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**Original Research Article**

# Factors associated with reduced left ventricular ejection fraction in patients with first-time myocardial infarction

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## ABSTRACT

**Background:** Heart failure (HF) is a leading cause of death after acute myocardial infarction (MI), and reduced left ventricular ejection fraction (LVEF) significantly worsens prognosis. However, comprehensive analyses integrating clinical, laboratory, and echocardiographic predictors of reduced LVEF after first-time MI remain scarce.
**Methods:** This single-centre retrospective study included 109 patients hospitalized with first-time acute MI between January 2024 and May 2026. Patients were divided into two groups: MI with reduced LVEF (<50%, n=50) and MI with preserved LVEF (≥50%, n=69). Clinical, laboratory, echocardiographic, and coronary angiography data were compared using non-parametric methods.
**Results:** Patients with reduced LVEF were significantly older (65.74 vs. 57.41 years, p<0.001), more often had hypertension (92% vs. 78%, p=0.047) and anterior MI (70% vs. 51%, p=0.040). Laboratory findings included lower hemoglobin and red blood cell counts (p<0.001), higher ESR (p<0.001), elevated urea (p=0.002), creatinine (p=0.006), glucose (p=0.018), and lactate dehydrogenase (p=0.01), with lower eGFR (p=0.037). Echocardiography showed larger LV dimensions, greater volumes, more akinesis/hypokinesis, and more pleural effusions (p<0.001). Coronary angiography revealed more left circumflex involvement (54% vs. 30%, p=0.014) and more affected vessels (p=0.011).
**Conclusions:** Reduced LVEF after first-time MI is associated with advanced age, hypertension, anterior infarction, anemia, inflammation, renal impairment, hyperglycemia, adverse remodelling, and more extensive coronary disease. Early identification of these factors may improve risk stratification and outcomes.

**Keywords:** Heart failure, Myocardial infarction, Left ventricular ejection fraction, Troponin, Coronary angiography, Echocardiography, Stenting

## INTRODUCTION

Heart failure (HF) is now the leading cause of death in patients with acute myocardial infarction (MI), with studies showing it develops in anywhere from 14% to 36% of those admitted for a heart attack.[1] Patients who go on to develop HF after MI face much worse outcomes than those who don't higher short-term and long-term mortality, more recurrent heart attacks, and repeated hospitalizations.[2] The role of left ventricular ejection fraction (LVEF) in all of this is particularly important. We've known for a long time that left ventricular systolic dysfunction is a major predictor of survival after acute MI, and when it gets worse, outcomes get worse too.[3] But here's something encouraging: among patients with reduced LVEF after MI, those whose systolic function actually recovers have been shown to have roughly an 8-fold reduction in all-cause mortality and a 10-fold reduction in cardiovascular mortality over long-term follow-up.[4]

The mechanisms behind post-MI heart failure are complicated and involve multiple interconnected processes. Adverse left ventricular remodeling where the

heart changes in size, shape, and function—sits right at the center of the transition from acute injury to chronic HF.[5] This process is driven by a mix of mechanical, neurohormonal, and inflammatory factors that together weaken the heart's pumping ability and lead to progressive systolic dysfunction. Residual myocardial ischemia has also been identified as an independent determinant of left ventricular systolic function after MI, and when it's present, contractility tends to worsen over time. More recent evidence suggests that inflammation, metabolic problems, and kidney impairment might also contribute to heart failure development and progression after MI.[6] All of this points to the need for a comprehensive approach to risk assessment–one that pulls together clinical information, laboratory markers, and imaging findings rather than relying on any single factor.

Despite how much we know about post-MI heart failure, there are still important gaps in our understanding. Specifically, we don't have a clear picture of which factors–especially those we can easily measure in routine practice are most strongly linked to reduced LVEF after a first-time MI. Previous studies have looked at various individual predictors, but comprehensive analyses that integrate clinical characteristics, laboratory biomarkers, and echocardiographic findings in one patient cohort are scarce.[7] If we can identify which factors-both modifiable and non-modifiable-are associated with reduced LVEF in this group, we could design targeted interventions to preserve left ventricular function and stop heart failure from progressing.

