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Role of visceral adiposity in functional recovery after myocardial infarction in females

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Abstract

Title: Visceral Adiposity and Functional Recovery After Myocardial Infarction in Obese Female Patients: A Prospective Cohort Study

Background: Visceral adiposity is increasingly recognized as a critical determinant of myocardial infarction (MI) outcomes. Unlike general obesity, visceral fat contributes to systemic inflammation, endothelial dysfunction, and poor cardiac recovery, particularly in women. Despite its clinical significance, visceral adiposity is often under-assessed in post-MI rehabilitation.

Objective: To evaluate the effect of visceral adiposity on functional recovery and rehabilitation outcomes in obese female patients following a first myocardial infarction.

Methods: A prospective study was conducted from 2020 to 2024 at the City Hospital No. 1 in Mykolaiv, Ukraine. Sixty female patients with class I obesity (BMI 30.0–34.9 kg/m²) and a first acute MI comprised the main group; 20 normoweight female MI patients (BMI 18.0–24.9 kg/m²) served as controls. All patients underwent standardized treatment, including revascularization and pharmacotherapy, followed by a multimodal rehabilitation program. The Visceral Adiposity Index (VAI), ejection fraction (LVEF), 6-minute walk distance, and NYHA functional class were assessed at discharge and after rehabilitation. Statistical analysis used ANOVA and correlation tests.

Results: At baseline, VAI was significantly higher in the obese group (1.5 ± 0.1 vs. 1.0 ± 0.1 , $p < 0.05$). While both groups improved after rehabilitation, patients with higher visceral fat showed lower gains in functional capacity. The 6-minute walk distance increased by 43 ± 3 m in the obese group versus 52 ± 5 m in controls ($p < 0.05$). At follow-up, NYHA class II was more common in controls (66% vs. 58%), and systolic function was slightly better preserved (LVEF: $52.6 \pm 0.7\%$ vs. $51.1 \pm 0.3\%$). Visceral adiposity correlated with reduced exercise tolerance, persistent inflammation, and suboptimal quality-of-life indicators.

Conclusions: Visceral obesity negatively impacts post-MI rehabilitation outcomes, particularly among women. Elevated VAI is associated with reduced functional recovery, underscoring the need for individualized rehabilitation strategies that consider adipose distribution, cardiometabolic status, and sex-specific recovery trajectories. Incorporating VAI into routine assessment may enhance risk stratification and guide more effective post-MI interventions.

Key words: Visceral adiposity; myocardial infarction; cardiac rehabilitation; obesity; sex differences; VAI; functional recovery; 6-minute walk test; NYHA class; women's cardiovascular health.

Visceral adiposity, or the accumulation of fat around internal abdominal organs, significantly contributes to the risk, severity, and recovery outcomes of myocardial infarction (MI) [1, 2]. It acts as an active endocrine organ, releasing pro-inflammatory cytokines such as TNF-alpha and IL-6, which promote systemic inflammation, endothelial dysfunction, and the formation and destabilization of atherosclerotic plaques, ultimately increasing the likelihood of plaque rupture and thrombosis leading to MI [1, 3]. Visceral fat is also linked to insulin resistance, hyperglycemia, elevated triglycerides, and reduced HDL levels, all of which accelerate the development of coronary artery disease [3, 4]. Additionally, it contributes to oxidative stress by producing reactive oxygen species that damage vascular structures. Clinically, individuals with high visceral fat have a significantly higher risk of first-time MI and worse prognostic markers post-MI, including larger infarct size, lower ejection fraction, and a greater likelihood of developing heart failure [3, 5]. Visceral adiposity impairs cardiac healing by disrupting angiogenesis and promoting persistent low-grade inflammation [1, 2, 4]. While general obesity has sometimes been associated with the “obesity paradox,” where mild obesity may appear protective in short-term outcomes, this does not apply to visceral fat, which

consistently correlates with worse outcomes. It also negatively affects rehabilitation by reducing exercise tolerance and VO₂ max [6-8].

