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Left ventricular systolic function in patients with myocardial infarction and iron deficiency during correction with iron supplements

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ABSTRACT

BACKGROUND: Iron deficiency is associated with worse contractile function of the heart in patients after myocardial infarction.**AIM:** To study the contractile function of the left ventricle in patients with myocardial infarction and iron deficiency for 12 months while taking iron supplements.**MATERIAL AND METHODS:** The study included 83 patients with myocardial infarction and iron deficiency. The average age was 62.0 ± 11 years. The patients underwent drug correction of iron deficiency by parenteral administration of iron carboxymaltose or oral administration of iron sulfate. After 3 months, the patients were divided into two groups depending on the compensation of iron deficiency. The first group consisted of 58 (70%) patients with compensated iron deficiency, the second group — 25 (30%) patients with persistent deficiency. The patients underwent echocardiography with assessment of the left ventricular ejection fraction and the total index of its myocardial mobility in the first 24 hours after hospitalization, after 3, 6 and 12 months. Comparison of mean values was performed using the Mann–Whitney U-test. Differences in indicators were considered statistically significant at $p < 0.05$.**RESULTS:** In the first 24 hours after hospitalization for myocardial infarction, the ejection fraction did not differ in patients: in the first group — 48% [45; 54], in the second — 53% [48; 54] ($p=0.07$). In the first group, an increase in the ejection fraction was found compared to the baseline value: 53% [46; 58] ($p < 0.001$) 6 months after myocardial infarction, 55% [48; 58] ($p < 0.001$) after 12 months. In the second group, the ejection fraction after 3, 6 and 12 months did not differ from the baseline. The total myocardial mobility index on the 1st day after myocardial infarction did not differ between the groups: 1.25 [1.19; 1.62] in the first group and 1.25 [1.12; 1.56] in the second group ($p=0.3$). Its decrease was found in the first group: 1.19 [1.06; 1.56] ($p < 0.001$) after 6 months and 1.12 [1.0; 1.44] ($p < 0.001$) after 12 months. In the second group, the values of the total myocardial mobility index after 3, 6 and 12 months did not differ from the initial ones.**CONCLUSION:** Iron deficiency compensation is associated with improved left ventricular systolic function within 12 months after myocardial infarction.**Keywords:** iron deficiency; myocardial infarction; left ventricular systolic function.

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Оригинальное исследование | УДК 616.12-008.1-616.152.72-616.1/.4-0728

Систолическая функция левого желудочка у пациентов с инфарктом миокарда и дефицитом железа на фоне коррекции препаратами железа

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АННОТАЦИЯ

Актуальность. Дефицит железа ассоциирован с худшими показателями сократительной функции сердца у пациентов, перенёсших инфаркт миокарда.

Цель. Изучить сократительную функцию левого желудочка у пациентов с инфарктом миокарда и дефицитом железа в течение 12 мес на фоне применения препаратов железа.

Материал и методы. В исследование включены 83 пациента с инфарктом миокарда и дефицитом железа. Средний возраст составил $62,0 \pm 11$ лет. Пациентам проводили медикаментозную коррекцию дефицита железа путём парентерального введения карбоксимальтозата железа или перорального приема железа сульфата. Через 3 мес пациенты были разделены на две группы в зависимости от компенсации дефицита железа. Первую группу составили 58 (70%) пациентов с компенсированным дефицитом железа, вторую группу — 25 (30%) пациентов с сохраняющимся дефицитом. Пациентам проведена эхокардиография с оценкой фракции выброса и индекса суммарной подвижности миокарда левого желудочка в первые 24 ч после госпитализации, через 3, 6 и 12 мес. Сравнение средних величин выполнено с использованием U-критерия Манна–Уитни. Различия показателей считали статистически значимыми при $p < 0,05$.