The aim of the current study was to reveal the clinical, laboratory, and echocardiographic factors associated with reduced LVEF in patients with first-time MI. By systematically characterizing this patient population and pinpointing what's linked to left ventricular systolic dysfunction, we hoped to contribute to better risk stratification strategies and highlight potential targets for treatment in this high-risk group.

## METHODS

### *Study design and patient selection*

This single-centre retrospective study included 119 patients hospitalized to the Grodno Regional Clinical Cardiological Centre (Grodno, Belarus) between January 2024 and May 2026 with newly diagnosed acute MI. Inclusion criteria were: age ≥18 years; confirmed acute MI based on the criteria of the fourth universal definition of MI; coronary angiography within 24 hours of symptom onset.

The diagnostic criteria for MI referred to clinical evidence of elevated or decreased levels of high sensitivity troponin level exceeding the upper limit of the reference value by at least 99%, accompanied by acute myocardial ischemia (as long as one condition is met), including myocardial ischemia clinical symptoms, new ischemic electrocardiographic changes, imaging evidence of abnormalities of ventricular wall phase motion and coronary angiography confirming coronary thrombosis.

### *Exclusion criteria*

Exclusion criteria from the study were: age <18 years; inflammatory diseases of the myocardium, presence of congenital or acquired valvular pathology requiring surgical correction; severe renal failure (glomerular filtration rate <30 ml/min/1.73 $m^2$); active inflammatory process of any localization of infectious, autoimmune or other etiology; oncological diseases; pregnancy or lactation; hypersensitivity to contrast used in coronary angiography; patient's refusal to participate in the study.

### *Assessment methods*

Clinical and demographic data, the results of basic laboratory tests, medical history of comorbidities were collected for all patients.

All patients underwent coronary angiography according to the Judkins technique (1967) in a cath lab using Philips Azurion 7 and GE Innova 3100 IQ angiographic systems. The GE Innova 3100 IQ computer program was used for quantitative assessment of stenoses.

Echocardiography was performed on Phillips iE33 device with a multi-frequency sensor (frequency 2.5-5.0 MHz). The examination was performed with the patient lying on his left side with his back to the researcher or on his back. The study protocol included the following indicators: LA and right atrium (RA) diameter in 2-chamber and 4-chamber mode, end-systolic diameter and end-diastolic diameter (mm) of the left ventricle (LV), LVEF; assessment of the state of the valvular apparatus of the heart, degree of regurgitation on the valves.

### *Statistical methods*

Statistical analysis was performed using the STATISTICA 12.0 computer software. Data distribution was assessed for normality via histogram analysis. Sets of quantitative indicators whose distribution deviated from normal were described using median (Me) values and lower and upper quartiles (Q1; Q3). Nominal data were described using absolute values and percentages. Given that most quantitative variables were not normally distributed, non-parametric methods were applied. Differences between two independent groups were evaluated by Mann-Whitney test, alongside a p-value of less than 0.05 considered statistically significant.

### *Ethical statement*

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

Based on the echocardiography data, two groups of patients were formed. The first group comprised of patients with MI+HF with reduced LVEF (LVEF<50%)–50 patients and the second comprised of patients with MI+HF with preserved LVEF (LVEF≥50%)–69 patients.

Clinical and anamnestic characteristics of the patients are presented in Table 1.

Gender distribution was similar between groups, with male patients making up 70% of Group 1 and 64% of Group 2. However, patients with reduced LVEF were significantly older (65.74 years on average compared to 57.41 years, p<0.001) suggesting that advanced age increases vulnerability to systolic dysfunction after MI.

