renewed increase in EFT and LVMMI during Rehabilitation Stage 3, coinciding with declining NO levels, points to persistent endothelial dysfunction and a chronic subclinical inflammatory milieu. These observations align with data from Viridis *et al.* (2016) [25] and Oikonomou and Antoniadis (2019) [26], underscoring the role of oxidative stress and adipokine imbalance in advanced stages of myocardial remodeling in obese

patients. Consequently, our study reinforces the necessity for long-term metabolic and anti-inflammatory management within cardiac rehabilitation programs following CABG.

The identified BMI threshold ($>28.08 \text{ kg/m}^2$) has practical significance for risk stratification in post-CABG patient management. A BMI exceeding this threshold may serve as a marker of an increased likelihood of reduced NO bioavailability and the development of endothelial dysfunction, justifying more aggressive lifestyle modification and intensification of pharmacological therapy. Specifically, such patients should be advised to adhere to strict body weight control, participate in expanded physical rehabilitation programs, and receive intensified hypolipidemic and antioxidant therapy – approaches consistent with contemporary guidelines from the American Heart Association (AHA, 2021) and the European Association for Cardiovascular Prevention and Rehabilitation (EACPR, 2023) for managing patients with obesity and coronary artery disease.

Conclusions

Overweight in patients after CABG is associated with increased EFT, reduced NO levels, and impaired myocardial diastolic function. The obtained data confirm the role of obesity as a significant predictor of endothelial dysfunction and adverse cardiac remodeling in the postoperative period.

Limitations

This study has several limitations that should be considered when interpreting the results. First, it was a single-center study with a limited sample size, which may restrict the generalizability of the findings to broader populations.

Second, the relatively small sample size may have limited statistical power to detect weaker or modest associations.

Third, the study had an observational design, which does not allow for the full establishment of causal relationships between overweight, EFT, NO levels, and echocardiographic parameters.

Furthermore, some potential confounders – including physical activity levels, dietary patterns, and the degree of medication adherence – were not formally assessed and may have influenced the observed associations.

Moreover, the limited follow-up duration precludes assessment of long-term clinical outcomes in this patient cohort.

Ethical Approval and Consent to Participate

The study was conducted in accordance with the ethical standards of the institutional research committee and the 1964 Declaration of Helsinki and its subsequent amendments. Written informed consent was obtained from all participants prior to enrollment. The study protocol was approved by the Ethics Committee of Karaganda Medical University (Karaganda, Republic of Kazakhstan; Protocol No. 16, March 15, 2021).

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Conflict of Interest

The authors declare no conflicts of interest.

Disclosure of AI Use

The authors confirm that no artificial intelligence tools or technologies were used in the preparation of this manuscript.

References

- Alberti KG, Zimmet P, Shaw J; IDF Epidemiology Task Force Consensus Group. The metabolic syndrome – A new worldwide definition. *Lancet* 2005; 366(9491): 1059-1062. [http://doi.org/10.1016/S0140-6736\(05\)67402-8](http://doi.org/10.1016/S0140-6736(05)67402-8).
- Neeland IJ, Ross R, Després JP, Matsuzawa Y, Yamashita S, Shai I, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol* 2019; 7(9): 715-725. [http://doi.org/10.1016/S2213-8587\(19\)30084-1](http://doi.org/10.1016/S2213-8587(19)30084-1).
- Elbaz-Greener G, Rozen G, Carasso S, Kusniec F, Yarkoni M, Marai I, et al. The relationship between body mass index and in-hospital mortality in patients following coronary artery bypass grafting surgery. *Front Cardiovasc Med* 2021; 8: 754934. <https://doi.org/10.3389/fcvm.2021.754934>.
- Yakhyaeva KB, Keren MA, Zavalikhina TV, Volkovskaya IV, Sigaev IYu. Prognostic impact of obesity in patients with coronary heart disease who underwent coronary artery bypass grafting. *Creative Cardiology* 2025; 19(3): 362-371. <https://dx.doi.org/10.24022/1997-3187-2025-19-3-362-371>.
- Mahabadi AA, Balcer B, Dykun I, Forsting M, Schlosser T, Heusch G, et al. Cardiac computed tomography-derived epicardial fat volume and attenuation independently distinguish patients with and without myocardial infarction. *PLoS One* 2017; 12(8): e0183514. <http://doi.org/10.1371/journal.pone.0183514>.
- Guglielmo M, Lin A, Dey D, Baggiano A, Fusini L, Muscogiuri G, et al. Epicardial fat and coronary artery disease: Role of cardiac imaging. *Atherosclerosis* 2021; 321: 30-38. <https://doi.org/10.1016/j.atherosclerosis.2021.02.008>.
- Iacobellis G, Bianco AC. Epicardial adipose tissue: emerging physiological, pathophysiological and clinical features. *Trends Endocrinol Metab* 2011; 22(11): 450-457. <http://doi.org/10.1016/j.tem.2011.07.003>.
- Parisi V, Cabaro S, D'Esposito V, Petraglia L, Conte M, Campana P, et al. Epicardial adipose tissue and IL-13 response to myocardial injury drives left ventricular remodeling after ST elevation myocardial infarction. *Front Physiol* 2020; 11: 575181. <https://doi.org/10.3389/fphys.2020.575181>.
- An KR, Sandner S, Redfors B, Alexander JH, Alzghari T, Caldonazo T, et al. Association between overweight and obesity with coronary artery bypass graft failure: an individual patient data analysis of clinical trials. *Eur J Cardiothorac Surg* 2024; 65(6): ezae221. <https://doi.org/10.1093/ejcts/ezae221>.
- Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: A scientific statement from the American Heart Association. *Circulation* 2021; 143(21): e984-e1010. <https://doi.org/10.1161/cir.0000000000000973>.
- Aronov D, Bubnova M, Iosseliani D, Orekhov A. Clinical efficacy of a medical centre- and home-based cardiac rehabilitation program for patients with coronary heart disease after coronary bypass graft surgery. *Arch Med Res* 2019; 50(3): 122-132. <https://doi.org/10.1016/j.arcmed.2019.07.007>.
- Madiyeva MI, Aripov MA, Goncharov AY, Zholdasbekova RY. Outcomes of myocardial revascularization in patients with obesity and multivessel coronary artery disease. *Egypt Heart J* 2024; 76(1): 114. <https://doi.org/10.1186/s43044-024-00548-5>.
- Esper RJ, Nordaby RA, Vilarino JO, Paragano A, Cacharrón JL, Machado RA. Endothelial dysfunction: a comprehensive appraisal. *Cardiovasc Diabetol* 2006; 5: 4. <http://doi.org/10.1186/1475-2840-5-4>.
- Förstermann U, Sessa WC. Nitric oxide synthases: regulation and function. *Eur Heart J* 2012; 33(7): 829-837, 837a-837d. <http://doi.org/10.1093/eurhearti/ehr304>.

