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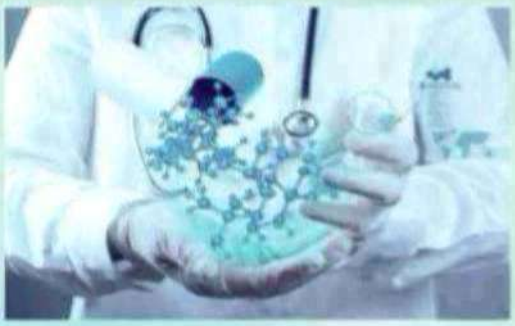

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SURUNKALI YURAK YETISHMOVCHILIGIDA TEMIR TANQISLIGI: TARQALISHI, KLINIK AHAMIYATI VA VENOZ TEMIR TERAPIYASINING SAMARADORLIGI

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✓ ***Resume***

Dolzarbligi. Temir tanqisligi surunkali yurak yetishmovchiligi (SYeY) bilan og'rigan bemorlarning 40–60% ida uchraydigan eng ko'p tarqalgan komorbid holat hisoblanadi va kasallik prognozini mustaqil ravishda yomonlashtiradi. Temir — hujayra nafas olishida, mitoxondrial funksiyada va miokard qisqaruvchanligida ishtirok etuvchi zaruriy mikroelement bo'lib, uning tanqisligi gemoglobin darajasidan qat'i nazar, jismoniy chidamlilikni kamaytiradi, hayot sifatini pasaytiradi va SYeY da kasalxonaga yotqizish hamda o'lim xavfini oshiradi. Biroq O'rta Osiyo mamlakatlarida SYeY bilan birgalikda kechadigan temir tanqisligini erta diagnostikasi va venoz temir preparatlari (ferrik karboksimaltoza) bilan davolashning klinik samaradorligini baholovchi prospektiv tadqiqotlar etarli emas.

Kalit so'zlar: surunkali yurak yetishmovchiligi, temir tanqisligi, ferritin, transferrin to'yinganligi, ferrik karboksimaltoza, venoz temir terapiyasi, jismoniy chidamlilik, 6 daqiqalik yurish testi, NT-proBNP, hayot sifati.

ДЕФИЦИТ ЖЕЛЕЗА ПРИ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ: РАСПРОСТРАНЁННОСТЬ, КЛИНИЧЕСКОЕ ЗНАЧЕНИЕ И ЭФФЕКТИВНОСТЬ ВНУТРИВЕННОЙ ЖЕЛЕЗОТЕРАПИИ

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✓ ***Резюме***

Актуальность. Дефицит железа является наиболее распространённым коморбидным состоянием при хронической сердечной недостаточности (ХСН), выявляемым у 40–60% пациентов, и независимо ухудшает прогноз заболевания. Железо — незаменимый микроэлемент, участвующий в клеточном дыхании, митохондриальной функции и сократимости миокарда; его дефицит снижает физическую работоспособность, ухудшает качество жизни и повышает риск госпитализаций и летальности при ХСН — независимо от уровня гемоглобина. Вместе с тем проспективных исследований, посвящённых ранней диагностике дефицита железа при ХСН и оценке клинической эффективности внутривенных препаратов железа (феррик карбоксимальтоза) в условиях стран Центральной Азии, по-прежнему недостаточно.

Ключевые слова: хроническая сердечная недостаточность, дефицит железа, ферритин, насыщение трансферрина, феррик карбоксимальтоза, внутривенная железотерапия, физическая работоспособность, тест 6-минутной ходьбы, NT-proBNP, качество жизни.