Patients with reduced LVEF had a lower median BMI (p=0.038) and lower obesity prevalence (22% vs. 41%; p=0.047). This apparent paradox may reflect the well-documented "obesity paradox" in cardiovascular disease, where obese patients sometimes show better short-term outcomes.

Hypertension was significantly more common in the reduced LVEF group (92% vs. 78%; p=0.047), though hypertension stage distribution was similar between groups. Diabetes mellitus prevalence was comparable (8% vs. 12%; p=0.75). Atrial fibrillation showed a trend toward being more frequent in the reduced LVEF group (16% vs. 4%; p=0.051), which is clinically noteworthy given its potential to exacerbate HF.

Stenting rates (p=0.698) and thrombolytic therapy use (p=1.000) were similar between groups, indicating consistent application of acute reperfusion strategies. However, anterior MI was significantly more common in the reduced LVEF group (70% vs. 51%; p=0.040), consistent with the understanding that anterior infarcts involve larger myocardial territories and are more likely to impair systolic function. Other infarct localizations did not differ significantly.

The reduced LVEF group experienced more in-hospital complications across the board. Ventricular arrhythmias were significantly more frequent (22% vs. 4%; p=0.007). Pulmonary edema (6% vs. 0%; p =0.072), cardiogenic shock (6% vs. 0%; p=0.073), and heart block (6% vs. 0%; p=0.072) all showed trends toward higher prevalence in the reduced LVEF group, though these did not reach statistical significance.

Laboratory parameters of patients are presented in Table 2.

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

| Parameters | Group 1 (n=50) | Group 2 (n=69) | P value |
|---|---|---|---|
| **Male gender, N (%)** | 35 (70%) | 44 (64%) | 0.557 |
| **Age, years** | 65.74 [59.2; 73.5] | 57.41 [50; 64] | <0.001 |
| **Body mass index, kg/m$^2$** | 28.28 [24.9; 29.8] | 29.83 [27; 32.3] | 0.038 |
| **Obesity, N (%)** | 11 (22%) | 28 (41%) | 0.047 |
| **Overweight, N (%)** | 22 (44%) | 19 (28%) | 0.079 |
| **Hypertension, N (%)** | 46 (92%) | 54 (78%) | 0.047 |
| **Stage 1, N (%)** | 8 (17%) | 13 (24%) | 0.809 |
| **Stage 2, N (%)** | 34 (74%) | 37 (69%) | 0.133 |
| **Stage 3, N (%)** | 4 (9%) | 4 (7%) | 0.719 |
| **Diabetes mellitus, N (%)** | 4 (8%) | 8 (12%) | 0.759 |
| **Atrial fibrillation, N (%)** | 8 (16%) | 3 (4%) | 0.051 |
| **Stenting, N (%)** | 34 (68%) | 44 (64%) | 0.698 |
| **Thrombolytic therapy, N (%)** | 8 (16%) | 11 (16%) | 1.000 |
| **Localization of MI** | | | |
| Anterior, N (%) | 35 (70%) | 35 (51%) | 0.040 |
| Inferior, N (%) | 5 (10%) | 13 (19%) | 0.437 |
| Lateral, N (%) | 0 (0%) | 0 (0%) | 1.000 |
| Anterior-lateral, N (%) | 1 (2%) | 5 (7%) | 0.399 |
| Inferior-lateral, N (%) | 6 (12%) | 6 (9%) | 0.556 |
| Posterior-lateral, N (%) | 1 (2%) | 0 (0%) | 0.420 |
| Posterior, N (%) | 1 (2%) | 1 (1%) | 1.000 |
| **Complications of MI** | | | |
| Pulmonary edema, N (%) | 3 (6%) | 0 (0%) | 0.072 |
| Cardiogenic shock, N (%) | 3 (6%) | 0 (0%) | 0.072 |
| Ventricular arrhythmia, N (%) | 11 (22%) | 3 (4%) | 0.007 |
| Heart block, N (%) | 3 (6%) | 0 (0%) | 0.072 |