Результаты. В первые 24 ч после госпитализации по поводу инфаркта миокарда фракция выброса у пациентов не различалась: в первой группе — 48% [45; 54], во второй — 53% [48; 54] ($p=0,07$). В первой группе обнаружено увеличение фракции выброса по сравнению с исходным значением: 53% [46; 58] ($p < 0,001$) через 6 мес после инфаркта миокарда, 55% [48; 58] ($p < 0,001$) через 12 мес. Во второй группе фракция выброса через 3, 6 и 12 мес не отличалась от исходной. Суммарный индекс подвижности миокарда в 1-е сутки после инфаркта миокарда не различался между группами: 1,25 [1,19; 1,62] в первой группе и 1,25 [1,12; 1,56] во второй группе ($p=0,3$). Было обнаружено его снижение в первой группе: 1,19 [1,06; 1,56] ($p < 0,001$) через 6 мес и 1,12 [1,0; 1,44] ($p < 0,001$) через 12 мес. Во второй группе значения суммарного индекса подвижности миокарда через 3, 6 и 12 мес не отличались от исходных.

Вывод. Компенсация дефицита железа ассоциирована с улучшением систолической функции левого желудочка в течение 12 мес после инфаркта миокарда.

Ключевые слова: дефицит железа; инфаркт миокарда; систолическая функция левого желудочка.

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BACKGROUND

Iron deficiency (ID) is the most prevalent micronutrient deficiency worldwide, resulting from an absolute decrease in iron stores and a decrease in the functional pool of iron [1]. Iron is crucial in oxygen transport and various metabolic processes. ID can disrupt mitochondrial metabolism in skeletal muscle cells and cardiomyocytes [2, 3]. Animal studies have shown that ID induce myocardial structural changes and myocardial dysfunction [4–6].

In Russia, coronary artery disease (CAD) prevalence reaches 15%–17%, with a high annual mortality rate of 27%. Myocardial infarction (MI) is the first manifestation in approximately 50% of patients with CAD [7]. ID is common in patients with CAD [8]; however, most studies have evaluated ID in patients with diagnosed anemia or established heart failure (HF). The effect of ID on cardiac function and myocardial structural changes in patients with CAD, particularly after MI, has not been adequately studied. Hence, ID is rarely diagnosed in patients without HF and remains untreated in most cases until anemia develops.

A few studies have shown that patients with ID have worse left ventricular (LV) systolic function than those with normal iron status, during and 6 months after hospitalization for MI [9, 10]. Moreover, ID is associated with a higher probability of adverse LV remodeling [9]. However, despite the existing evidence on the effects of ID on systolic cardiac function in patients with MI, there are no virtually available data on the effects of ID treatment in this group of patients.

This study aimed to evaluate the contractile function of LV by assessing changes in its ejection fraction and total myocardial mobility index in patients with MI and ID during 12 months of iron supplementation.

MATERIALS AND METHODS

This open prospective study included 83 patients with ID (46 [56%] men and 37 [44%] women) admitted to the Emergency Cardiology Department of the M.N. Sadykov City Clinical Hospital No. 7 (Kazan) in 2022–2023 for ST segment elevation MI and emergency percutaneous coronary intervention with successful revascularization of the MI-related artery. The mean age of the patients was 62.0 ± 11.0 years.

The eligibility criteria were as follows:

- Age over 18 years
- Ability to provide informed voluntary consent
- Hospitalization for an MI diagnosis confirmed with the Fourth Universal Definition of MI (ESC, 2018) [11], with akinesia and/or hypokinesia in at least two adjacent segments of the LV myocardium according to echocardiography (ECG) obtained within 24 hours after MI
- Hemoglobin (Hb) >90 g/dL and <150 g/dL

The following ID criteria were used: absolute ID was diagnosed in case of a decrease in plasma ferritin <100 $\mu\text{g/L}$, and relative ID was diagnosed in case of plasma ferritin

100–299 $\mu\text{g/L}$ with transferrin saturation coefficient $<20\%$ [12–14].

Written informed consent for participation in the study was obtained from each participant.

The non-inclusion criteria were as follows:

- Hypersensitivity reactions to iron products
- History of blood transfusion or acquired hemosiderosis
- Use of parenteral iron products and/or erythropoiesis-stimulating agents within 3 months prior to the study
- Killip class II–III HF during the hospital stay
- Glomerular filtration rate <15 mL/min/1.73 m² or hemodialysis or peritoneal dialysis at the time of enrollment or in the next 6 months following the start of the study
- Current or recent (in the past month) infectious disease
- Established liver disease or active hepatitis (The International Classification of Diseases, 10th Revision, codes K70–K77)
- Current or recent (within the past 3 years) malignancy
- Active gastrointestinal bleeding
- Pregnancy and lactation
- Inability to attend scheduled visits and maintain contact for the required period of time

On hospitalization day 1, a detailed medical history was obtained from all patients, and a physical examination was performed, as well as blood chemistry and complete blood count to assess iron status, transthoracic ECG to evaluate LV ejection fraction (LVEF), and total myocardial mobility index (TMI) of the LV using a 16-segment model [15]. During hospitalization, patients were examined for clinical status, comorbidities, and chronic HF (according to current guidelines) [14].