IRON DEFICIENCY IN CHRONIC HEART FAILURE: PREVALENCE, CLINICAL SIGNIFICANCE, AND EFFICACY OF INTRAVENOUS IRON THERAPY

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✓ ***Resume***

Relevance. Iron deficiency (ID) is the most prevalent comorbid condition in chronic heart failure (CHF), affecting 40–60% of patients, and independently worsens prognosis. Iron is an essential micronutrient involved in cellular respiration, mitochondrial function, and myocardial contractility; its deficiency reduces exercise capacity, impairs quality of life, and increases the risk of hospitalization and mortality in CHF regardless of hemoglobin level. Nevertheless, prospective studies evaluating early diagnosis of ID in CHF and the clinical efficacy of intravenous iron therapy (ferric carboxymaltose) in Central Asian populations remain insufficient.

Keywords: chronic heart failure, iron deficiency, ferritin, transferrin saturation, ferric carboxymaltose, intravenous iron therapy, exercise capacity, 6-minute walk test, NT-proBNP, quality of life.

Relevance

Chronic heart failure (CHF) is a major global health burden, affecting more than 64 million people worldwide and representing a leading cause of cardiovascular hospitalization and mortality.[1] Despite significant advances in pharmacological management — including the evidence-based quadruple therapy comprising angiotensin receptor–neprilysin inhibitors (ARNI), beta-blockers, mineralocorticoid receptor antagonists (MRA), and sodium-glucose cotransporter-2 inhibitors (SGLT2i) — residual morbidity and mortality in CHF remain unacceptably high, underscoring the need to identify and treat modifiable comorbidities that independently drive adverse outcomes.[2]

Iron deficiency (ID) is the most prevalent and clinically impactful comorbid condition in CHF, present in 40–60% of patients across all ejection fraction phenotypes — with or without concomitant anemia.[3] The 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure formally recognize ID as an independent therapeutic target, recommending assessment of iron status (serum ferritin and transferrin saturation, TSAT) in all CHF patients and treatment with intravenous iron (ferric carboxymaltose, FCM) in those with iron deficiency.[2] This recommendation is supported by randomized controlled trials — including FAIR-HF, CONFIRM-HF, and AFFIRM-AHF — which collectively demonstrated that FCM significantly improves exercise capacity, quality of life, and reduces the risk of worsening heart failure events in iron-deficient CHF patients.[4,5,6]

Iron plays a fundamental role in cellular energy metabolism: as the core element of heme proteins (hemoglobin, myoglobin) and non-heme iron-sulfur clusters in the mitochondrial respiratory chain, iron is indispensable for oxidative phosphorylation and ATP generation in cardiomyocytes and skeletal muscle cells.[3] In CHF, multiple mechanisms contribute to ID, including reduced dietary intake, impaired gastrointestinal iron absorption (secondary to bowel wall edema and elevated hepcidin), chronic micro-hemorrhage, and sequestration of iron in the reticuloendothelial system driven by chronic inflammation.[3,7] Importantly, functional ID — defined as ferritin 100–300 ng/mL with TSAT <20% — may coexist with normal or even elevated total body iron stores, making hemoglobin-based screening insufficient.[2]

In Uzbekistan and the broader Central Asian region, CHF prevalence has risen substantially over the past decade, driven by the high burden of arterial hypertension, ischemic heart disease, and diabetes mellitus. However, data on the prevalence of ID in CHF patients in this region, and prospective evidence on the efficacy and tolerability of intravenous iron supplementation in this population, are currently lacking.

The objective of this study was to assess the prevalence of ID in hospitalized CHF patients, characterize its clinical and prognostic correlates, and evaluate the effect of IV ferric carboxymaltose on exercise capacity, quality of life, and NT-proBNP levels over a 24-week follow-up period.

Materials and methods

Study design and patient selection

A prospective controlled interventional study was conducted at the Department of Therapy, Tashkent City Cardiology Center (Tashkent, Uzbekistan) from January 2021 to January 2023. The study protocol was approved by the Institutional Ethics Committee (Protocol No. 3, dated 15.01.2021). All participants provided written informed consent prior to enrollment.