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

| Parameters | Group 1 (n=50) | Group 2 (n=69) | P value |
|---|---|---|---|
| **RBC, $10^{12}$/l** | 4.39 [4.06;4.84] | 4.805 [4.49; 5.11] | <0.001 |
| **Hemoglobin, g/l** | 136.9 [125;153] | 150.2 [142; 160] | <0.001 |
| **WBC, $10^9$/l** | 9.63 [7.8; 11.4] | 10.059 [7.3;11.6] | 0.497 |
| **ESR, mm/h** | 18.24 [8; 31] | 8.58 [4; 10] | <0.001 |
| **Platelets, $10^9$/l** | 234.73 [199; 254] | 250.1 [199.25;288] | 0.350 |
| **Urea, mmol/l** | 7.5 [5.6; 8.5] | 5.4 [4.3;6.3] | 0.002 |
| **Creatinine, µmol/l** | 92.1 [70; 101] | 76.2 [65.1;83] | 0.0062 |
| **eGFR, ml/min/1.73m$^2$** | 73.5 [49.2; 86.9] | 84.98 [72.1; 101] | 0.037 |
| **Total cholesterol, mmol/l** | 4.97 [4.1; 5.6] | 5.298 [4.24; 5.98] | 0.317 |
| **Glucose, mmol/l** | 9 [5.9; 9.1] | 6.9 [5.1; 7.35] | 0.018 |
| **LDL, mmol/l** | 2.73 [2.1;3.26] | 2.48 [1.7; 2.8] | 0.188 |
| **HDL, mmol/l** | 1.09 [0.8; 1.31] | 3.38 [0.86; 1.15] | 0.690 |
| **Triglycerides, g/l** | 1.51 [1.09; 1.85] | 2.11 [1.21; 2.24] | 0.109 |
| **AST, IU/l** | 66.9 [25.67; 83.42] | 65.01 [22.9; 83.9] | 0.652 |
| **ALT, IU/l** | 50.3 [24.1; 60.9] | 38.2 [23; 47.7] | 0.526 |
| **Sodium, mEq/l** | 139.8 [138; 142] | 140.5 [140; 142] | 0.176 |
| **Potassium, mEq/l** | 4.3 [3.6; 4.5] | 4.3 [4; 4.64] | 0.117 |
| **Troponin, ng/l** | 14846 [402; 25002] | 13165 [1027; 15738] | 0.786 |
| **Lactate dehydrogenase, IU/l** | 997 [451; 897] | 601.21 [339.25; 827] | 0.010 |
| **Creatine phosphokinase MB, IU/l** | 533.4 [82.5; 737] | 605.64 [150.25; 745] | 0.605 |

RBC–red blood cells; WBC–white blood cells; ESR - erythrocyte sedimentation rate; eGFR–estimated glomerular filtration rate; LDL–low density lipoproteins, HDL–high density lipoproteins, AST–aspartate aminotranferase; ALT–alanin aminotranferase.

Regarding the reported source of fire, cylinder gas explosions accounted for the largest proportion of injuries, 40.0%, followed closely by wood fires, 37.2%. Concerning the location of the incident, kitchens accounted for 38.0% of cases and courtyards for 25.6%, whereas workplaces accounted for 14.0%. By motive, 218 (87.2%) injuries were reported as accidental, while 7.2% were self-inflicted and 5.6% were politically motivated (Table 2).

Several blood count measures differed meaningfully between the two groups. Patients with reduced LVEF had significantly lower red blood cell counts (p<0.001) and lower hemoglobin levels (p<0.001). White blood cell counts were similar between groups (p=0.497), however, the erythrocyte sedimentation rate was substantially higher in the reduced LVEF group (p<0.001).

Kidney function markers revealed important differences. Patients with reduced LVEF had significantly higher serum urea (p=0.002) and creatinine levels (p=0.006), along with a significantly lower estimated glomerular filtration rate (p=0.037). Blood glucose levels were significantly higher in the reduced LVEF group (p=0.018), suggesting that hyperglycemia may contribute to adverse outcomes after MI. Lipid profiles, however, showed no significant differences between groups (p<0.05).

