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**Original Research Article**

# Impact of obesity on clinical and angiographic characteristics in patients with ST-elevation myocardial infarction

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## ABSTRACT

**Background:** Obesity is a well-established cardiovascular risk factor and is strongly associated with metabolic dysregulation, systemic inflammation, and accelerated atherosclerosis. However, its influence on clinical presentation, laboratory parameters, and coronary angiographic characteristics in patients with ST-elevation myocardial infarction (STEMI) remains controversial.
**Methods:** The study included 75 patients with STEMI who were admitted to the Grodno Regional Cardiological Center (Belarus) for treatment from July to October 2025. Patients were divided into two groups according to body mass index (BMI): group 1 (obese, BMI≥30 kg/m$^2$; n=37) and group 2 (non-obese, BMI≤29.9 kg/m$^2$; n=38). All patients underwent clinical, laboratory, and instrumental studies. Statistical analysis was performed using STATISTICA 12.0.
**Results:** Obese patients had a significantly higher prevalence of diabetes mellitus compared with non-obese patients (35.13% versus 7.89%, p=0.04). They also demonstrated a distinct metabolic profile characterized by higher triglycerides (p=0.001) and lower HDL cholesterol concentrations (p=0.037). No significant differences were observed in cardiac biomarkers, or renal function parameters. Angiographically, involvement of the left anterior descending artery (p=0.025) and left circumflex artery (p=0.03) was more frequent in obese patients. The rate of percutaneous coronary intervention and the number of implanted stents were comparable between groups.
**Conclusions:** Obesity in STEMI patients is associated with a higher burden of metabolic disturbances and more frequent multi-vessel coronary involvement, particularly affecting the left coronary system. These findings highlight the complex interaction between adiposity, metabolic dysregulation, and atherosclerotic severity in acute coronary syndromes.

**Keywords:** Atherosclerosis, Coronary angiography, Diabetes mellitus, Metabolic disorders, Obesity, ST-elevation myocardial infarction

## INTRODUCTION

Cardiovascular diseases remain the leading cause of mortality worldwide, with acute coronary syndromes (ACS) representing one of the most critical manifestations of coronary artery disease.[1,2] ST-elevation myocardial infarction (STEMI) is characterized by complete occlusion of a coronary artery due to acute atherothrombotic events and requires urgent reperfusion therapy to prevent irreversible myocardial damage.[1,3]

Obesity has reached epidemic proportions globally and represents a major modifiable cardiovascular risk factor.[4] Excess adiposity is associated with insulin resistance, dyslipidemia, systemic inflammation, endothelial dysfunction, and prothrombotic states. These mechanisms collectively accelerate atherosclerotic plaque formation and instability, increasing the risk of acute coronary events.[5,6]

Despite the clear association between obesity and cardiovascular disease, its impact on the severity and outcomes of STEMI remains debated.[3] Some studies describe the so-called "obesity paradox," suggesting improved short-term survival among overweight and obese patients after myocardial infarction.[3,7] However,

other investigations demonstrate that obesity contributes to more extensive coronary artery disease, higher inflammatory burden, and increased metabolic complications.[8]

In addition to metabolic alterations, obesity influences vascular remodelling, platelet activity, and neurohormonal activation, which may affect infarct size, reperfusion success, and myocardial recovery.[6] Understanding how obesity modifies clinical presentation and angiographic characteristics in STEMI patients is therefore essential for optimizing risk stratification and therapeutic strategies.[2]

Therefore, the aim of the study was to compare clinical and coronary angiographic characteristics in obese and non-obese patients with ST-elevation myocardial infarction.

## METHODS

The study included 75 patients with STEMI who were admitted to the Grodno Regional Cardiological Center (Belarus) for treatment from July 2025 to October 2025. In this study, patients were diagnosed with STEMI based on clinical symptoms, signs, ECG findings, and cardiac biomarker values, and they underwent coronary angiography with possible percutaneous coronary intervention at the same center in the x-ray operating room on the Philips Azurion 7 and GE Innova 3100 IQ angiographic units.