All patients received ID treatment with the following iron products: ferrous sulfate at 200 mg/day for 2 months or ferric carboxymaltose for the period of hospital stay. The dose was based on body weight and Hb level according to the prescribing information.

After discharge, the patients were prescribed treatment according to existing CAD guidelines [7]. They were followed for 12 months with treatment adjustments if necessary. The patients underwent repeat ECG to assess LVEF and LV TMI during follow-up visits at 3, 6, and 12 months.

After 3 months, all patients were assessed for iron status and divided into two groups: group 1 included patients with normal iron status 3 months after MI and group 2 included those with persistent ID.

Statistical analysis was conducted using StatTech v.4.2.6 (StatTech LLC, Russia). Quantitative parameters were tested for normality with the Shapiro–Wilk test. Data are presented as arithmetic means and their standard deviations ($M \pm \sigma$) for normal distribution. For non-normal distributions, the results were described using the median and 25% and 75% quartiles ($Me [Q_1; Q_3]$). Means were compared using the Student's t-test for normal distribution and Mann–Whitney U test for non-normal distribution. Nominal data were compared using Pearson's χ^2 test with Yates' correction or Fisher's exact test. $P < 0.05$ indicated significant differences.

The study protocol was approved by the Ethics Committee of the Kazan State Medical University of the Ministry of Health of the Russian Federation (meeting minutes No. 4, dated April 20, 2021).

RESULTS AND DISCUSSION

All patients enrolled in the study were prescribed iron products; 36 (43%) patients received intravenous ferric carboxymaltose and 47 (57%) received ferrous sulfate tablets.

At 3 months, all 36 patients on ferric carboxymaltose showed compensated ID. In 25 (53%) patients on ferrous sulfate, ID was maintained at 3 months. Group 1 included 58 (70%) patients with compensated ID, and group 2 included 25 (30%) patients with persistent ID.

The groups were comparable for sex, age, and most comorbidities. Incidence of hypertension, CAD, previous MI and stroke, chronic HF, chronic obstructive pulmonary disease, chronic kidney disease, previous percutaneous coronary intervention, and coronary artery bypass grafting was assessed in the groups. All patients with atrial fibrillation had a permanent form. More patients with type 2 diabetes mellitus were recorded in the group with persistent ID.

Statistical differences were observed in prevalence of functional class of HF in the two groups. Patients with class II HF predominated in group 1 (18 patients, 81.1%) those with class III HF in group 2 (5 patients, 38.5%; $p = 0.009$). In group 2, 4 (16%) patients had a history of ischemic stroke ($p = 0.007$). Patients with heart valve disease, pacemaker implantation, cardioversion, or pre-hospital or hospital intensive care were excluded.

The clinical characteristics of the patients at enrollment did not differ significantly. The groups were comparable regarding the treatment they received, with the exception of metformin, which was more commonly used by patients in group 2. No cardiovascular events or deaths were reported in either group during the 12-month follow-up.

The key laboratory parameters, including Hb, red blood cell counts, red blood cell size and shape, and C-reactive protein, did not differ between the groups. Alanine aminotransferase was higher in group 1 than in group 2 (30 [19; 43] units/L vs 20 [15; 29] units/L; $p = 0.011$). However, both medians were within the reference range. The median ferritin level in group 1 was higher than that in group 2. Anemia was diagnosed according to the World Health Organization criteria (Hb <130 g/L in men and <120 g/L in women) in 10 (17%) patients in group 1 and 2 (8%) patients in group 2 ($p = 0.3$) [16].