Among cardiac injury markers, lactate dehydrogenase was significantly higher in the reduced LVEF group (997.55 [451; 897] IU/L vs. 601.21 [339.25; 827] IU/L; p=0.01), suggesting more extensive myocardial damage in these patients. Troponin levels, while numerically higher in the reduced LVEF group, did not differ significantly between groups (p=0.786). Similarly, creatine phosphokinase MB levels were comparable (p=0.605). Liver enzymes also showed no significant differences between groups (p<0.05).

Left ventricular size parameters revealed striking differences between the two groups (Table 3). Left ventricular end-systolic diameter was significantly larger in the reduced LVEF group (p<0.001), indicating greater chamber dilation during systole (Table 3). Left ventricular end-diastolic diameter was also significantly greater in patients with reduced LVEF (p=0.003).

Volumetric assessment by M-mode echocardiography showed that patients with reduced LVEF had substantially larger left ventricular end-systolic volumes, p=0.001. As expected, based on group assignment, LVEF was significantly lower in Group 1 (p<0.001). When assessed by B-mode echocardiography, similar patterns emerged. The consistency between M-mode and B-mode findings reinforces the reliability of these observations.

Septal thickness during systole was significantly lower in the reduced LVEF group (16.18 mm vs. 17.23 mm; p=0.042), suggesting reduced systolic thickening of the interventricular septum. However, septal thickness during diastole was nearly identical between groups (12.84 mm vs. 12.81 mm; p=0.912). Posterior wall thickness during systole was also significantly lower in the reduced LVEF

group (15.18 mm vs. 15.92 mm; p=0.037), while posterior wall thickness during diastole showed no significant difference (p=0.891). These findings indicate impaired systolic thickening of the ventricular walls in patients with reduced LVEF, consistent with diminished contractile function.

**Table 3: Echocardiographic parameters of patients (Me [25%;75%]).**

| Parameters | Group 1 (n=50) | Group 2 (n=69) | P value |
|---|---|---|---|
| **LA diameter (2 chamber), mm** | 38.29 [36;42] | 37.25 [35;45] | 0.285 |
| **LA diameter (medial to lateral), mm** | 40.75 [38;44] | 38.49 [36;40] | 0.0045 |
| **LA diameter (front to back), mm** | 54.81 [50;59] | 51.96 [49;54] | 0.036 |
| **RA diameter (medial to lateral), mm** | 37.93 [34.5;42] | 35.76 [34;38] | 0.059 |
| **RA diameter (front to back), mm** | 51.08 [46;55.75] | 48.06 [45.25;51] | 0.153 |
| **LV ESD, mm** | 39.46 [35;44.75] | 33.89 [32;35] | <0.001 |
| **LV EDD, mm** | 53.16 [49;57.75] | 50 [48;52] | 0.003 |
| **M-mode** | | | |
| LV ESV, ml | 81.16 [62.5;101.5] | 46.93 [39;52.25] | <0.001 |
| LV EDV, ml | 150.65 [125;175.5] | 122.71 [108; 134] | <0.001 |
| LVEF, % | 46.77 [42;53] | 61.51 [57.75; 64.25] | <0.001 |
| LV ESV, ml | 81.16 [62.5;101.5] | 46.93 [39;52.25] | <0.001 |
| **B-mode** | | | |
| LV ESV, ml | 83.82 [67.75;103] | 52.02 [47;55] | 0.0000 |
| LV EDV, ml | 145.18 [116; 171] | 120.21 [107.75;128] | <0.001 |
| LVEF, % | 41.5 [36.25; 47] | 56.74 [54;59] | <0.001 |
| Septal Thickness (systolic), mm | 16.18 [15;18] | 17.23 [16;18] | 0.042 |
| Septal Thickness (diastolic), mm | 12.84 [11;14] | 12.81 [11;14] | 0.912 |
| Posterior wall thickness(systolic), mm | 15.18 [13.25;16.75] | 15.92 [15;17] | 0.037 |
| Posterior wall thickness (diastolic), mm | 11.42 [10;13] | 11.37 [10;12] | 0.891 |
| Right ventricle diameter, mm | 26.55 [24;28.5] | 26.44 [24;27] | 0.426 |
| Contractility index | 1.69 [1.4;1.94] | 1.24 [1.13;1.38] | <0.001 |
| Akinesis areas. | 42 (84%) | 36 (52%) | <0.001 |
| Akinesis mean number | 3.72 [1;6] | 3.08 [0;2] | <0.001 |
| Hypokinesis areas. | 41 (82%) | 38 (55%) | 0.003 |
| Hypokinesis mean number | 3.42 [1; 5] | 3.1 [0; 3] | <0.001 |
| Pericardial effusion, N (%) | 3 (6%) | 1 (1.45%) | 0.308 |
| Pleural effusion, N (%) | 11 (22%) | 1 (1.45%) | <0.001 |