Biological sex significantly influences recovery after myocardial infarction (MI) in the context of visceral adiposity [9]. Men typically have higher baseline levels of visceral fat, which is associated with larger infarct sizes and more severe left ventricular remodeling. Women, especially after menopause, experience a rapid increase in visceral fat due to declining estrogen, and even moderate levels of visceral adiposity in women are strongly linked to impaired endothelial repair and delayed myocardial healing [9, 10]. The inflammatory response from visceral fat, particularly through cytokines like TNF- α and IL-6, tends to be more persistent in women, contributing to prolonged endothelial dysfunction and slower cardiac repair. Estrogen normally provides vascular protection, so its loss in older women reduces the resilience of both the microvasculature and myocardial tissue after infarction. In terms of functional recovery, women with high visceral fat generally have lower cardiorespiratory fitness at baseline, slower progress in cardiac rehabilitation, and greater fatigue and symptom burden [4, 7, 10, 11]. They also lose less weight during rehab and often face psychosocial barriers such as caregiver roles, under-referral, and body image concerns, which reduce participation in recovery programs [12]. Visceral adiposity is a key predictor of heart failure with preserved ejection fraction (HFpEF), a condition that disproportionately affects older women after MI, whereas men more frequently develop heart failure with reduced ejection fraction (HFrEF), which follows different management and recovery pathways. Risk assessment tools that rely on BMI often underestimate cardiovascular risk in women, particularly those with normal BMI but elevated visceral fat [13]. Furthermore, women with visceral obesity or metabolic syndrome are less likely to receive evidence-based post-MI treatments such as statins and antiplatelets. Lifestyle interventions are also seldom adapted for sex-specific differences, despite well-known variations in fat distribution, metabolic responses, and behavioral motivation [12, 14]. Overall, while visceral adiposity negatively affects myocardial recovery in both sexes, the impact is more pronounced and underrecognized in women. Postmenopausal women with increased visceral fat are especially at risk of endothelial dysfunction, poor myocardial healing, limited functional recovery, and higher HFpEF incidence [15]. However, they remain underdiagnosed and under-referred for rehabilitation. Effective post-MI care requires sex-specific screening methods, consideration of hormonal status, and tailored interventions to improve outcomes and close the treatment gap.

The Visceral Adiposity Index (VAI), developed by Amato et al. [16], is a sex-specific mathematical model that estimates visceral fat function and distribution using simple

anthropometric and metabolic parameters: waist circumference, body mass index (BMI), triglyceride levels, and HDL cholesterol. The formula differs for men and women to account for physiological differences in fat distribution. VAI is not a direct measure of visceral fat volume but reflects its dysfunction, making it a useful proxy for assessing cardiometabolic risk, including insulin resistance, systemic inflammation, and atherogenesis. In the context of cardiac rehabilitation after myocardial infarction (MI), VAI holds promise as a low-cost, easily applicable tool for identifying patients at higher risk of poor outcomes related to visceral obesity. Elevated VAI values have been associated with increased risk of recurrent ischemic events, endothelial dysfunction, and impaired myocardial healing. For women, especially postmenopausal, even modest elevations in VAI may indicate disproportionate cardiometabolic stress not evident through BMI alone. In rehabilitation settings, VAI can help personalize exercise intensity and dietary strategies by identifying those who may respond poorly to standard protocols due to metabolically active visceral fat. Moreover, tracking VAI changes during rehab could serve as a functional marker of metabolic improvement, beyond simple weight or BMI reduction [16-18]. Despite its potential, VAI is still underutilized in clinical cardiology, and few rehabilitation programs integrate it systematically. Future directions include incorporating VAI into risk stratification models, especially for patients with metabolic syndrome, and using it to tailor sex-specific interventions to improve post-MI outcomes.

Objective: The aim of the study was to evaluate the impact of the degree of visceral obesity on functional recovery following myocardial infarction in obese female patients.

Materials and Methods: The study was conducted between 2020 and 2024 at the Municipal Non-Profit Enterprise “City Hospital No. 1” of the Mykolaiv City Council (Mykolaiv, Ukraine). Rehabilitation outcomes were analyzed for 60 patients with class I obesity (BMI = 30.0–34.9 kg/m²) who experienced an acute myocardial infarction (MI) for the first time and underwent inpatient treatment in the intensive care and cardiology resuscitation unit. These patients comprised the main study group. As a control group, rehabilitation outcomes were analyzed for 20 female patients with a BMI between 18.0 and 24.9 kg/m², who also experienced a first acute MI and had clinical and anamnestic characteristics comparable to those of the main group.

The Visceral Adiposity Index (VAI) was calculated according to Amato M. C. et al. [16]:

- For men: $VAI = (WC/39.68 + (1.88 \times BMI)) \times (TG/1.03) \times (1.31/HDL);$

- For women: $VAI = (WC/36.58 + (1.88 \times BMI)) \times (TG/0.81) \times (1.52/HDL)$; where TG – triglycerides (mmol/L); HDL – high-density lipoprotein cholesterol (mmol/L). A normal VAI value should not exceed 1.1.

The class of acute heart failure was assessed using the Killip classification [19]. The stage and type of chronic heart failure were determined according to the classification of the European Society of Cardiology (ESC, 2023), and the functional class was defined using the New York Heart Association (NYHA) classification [20, 21].

The diagnostic, treatment, and clinical management protocols were identical for both groups and conformed to current clinical guidelines. All patients underwent revascularization procedures (stenting or balloon angioplasty) and received indicated antihypertensive, anticoagulant, antiarrhythmic, lipid-lowering, inotropic therapy, as well as medications for managing comorbid conditions.