Obesity was defined as BMI≥30 kg/m$^2$. Exclusion criteria from the study were: age <18 years, underweight patients (BMI<18.5 kg/m$^2$), valvular pathology of the heart requiring surgical correction, infective endocarditis, myocarditis, hypertrophic cardiomyopathy, oncological diseases and severe concomitant extracardiac pathology.

The patients were further stratified according to their BMI values. Group 1 included 37 patients with obesity stage 1-3 (BMI≥30.0 kg/m$^2$), group 2 included 38 patients with BMI≤29.9 kg/m$^2$.

Statistical analysis was performed using the STATISTICA 12.0 software with a preliminary check for normal distribution using a distribution histogram. Quantitative data, the distribution of which was not normal, were given as a median, 25% and 75% quartiles. Since most of the quantitative characteristics did not obey the normal distribution law, non-parametric methods were used for comparison. The Mann-Whitney test was used to assess differences in quantitative traits between two independent groups. Statistical significance of differences between qualitative characteristics was assessed using the $\chi^2$-Pearson test. At a significance level of p less than 0.05, it was believed that the studied indicator in the compared groups had statistically significant differences.

The study was performed in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion in the study.

## RESULTS

Clinical characteristics of the patients are presented in Table 1.

**Table 1: Clinical characteristics of patients.**

| Parameters | Group 1 (n=37) | Group 2 (n=38) | P value |
|---|---|---|---|
| **Male gender, N (%)** | 24 (64.9) | 30 (78.9) | >0.05 |
| **Age, years** | 62 (57; 70) | 65 (57.3; 72) | >0.05 |
| **Body mass index, kg/m$^2$** | 32.7 (31.6; 34.7) | 24 (26; 28) | 0.001 |
| **Hypertension, N (%)** | 36 (97.29) | 33 (86.84) | >0.05 |
| **Diabetes mellitus, N (%)** | 13 (35.13) | 3 (7.89) | 0.04 |
| **Atrial fibrillation, N (%)** | 4 (10.81) | 5 (13.15) | >0.05 |
| **History of MI, N (%)** | 19 (51.36) | 11 (28.9) | >0.05 |
| **Localization of MI** | - | - | |
| Anterior, N (%) | 14 (37.83) | 17 (44.73) | >0.05 |
| Inferior, N (%) | 15 (40.54) | 16 (42.1) | >0.05 |
| Lateral, N (%) | 7 (18.91) | 7 (18.42) | >0.05 |
| Anterior-lateral, N (%) | 3 (8.1) | 3 (7.89) | >0.05 |
| Posterior, N (%) | 7 (18.9) | 5 (13.15) | >0.05 |
| **Complications** | | | |
| Pulmonary edema, N (%) | 11 (29.72) | 12 (31.57) | >0.05 |
| Ventricular arrhythmias, N (%) | 4 (10.81) | 2 (5.26) | >0.05 |
| Heart block, N (%) | 11 (29.72) | 10 (26.3) | >0.05 |