LA–left atrium; RA–right atrium; LV–left ventricle; ESD–end-systolic diameter; EDD–end-diastolic diameter; ESV–end-systolic volume; EDV–end-diastolic volume; LVEF–left ventricular ejection fraction.

**Table 4: Coronary angiography parameters.**

| Parameters | Group 1 (n=50) | Group 2 (n=69) | P value |
|---|---|---|---|
| **Affected vessel** | | | |
| Left main coronary artery, N (%) | 6 (12%) | 5 (7%) | 0.523 |
| Left anterior descending artery, N (%) | 36 (72%) | 40 (58%) | 0.127 |
| Left circumflex artery, N (%) | 27 (54%) | 21 (30%) | 0.014 |
| Right coronary artery, N (%) | 17 (34%) | 27 (39%) | 0.701 |
| Left posterior descending artery, N (%) | 6 (12%) | 7 (10%) | 0.773 |
| Diagonal branch, N (%) | 12 (24%) | 12 (17%) | 0.488 |
| Obtuse marginal artery 1, N (%) | 15 (30%) | 12 (17%) | 0.124 |
| Obtuse marginal artery 2, N (%) | 8 (16%) | 4 (6%) | 0.120 |
| Stenting, N (%) | 30 (60%) | 43 (62%) | 0.850 |
| Total number of stents | 2.3 [2;3] | 2.24 [2;3] | 0.069 |
| Balloon dilation, N (%) | 29 (58%) | 31 (45%) | 0.195 |
| Total number of balloons | 2.03 [1;3] | 1.97 [1;3] | 0.015 |
| Total occlusion, N (%) | 36 (72%) | 48 (70%) | 0.273 |
| Number of vessels affected | 3 [2;4] | 2.93 [2;4] | 0.011 |

A significantly greater proportion of patients in the reduced LVEF group demonstrated akinesis (p<0.001), and the mean number of akinetic segments was also higher (p<0.001). Similarly, hypokinesis was more common in the reduced LVEF group (p=0.003), with a higher mean number of hypokinetic segments (p<0.001). These findings confirm that patients who develop reduced LVEF after MI have more extensive myocardial damage and wall motion abnormalities.

Pericardial effusion was uncommon in both groups; however, pleural effusion was significantly more common in the reduced LVEF group (22% vs. 1.45%, p=0.0003), reflecting the clinical consequences of elevated filling pressures and fluid retention in HF.

Left main coronary artery disease was present in 12% of Group 1 patients compared to 7% of Group 2, though this difference was not statistically significant (p=0.523) (See Table 4). Left anterior descending artery involvement was more common in the reduced LVEF group (72% vs. 58%), but again this did not reach statistical significance (p=0.127). However, left circumflex artery involvement was significantly more frequent in patients with reduced LVEF (54% vs. 30%; p=0.014).