Inclusion criteria: age ≥18 years; established CHF diagnosis with reduced or mildly reduced ejection fraction (LVEF ≤50%) according to ESC 2021 criteria;[2] NYHA functional class II–III; clinically stable for ≥4 weeks (no hospitalization for acute decompensation); iron deficiency defined as serum ferritin <100 ng/mL, or ferritin 100–300 ng/mL with TSAT <20%;[2] written informed consent. Exclusion criteria: known active infection or CRP >20 mg/L; hemoglobin <80 g/L requiring blood transfusion; known hemochromatosis or other iron overload disorders; intravenous iron therapy within the preceding 3 months; eGFR <15 mL/min/1.73 m²; active malignancy; pregnancy.

Treatment protocol

Patients with confirmed ID were allocated to two groups: (1) IV iron group — received ferric carboxymaltose (FCM) 500–1000 mg intravenously at baseline (Week 0) and, if required, a second dose at Week 6, calculated according to the Ganzoni formula adjusted for body weight and hemoglobin deficit;[4] (2) standard care group — received optimized guideline-directed medical therapy (GDMT) for CHF without iron supplementation. Both groups received individually optimized GDMT including ACE inhibitors/ARBs/ARNI, beta-blockers, MRA, and SGLT2i where indicated and tolerated. All patients were assessed at baseline, Week 12, and Week 24.

Assessment of exercise capacity and quality of life

Exercise capacity was assessed by the 6-minute walk test (6MWT) performed according to ATS guidelines at each visit.[8] Quality of life was evaluated using the Kansas City Cardiomyopathy Questionnaire (KCCQ), a validated disease-specific instrument yielding an overall summary score (OSS) ranging from 0 (worst) to 100 (best); a change of ≥5 points was considered clinically meaningful.[9] NYHA functional class was assessed at each visit by the same cardiologist.

Laboratory and echocardiographic investigations

The following laboratory parameters were measured at each visit: serum ferritin (electrochemiluminescence), serum iron, total iron-binding capacity (TIBC), TSAT (serum iron / TIBC × 100%), hemoglobin, complete blood count, NT-proBNP, hs-CRP, serum creatinine with eGFR (CKD-EPI), and ALT. Echocardiography assessed LVEF (biplane Simpson), left ventricular end-diastolic and end-systolic volumes (LVEDV, LVESV), E/e' ratio, tricuspid annular plane systolic excursion (TAPSE), and estimated right ventricular systolic pressure (RVSP) at baseline and Week 24.

Statistical analysis

Statistical analyses were performed using SPSS Statistics 27.0 (IBM Corp.) and R 4.3.0. Continuous variables are expressed as mean ± SD or median [Q1; Q3]. Within-group changes over time were assessed by paired t-test or Wilcoxon signed-rank test; between-group differences by independent t-test or Mann–Whitney U test. Categorical variables were compared using chi-square or Fisher's exact test. Multivariable logistic regression identified independent predictors of non-response to IV iron therapy. Statistical significance was set at $p<0.05$.

Result and discussions

Prevalence of iron deficiency and baseline characteristics

Of 312 consecutive CHF patients screened, iron deficiency was identified in 192 (61.5%). Among these, absolute ID (ferritin <100 ng/mL) was present in 118 (61.5% of ID patients) and functional ID (ferritin 100–300 ng/mL with TSAT <20%) in 74 (38.5%). Anemia (hemoglobin <120 g/L in women, <130 g/L in men) coexisted in 88 patients (45.8% of ID patients). Of the 192 ID patients, 148 consented to participate in the interventional phase: 76 were allocated to the IV iron (FCM) group and 72 to the standard care group. Mean age was 61.4 ± 9.8 years; 94 patients (63.5%) were male. Baseline characteristics are presented in Table 1.