**Table 2: Laboratory parameters of patients [Me (25%;75%)].**

| Parameters | Group 1 (n=37) | Group 2 (n=38) | P value |
|---|---|---|---|
| **RBC, $10^{12}$/l** | 4.95 (4.53; 5.34) | 4.57 [4.26; 5.01) | 0.034 |
| **Hemoglobin, gm/l** | 148.5 (129.5;158.5) | 130.25 (142;152) | >0.05 |
| **WBC, $10^9$/l** | 9.25 (6.97; 10.49) | 9.6 (8.35; 11.1) | >0.05 |
| **ESR, mm/hour** | 13 (6.5; 20.5) | 10.5 (6.25; 17) | >0.05 |
| **Platelets, $10^9$/l** | 237 (191.5; 279.5) | 239.5 (194.5; 263.75) | >0.05 |
| **Urea, mmol/l** | 6.6 (5.1; 7.4) | 5.5 (4.6; 7.3) | >0.05 |
| **Creatinine, µmol/l** | 91.3 (78; 107) | 91 (75.5; 107) | >0.05 |
| **eGFR, ml/minute/1.73 $m^2$** | 75.8 (60.1; 89) | 85 (61.75; 92.625) | >0.05 |
| **Total cholesterol, mmol/l** | 4.55 (3.58; 5.62) | 5.58 (4.04; 6.71) | 0.034 |
| **LDL, mmol/l** | 2.3 (1.7; 3.39) | 2.34 (2.05; 2.7) | >0.05 |
| **HDL, mmol/l** | 0.9 (0.7; 1) | 1.1 (0.89; 1.3) | 0.037 |
| **Triglycerides, gm/l** | 2 (1.6; 2.2) | 1.26 (0.9; 1.6) | 0.001 |
| **Glucose, mmol/l** | 8.65 (6.25; 11.92) | 7.3 (6.2; 8.8) | >0.05 |
| **Sodium, mEq/l** | 140.65 (138.75; 142.25) | 140 (137; 143) | >0.05 |
| **Troponin, ng/l** | 1949 (136; 10685.75) | 1656 (146.7; 14076) | >0.05 |
| **Lactate dehydrogenase, IU/l** | 537 (427.5;699) | 488.5 (417.5;754.25) | >0.05 |
| **Creatine phosphokinase MB, IU/l** | 263 (106.75;547.25) | 286 (121;976) | >0.05 |

**Table 3: Coronary angiography parameters.**

| Parameters | Group 1 (n=37) | Group 2 (n=38) | P value |
|---|---|---|---|
| **Affected vessel** | - | - | - |
| Left main coronary artery, N (%) | 8 (21.62) | 7 (18.43) | >0.05 |
| Left anterior descending artery, N (%) | 33 (89.3) | 25 (65.78) | 0.025 |
| Left circumflex artery, N (%) | 24 (64.86) | 15 (39.47) | 0.03 |
| Right coronary artery, N (%) | 24 (64.86) | 20 (52.63) | >0.05 |
| Ramus intermedius, N (%) | 22 (59.45) | 17 (44.73) | >0.05 |
| Stenting, N (%) | 34 (91.89) | 29 (76.31) | >0.05 |
| Total number of stents | 2 (1; 2) | 1 (1; 2) | >0.05 |
| CABG, N (%) | 3 (8.11) | 9 (23.7) | >0.05 |

Note: CABG- coronary artery bypass graft.

The groups were comparable in age and sex distribution (p>0.05). As expected, body mass index differed significantly between groups (p=0.001). Hypertension prevalence did not differ significantly between obese and non-obese patients (p>0.05).

A notable finding was the significantly higher prevalence of diabetes mellitus among obese patients (35.13% versus 7.89%, p=0.04), reflecting the close pathophysiological relationship between adiposity and glucose metabolism disturbances.

There were no statistically significant differences in myocardial infarction localization, atrial fibrillation prevalence, prior MI history, or most acute complications including pulmonary edema, ventricular arrhythmias, and heart block (p>0.05).

Laboratory parameters of patients are presented in Table 2.

Obese patients demonstrated significantly higher RBC counts (p=0.034). Lipid profile analysis revealed significantly lower total cholesterol (p=0.034) but markedly higher triglyceride levels (p=0.001) and lower HDL cholesterol levels (p=0.037) in the obese group. LDL levels did not differ significantly.

Renal function parameters (urea, creatinine, eGFR), inflammatory markers (WBC, ESR), and cardiac biomarkers (troponin, LDH, CK-MB) were comparable between groups (p>0.05).

Coronary angiography parameters of patients with STEMI are presented in Table 3.