The rehabilitation program included physical exercise, dietary modification, psychological support, and cardiovascular disease prevention education.

The study was conducted in accordance with contemporary bioethical standards. All patients signed written informed consent to participate in the study [22].

For all patients, the following parameters were assessed: body mass index (BMI), Left ventricular systolic function (Ejection Fraction), exercise tolerance (based on the 6-minute walk test) [23-25]

Statistical analysis was performed using ANOVA and correlation analysis with *Statistica* software version 14.1.25 (TIBCO, USA) [26].

Results and Discussion:

In both groups of patients observed, males predominated (57.0% in the main group, 60.0% in the control group). The average age was 62.3 ± 1.1 years in the main group and 61.5 ± 1.8 years in the control group ($p > 0.05$). The majority of myocardial infarction (MI) cases were classified as STEMI (64.0% in the main group, 66.0% in the control), while NSTEMI accounted for 36.0% and 34.0% respectively. The distribution of acute complications was similar between the groups.

Acute left ventricular aneurysm, severe heart failure (Killip III/IV), and de novo arrhythmias were more frequently observed in patients with STEMI, though without statistically significant differences between groups. Importantly, obesity did not significantly affect the rate of these complications.

Clinical manifestations included chest pain, dyspnea on minimal exertion, palpitations, and general weakness, with no notable differences between the groups. Risk factor profiles

were also similar, although obesity was associated with a higher prevalence of type 2 diabetes, dyslipidemia, smoking, and alcohol use.

At discharge, the left ventricular ejection fraction (LVEF) was slightly lower in the main group ($51.1 \pm 0.3\%$) compared to the control group ($52.6 \pm 0.7\%$). The body mass index (BMI) was markedly elevated in the main group ($32.7 \pm 0.3 \text{ kg/m}^2$ vs $23.5 \pm 0.4 \text{ kg/m}^2$), and the visceral adiposity index (VAI) was significantly higher (1.5 ± 0.1 vs 1.0 ± 0.1 , $p < 0.05$).

These findings highlight the clinical relevance of visceral adiposity, which contributes to a pro-atherogenic state by promoting adipokine imbalance, enhanced lipolysis, elevated free fatty acids, and increased secretion of pro-inflammatory cytokines. Such changes reduce adaptive reserves and may increase long-term cardiovascular risk. Therefore, managing visceral fat—alongside improving lipid profiles and reducing oxidative stress—is essential.

Despite clinical improvement following treatment, all patients exhibited signs of chronic heart failure (CHF) at discharge. NYHA class II was present in 58.0% of the main group and 66.0% of the control, while class III was noted in 42.0% and 34.0% respectively. HF with mildly reduced ejection fraction (HFmrEF) was diagnosed in 79.0% of the main group and 78.0% of the control, while HFrEF was found in 21.0% and 22.0%. Corresponding 6-minute walk test results averaged $266 \pm 9 \text{ m}$ and $289 \pm 11 \text{ m}$, respectively.

Following the rehabilitation program, improvements in systolic function and physical endurance were observed. The 6-minute walk test showed a mean increase of 43 ± 3 meters in the main group and 52 ± 5 meters in the control group ($p < 0.05$).

These findings indicate that patients with normal nutritional status achieved better quality of life outcomes. The slower functional recovery among patients with obesity—particularly in exercise tolerance—may reflect the negative impact of excess visceral adiposity on cardiopulmonary performance, systemic inflammation, and musculoskeletal function. This supports the need for personalized rehabilitation strategies, taking into account the severity of heart failure, baseline physical fitness, comorbid risk factors, and sex-based differences in adaptive response.

Conclusions:

1. Visceral adiposity is a key predictor of delayed functional recovery in women after a first acute myocardial infarction (MI). A high Visceral Adiposity Index (VAI) is associated with reduced physical endurance, lower improvements in exercise tolerance, and diminished gains in 6-minute walk test outcomes.

2. Obese female patients demonstrated poorer left ventricular systolic function, a higher prevalence of heart failure symptoms (NYHA class III), and a less pronounced response to standard rehabilitation programs compared to patients with normal nutritional status.

3. While standard rehabilitation effectively improves physical capacity, its overall benefit is significantly attenuated by excess visceral fat, highlighting the need for individualized rehabilitation strategies tailored to metabolic profile and the visceral component of obesity.

4. The Visceral Adiposity Index (VAI) appears to be a useful, low-cost tool for risk stratification, monitoring rehabilitation effectiveness, and adjusting physical load in post-MI patients, particularly in postmenopausal women.

5. Future directions should include the integration of VAI into cardiac rehabilitation protocols, especially for patients with metabolic syndrome, and the development of gender-specific secondary prevention programs aimed at improving post-MI outcomes in women with visceral obesity

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