Stenting was performed in similar proportions of patients in both groups (p=0.850). The total number of stents used showed a trend toward being higher in the reduced LVEF group, though this difference approached but did not reach statistical significance (p=0.069). Balloon dilation was performed in 58% of Group 1 patients compared to 45% of Group 2 (p=0.195). Total occlusion of the infarct-related artery was observed in 72% of Group 1 patients and 70% of Group 2 patients, a difference that was not statistically significant (p=0.273). However, the number of vessels affected was significantly higher in the reduced LVEF group (p=0.011), indicating more extensive coronary artery disease in patients who went on to develop left ventricular systolic dysfunction.

## DISCUSSION

Identifying predictors of a decline in LVEF in patients with MI remains an important clinical challenge. Currently, medicine offers a wide range of tools to modify risk factors for the development of chronic HF in such patients. Early restoration of adequate coronary blood flow helps limit the infarct zone, preserve left ventricular systolic function, and reduce both pathological LV remodeling and the likelihood of LV aneurysm formation. Identifying the factors that influence LVEF and the development of HF in patients with MI may help pinpoint those individuals who would derive the greatest benefit from these therapies.

In a study by Wohlfahrt et al, 52.9% of patients with myocardial infarction (MI) (65.4% of whom had STEMI) presented with LV systolic dysfunction at the time of hospital admission: 22.3% had reduced LV ejection fraction (LVEF), and 30.6% had impaired (but not reduced) LVEF.[9] In a study by Lenselink et al, 54.9% of patients with STEMI had LV systolic dysfunction: 19.3% of patients had reduced LVEF, and 35.9% had impaired (but not reduced) LVEF.[10]

Looking at the clinical and anamnestic characteristics of our patients adds more context. Those with reduced LVEF had a lower body mass index (28.28 vs. 29.83 kg/m²; p=0.038) and lower obesity prevalence (22% vs. 41%; p=0.047). At first glance this seems odd given that obesity is a cardiovascular risk factor. But it might be explained by the "obesity paradox"—where obese patients with existing heart disease sometimes do better in the short term, perhaps because they have more metabolic reserve or different inflammatory profiles.[11] AF was more common in the reduced LVEF group (16% vs. 4%; p=0.051), and while this just missed statistical significance, it's clinically important because AF can worsen heart failure by eliminating the atrial contribution to filling and causing irregular ventricular rates.[12,13] Hypertension stage distribution didn't differ much between groups, though overall hypertension prevalence was higher in the reduced LVEF group.

A study by Choi et al, found that hypertension, diabetes, AF, CKD, and peripheral vascular disease were significant predictors of the development of HF after MI.[14] Another study by Gho et al demonstrated that a history of diabetes, AF, and smoking were independent prognostic factors for the development of HF after MI.[15]

Laboratory results showed lower red blood cell counts and hemoglobin, higher ESR, and higher glucose levels in patients with reduced LVEF. Lower oxygen-carrying capacity could plausibly aggravate myocardial oxygen imbalance, while elevated glucose during acute MI may reflect stress hyperglycemia or pre-existing dysglycemia.[3] The higher ESR may indicate a greater inflammatory response, although it is a nonspecific marker and timing of measurement can affect its interpretation.[6]

Renal markers were less favorable in the reduced-LVEF group, with higher urea and creatinine and lower eGFR. This pattern is clinically relevant because kidney dysfunction and HF can interact through changes in perfusion, venous congestion, and neurohormonal activation.[7,10]

The echocardiographic findings were consistent with more pronounced systolic impairment and adverse ventricular remodeling in the reduced-LVEF group. Differences in systolic wall thickness, with no corresponding differences in diastolic thickness, may reflect impaired systolic thickening rather than differences in baseline wall structure.