No difference was noted in the incidence of any MI site between the groups. Anterior MI was diagnosed in 30 (52%) patients in group 1 and 16 (64%) in group 2 ($p = 0.08$). Inferior LV wall MI was diagnosed in 22 (38%) patients in group 1 and 9 (36%) patients in group 2 ($p = 0.7$). In group 1, 2 (3%) patients had lateral MI ($p = 0.3$) and 4 (7%) had anterior lateral MI ($p = 0.07$). ECG results on day 1 after MI did not differ between the groups.

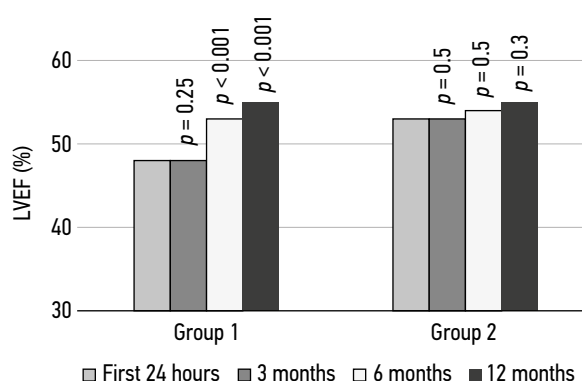


Fig. 1. Left ventricular (LV) ejection fraction (EF) changes in groups during 12 months after myocardial infarction

In the first 24 hours of hospitalization, the median LVEF was 48% [45; 54] in group 1 and 53% [48; 54] in group 2 ($p = 0.07$). In group 1, the median LVEF at 3 months was 48% [46; 54] ($p = 0.25$) and did not differ from the values upon admission. A significant increase was found in LVEF in group 1 according to ECG at 6 and 12 months. The median LVEF at 6 months was 53% [46; 58], significantly higher than the baseline ($p < 0.001$) and 3-month ($p < 0.001$) LVEF results. An increase in LVEF was observed at 12 months compared to 6 months ($p = 0.007$). The median LVEF at 12 months was 55% [48; 58] and remained higher than the baseline ($p < 0.001$).

In group 2, the median LVEF at 3, 6, and 12 months did not differ from baseline and was 53% [46; 57] ($p = 0.75$), 54% [48; 57] ($p = 0.2$), and 55% [48; 58] ($p = 0.3$), respectively (Figure 1).

Ten (17%) patients in the compensated ID group had a 10% increase in LVEF at 12 months compared to the first 24 hours after hospitalization. No increase was noted in LVEF in the persistent ID group ($p = 0.028$). The possibility of an increase in LVEF was 1.5 times higher in the compensated ID group than in the persistent ID group (hazard ratio: 1.5; 95% confidence interval: 1.3; 1.9).

The median LV TMI on day 1 after MI did not differ between the groups and was 1.25 [1.19; 1.62] in group 1 and 1.25 [1.12; 1.56] in group 2 ($p = 0.3$). At 3 months, the median LV TMI in group 1 did not differ from baseline and was 1.25 [1.19; 1.5] ($p = 0.5$). At 6 months, the median LV TMI was 1.19 [1.06; 1.56], which was significantly lower than the baseline ($p < 0.001$) and the results obtained 3 months after MI ($p < 0.001$). A mild decrease in the LV systolic function was observed at 12 months compared to that at 6 months ($p = 0.3$); the median LV systolic function at 12 months was 1.12 [1.0; 1.44], which was significantly lower than the baseline ($p < 0.001$) and that at 3 months ($p < 0.001$), indicating improvement in the LV systolic function after ID compensation.

In group 2, the median LV TMI did not differ from the baseline at 3, 6, and 12 months and was 1.25 [1.03; 1.5] ($p = 0.5$), 1.25 [1.12; 1.5] ($p = 0.5$), and 1.25 [1.0; 1.5] ($p = 0.3$), respectively (Figure 2).