15. Matsuzawa Y, Lerman A. Endothelial dysfunction and coronary artery disease: assessment, prognosis and treatment. *Coron Artery Dis* 2014; 25(8): 713–724. <https://doi.org/10.1097/mca.000000000000178>.
16. Metelskaya VA, Gumanova NG. Screening as a method for determining the se-rum level of nitric oxide metabolites. *Klinicheskaya Laboratornaya Diagnostika* 2005; (6): 15. <https://www.elibrary.ru/ojclrv>.
17. Lavie CJ, Milani RV, Ventura HO. Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss. *J Am Coll Cardiol* 2009; 53(21): 1925-1932. <http://doi.org/10.1016/j.jacc.2008.12.068>.
18. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation* 2021; 143(21): e984-e1010. <http://doi.org/10.1161/CIR.0000000000000973>.
19. Terada T, Johnson JA, Norris C, Padwal R, Qiu W, Sharma AM, et al. Severe obesity is associated with increased risk of early complications and extended length of stay following coronary artery bypass grafting surgery. *J Am Heart Assoc* 2016; 5(6): e003282. <https://doi.org/10.1161/jaha.116.003282>.
20. Bis J, Kania-Olejnik P, Padaż K, Malinowski M, Deja MA. Impact of body mass index on long-term mortality after coronary artery bypass grafting: a retrospective cohort study. *Ann Med Surg (Lond)* 2025; 87(7): 4066-4072. <https://doi.org/10.1097/ms9.0000000000003432>.
21. Benedetto U, Danese C, Codispoti M. Obesity paradox in coronary artery bypass grafting: myth or reality? *J Thorac Cardiovasc Surg* 2014; 147(5): 1517-1523. <https://doi.org/10.1016/j.jtcvs.2013.05.028>.
22. Ri M, Aikou S, Seto Y. Obesity as a surgical risk factor. *Ann Gastroenterol Surg* 2017; 2(1): 13-21. <https://doi.org/10.1002/ags3.12049>.
23. Corradi D, Maestri R, Callegari S, Pastori P, Goldoni M, Luong TV, et al. The ventricular epicardial fat is related to the myocardial mass in normal, ischemic and hypertrophic hearts. *Cardiovascular Pathology* 2004; 13(6): 313-316. <https://doi.org/10.1016/j.carpath.2004.08.005>.
24. Willeit P, Freitag DF, Laukkanen JA, Chowdhury S, Gobin R, Mayr M, et al. Asymmetric dimethylarginine and cardiovascular risk: systematic review and meta-analysis of 22 prospective studies. *J Am Heart Assoc* 2015; 4(6): e001833. <https://doi.org/10.1161/jaha.115.001833>.
25. Virdis A. Endothelial dysfunction in obesity: Role of Inflammation. *High Blood Press Cardiovasc Prev* 2016; 23(2): 83-85. <https://doi.org/10.1007/s40292-016-0133-8>.
26. Oikonomou EK, Antoniades C. The role of adipose tissue in cardiovascular health and disease. *Nat Rev Cardiol* 2019; 16(2): 83-99. <http://doi.org/10.1038/s41569-018-0097-6>.

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