LVEF determined by transthoracic echocardiography, remains an important predictor of survival in patients with

acute coronary syndrome and is widely used in routine clinical practice. A value of <40% is associated with the death of 15% of patients within 6 months following the index event.[6]

Angiography showed more frequent left circumflex artery involvement and a higher number of affected vessels in the reduced-LVEF group. These findings suggest that the burden and distribution of coronary atherosclerosis may contribute to post-MI systolic dysfunction. In contrast, involvement of the left main, LAD, and right coronary arteries did not differ significantly, and rates of stenting and total occlusion were broadly similar.

These findings show just how complicated the relationship is between traditional risk factors and post-MI HF. They also remind us that we need to look at the whole picture, not just individual parameters, when assessing risk. Combining a patient's clinical features with selected invasive or non-invasive risk stratification methods can significantly improve risk stratification following MI.

Clinical prediction rules are the most useful methodological tool for addressing the issues outlined. These rules combine various indicators such as medical history, clinical signs, and diagnostic test results to forecast the course of a disease or its outcome, thereby guiding clinical decision-making regarding a patient's subsequent management.

### *Study limitations*

Our study has several limitations that need to be acknowledged. The sample size was small (n=119) and came from a single centre, which limits how well our findings apply to other populations and reduces our ability to detect small but meaningful differences. The retrospective design means we can't establish cause and effect, and there's potential for selection bias. We also didn't assess several factors that could influence post-MI left ventricular function—things like genetic polymorphisms, socioeconomic status, medication adherence, and detailed treatment characteristics such as door-to-balloon time or whether revascularization was complete. Without long-term follow-up data, we can't say much about what our findings mean for prognosis. Using different echocardiographic modalities (M-mode and B-mode) for LVEF assessment might have introduced some measurement variability, though both methods showed consistent differences between groups. Finally, we only included patients with first-time MI, so our results may not apply to those with recurrent events or prior revascularization. Despite these limitations, our study offers useful insights into the clinical, laboratory, and echocardiographic factors linked to reduced LVEF after first-time MI, and it highlights several potentially modifiable targets for future research.

## CONCLUSION

To sum up, this single-centre retrospective study pinpointed several clinical, laboratory, and echocardiographic factors associated with reduced left ventricular ejection fraction in patients with first-time acute MI. Those who developed HF with reduced LVEF tended to be older, more often had hypertension, were more likely to have anterior MI, and experienced more in-hospital complications like ventricular arrhythmias, pulmonary edema, and cardiogenic shock. On the laboratory side, reduced LVEF was linked to lower red blood cell counts and hemoglobin, higher inflammatory markers (ESR), worse kidney function (higher urea and creatinine, lower eGFR), higher blood glucose, and elevated lactate dehydrogenase. This suggests that anemia, inflammation, renal dysfunction, and metabolic problems all contribute to the pathophysiology of post-MI systolic dysfunction. Echocardiography showed that patients with reduced LVEF had larger left atrial and ventricular dimensions, greater end-systolic and end-diastolic volumes, more extensive wall motion abnormalities, and more pleural effusions—all signs of adverse remodeling and high filling pressures. Coronary angiography revealed more extensive coronary disease in the reduced LVEF group, with more left circumflex involvement and more affected vessels, confirming that the extent and severity of coronary atherosclerosis are key determinants of left ventricular function after MI.

These findings matter clinically because they suggest that catching patients at risk for reduced LVEF early using a mix of clinical, laboratory, and imaging parameters could help us start evidence-based treatments sooner. That includes guideline-directed medical therapy, cardiac rehabilitation, and considering device therapy when appropriate, all of which could improve outcomes for this high-risk group. Larger, multicentre, prospective studies with long-term follow-up are needed to confirm and build on what we found, and to develop risk prediction models that can actually guide clinical decisions for patients with first-time MI. Future research should also dig into how genetic, socioeconomic, and treatment-related factors affect the risk of post-MI left ventricular dysfunction, and should test whether targeted interventions can preserve left ventricular function and stop heart failure from getting worse in this vulnerable population.

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