In the present study, normal iron status was reported at 12 months in all patients who received ferric carboxymaltose

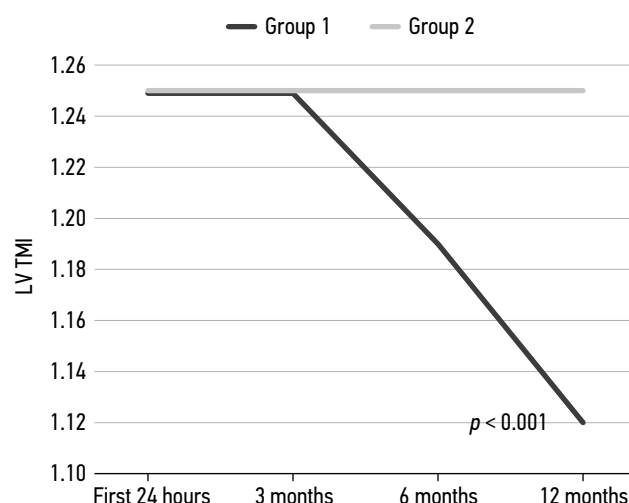


Fig. 2. Change in the total myocardial mobility index (TMI) of the left ventricle (LV) in groups during 12 months after myocardial infarction

for ID treatment, whereas ID was maintained at 12 months in 53% of patients who received ferrous sulfate for 2 months. Randomized studies [13, 14] showed that the intravenous use of ferric carboxymaltose in patients with HF and ID improves quality of life, functional capacity, and prognosis. This is reflected in the Russian guidelines for the treatment of patients with HF, which recommend the use of ferric carboxymaltose for ID treatment in patients with HF and do not recommend the use of oral iron products [15].

The current study revealed that patients with persistent ID and chronic HF initially had higher HF class. The literature indicates that ID is associated with more severe HF, a higher functional class, and a more advanced chronic HF. This association between ID and HF severity may be due to malnutrition, intestinal mucosal swelling, and malabsorption syndrome in patients with severe chronic HF [17].

The high prevalence of diabetes mellitus in the persistent ID group may be because of the known iron metabolism disorders in patients with diabetes mellitus, which are associated with chronic inflammation and increased hepcidin synthesis [18].

The LV systolic function did not change in both groups after 3-month follow-up. However, data showed an improvement in the LV systolic function in patients with compensated ID at 6 and 12 months after MI as evidenced by increased LVEF and decreased LV TMI. LV TMI was associated with a decrease in akinesis/hypokinesis regions shown on ECG. In the persistent ID group, LVEF and LV TMI did not differ from hospitalization day 1 and at 6 and 12 months. In group 1, the significant improvement in LV systolic function at 6 months and lack of change at 3 months may be related to replenishment of iron stores, which, according to the literature, occurs 2–3 months after the start of iron supplementation.

ID treatment was found to be associated with improvement in the LV systolic function within 6 months after MI. However, iron status was only considered at screening, and the degree of ID compensation was not assessed [19]. In this study, the patients were divided based on ID compensation at 3 months, because it is crucial to identify and eliminate ID in patients with MI and determine the iron status over time to assess and achieve ID compensation.

In a study by Huang et al., decreased blood iron was an independent predictor of no increase in LVEF 6 months after coronary balloon angioplasty for MI [9]. The present study showed positive changes in LVEF at 6 months in the compensated ID group and no change in LVEF at 3, 6, and 12 months in the persistent ID group.

Our data are consistent with those of Inserte et al.; ID was associated with adverse LV remodeling risk in patients with ST segment elevation MI after percutaneous coronary intervention [10].

Our data indicate an improvement in myocardial systolic function in patients with ID after MI and percutaneous coronary intervention, provided that ID is compensated. However, because of the small number of studies addressing this issue and inconsistency of data, the effects of ID and possible ID treatments on LV systolic function in patients with MI require further investigation.

The small sample size is a limitation of the study.

CONCLUSION

Compensation for ID is associated with improved left ventricular systolic function, as evidenced by increased LVEF and decreased LV TMI within 12 months after MI.

ADDITIONAL INFORMATION

Authors' contribution. D.R.Kh., N.A.T., I.Kh.V. — methodology, validation, formal analysis, investigation, writing — original draft; N.R.Kh. — conceptualization, formal analysis, writing — review and editing, supervision.

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Competing interests. The authors declare no conflict of interest regarding the presented article.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Д.Р.Х., Н.А.Т., И.Х.В. — методология, валидация, анализ, исследование, создание черновика; Н.Р.Х. — концептуализация, анализ, редактирование рукописи, общее руководство
Источник финансирования. Исследование не имело спонсорской поддержки.

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