Obese patients more frequently had involvement of the left anterior descending artery (LAD) (89.3% versus 65.78%, p=0.025) and left circumflex artery (LCx) (64.86% versus 39.47%, p=0.03). No significant differences were observed in right coronary artery or ramus intermedius involvement (p>0.05).

The rate of percutaneous coronary intervention and the median number of implanted stents were comparable between groups (p>0.05). Coronary artery bypass grafting rate was almost 3 times higher in non-obese patients, however the difference didn't reach statistically significant margin (p=0.11).

## DISCUSSION

In the present cohort, obesity was primarily associated with metabolic abnormalities rather than with differences in acute clinical presentation.[2] The higher prevalence of diabetes mellitus among obese patients reflects the well-established relationship between excess adiposity and insulin resistance.[5] Adipose tissue is not merely a passive energy storage organ but an active endocrine structure that secretes adipokines, pro-inflammatory cytokines (such as TNF-α and IL-6), and prothrombotic mediators.[6] These factors contribute to endothelial dysfunction, plaque vulnerability, and accelerated coronary atherosclerosis.[2]

Interestingly, although total cholesterol levels were lower in obese patients, triglycerides were significantly higher and HDL cholesterol was significantly reduced. This pattern is typical of atherogenic dyslipidemia and is strongly associated with small dense LDL particles, increased oxidative stress, and enhanced atherothrombosis risk.[10] Therefore, traditional total cholesterol measurements may underestimate cardiovascular risk in obese individuals.[11]

From an angiographic perspective, obese patients more frequently demonstrated involvement of the left coronary circulation, particularly the LAD and LCx.[7,12] The LAD supplies a large portion of the left ventricular myocardium, and its involvement is associated with larger infarct size and worse prognosis.[1] The higher prevalence of LAD and LCx lesions suggests that obesity may be linked to more extensive and anatomically complex coronary artery disease.[13]

Despite these differences in coronary distribution, troponin levels and procedural revascularization rates were comparable between groups. This finding may support the concept that obesity does not necessarily increase acute infarct size but may influence the chronic atherosclerotic burden.[8]

The absence of differences in inflammatory markers such as WBC and ESR suggests that acute inflammatory response to myocardial necrosis may not be significantly modified by BMI alone. However, chronic low-grade inflammation associated with obesity likely contributes to long-term vascular remodelling and plaque instability.[5]

The comparable rates of acute complications, including pulmonary edema and arrhythmias, may indicate that short-term in-hospital outcomes are not significantly worsened by obesity in STEMI patients, aligning partially with observations described in the obesity paradox literature.[13,14] Nevertheless, long-term cardiovascular risk in obese patients remains elevated due to persistent metabolic and vascular abnormalities.[14]

Overall, our findings suggest that obesity in STEMI patients is primarily associated with metabolic dysregulation and more frequent multi-vessel coronary involvement, rather than differences in immediate procedural outcomes.

This study has several limitations. First, it was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Second, obese patients were not stratified according to obesity class (I, II, III), preventing analysis of dose-response relationships between BMI severity and coronary involvement. Third, measures of central obesity (waist circumference, visceral fat assessment) were not evaluated, although they may better reflect cardiometabolic risk than BMI alone. Finally, long-term follow-up data were not included, which restricts assessment of prognostic implications.

Future multicenter studies with larger populations and extended follow-up are necessary to clarify the long-term impact of obesity on outcomes after STEMI.

## CONCLUSION

Obesity in patients with STEMI is associated with a higher prevalence of diabetes mellitus and atherogenic dyslipidemia characterized by elevated triglycerides and reduced HDL cholesterol levels. Angiographically, obese patients more frequently demonstrate involvement of the LAD and LCx arteries, suggesting more extensive coronary artery disease. However, acute laboratory markers of myocardial necrosis, renal function, and in-hospital complication rates did not significantly differ between obese and non-obese patients.

These findings highlight the complex interaction between metabolic disturbances and coronary atherosclerosis in obese individuals and emphasize the importance of aggressive risk factor modification in this population.

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