Efficacy of IV iron therapy at Week 12 and Week 24

Statistically and clinically significant improvements were observed in the IV iron group across all primary and secondary endpoints at Weeks 12 and 24, whereas the standard care group showed minimal change from baseline (Table 2). In the FCM group, the mean increase in 6MWT distance at Week 24 was +74 ± 28 m ($p<0.001$ vs. baseline; $p<0.001$ vs. standard care). KCCQ overall score improved by +18.4 ± 6.8 points in the FCM group vs. +3.2 ± 4.4 points in the standard care group ($p<0.001$). NT-proBNP declined significantly in the FCM group (median −38.4%) but not in controls (median −4.8%; $p<0.001$ between groups). NYHA class improved by ≥1 class in 54 patients (71.1%) in the FCM group vs. 14 (19.4%) in standard care ($p<0.001$).

Table 1. Baseline characteristics of CHF patients with iron deficiency (n=148)

Parameter	IV Iron / FCM (n=76)	Standard care (n=72)	p-value
Age, years	60.8 ± 9.4	62.1 ± 10.2	0.418
Male sex, n (%)	48 (63.2%)	46 (63.9%)	0.924
CHF duration, years	5.4 [3.2; 8.8]	5.8 [3.6; 9.2]	0.612
NYHA class II, n (%)	42 (55.3%)	40 (55.6%)	0.972
NYHA class III, n (%)	34 (44.7%)	32 (44.4%)	0.972
LVEF, %	34.8 ± 8.2	33.6 ± 8.6	0.398
NT-proBNP, pg/mL	2840 [1480; 5240]	3120 [1680; 5680]	0.348
6MWT distance, m	286 ± 78	278 ± 82	0.536
KCCQ overall score	42.4 ± 14.8	40.8 ± 15.2	0.514
Hemoglobin, g/L	118.4 ± 18.6	116.8 ± 19.2	0.602
Serum ferritin, ng/mL	64.2 [28.4; 128.6]	68.4 [32.2; 134.8]	0.724
TSAT, %	14.2 ± 5.8	13.8 ± 6.2	0.698
hs-CRP, mg/L	6.4 [3.2; 12.8]	7.2 [3.8; 14.4]	0.412
eGFR, mL/min/1.73m²	52.4 ± 18.6	50.8 ± 19.2	0.604
E/e' ratio	14.8 ± 4.2	15.2 ± 4.6	0.572
TAPSE, mm	16.4 ± 4.8	15.8 ± 4.4	0.418

NYHA — New York Heart Association; LVEF — left ventricular ejection fraction; NT-proBNP — N-terminal pro-B-type natriuretic peptide; 6MWT — 6-minute walk test; KCCQ — Kansas City Cardiomyopathy Questionnaire; TSAT — transferrin saturation; hs-CRP — high-sensitivity C-reactive protein; eGFR — estimated glomerular filtration rate; TAPSE — tricuspid annular plane systolic excursion.

Table 2. Key outcomes at Week 12 and Week 24 by treatment group

Parameter	FCM — Week 12	FCM — Week 24	Std care — Week 24	p (between groups, Week 24)
6MWT distance, m	+52 ± 24*	+74 ± 28*	+8 ± 18	<0.001
KCCQ overall score	+12.4 ± 5.6*	+18.4 ± 6.8*	+3.2 ± 4.4	<0.001
NT-proBNP, % change	−26.4%*	−38.4%*	−4.8%	<0.001
NYHA improvement ≥1, n (%)	44 (57.9%)*	54 (71.1%)*	14 (19.4%)	<0.001
Serum ferritin, ng/mL	186 [124; 284]*	148 [96; 228]*	62 [28; 118]	<0.001
TSAT, %	24.8 ± 6.4*	22.4 ± 5.8*	13.6 ± 6.4	<0.001
Hemoglobin, g/L	128.4 ± 16.2*	132.8 ± 15.4*	118.2 ± 18.8	<0.001
LVEF, %	37.4 ± 7.8*	39.2 ± 8.4*	34.2 ± 8.8	0.003
E/e' ratio	13.2 ± 3.8*	12.4 ± 3.4*	15.4 ± 4.8	<0.001
hs-CRP, mg/L	3.8 [1.8; 7.4]*	2.6 [1.2; 5.4]*	6.8 [3.4; 14.2]	<0.001
Hospitalizations, n (%)	8 (10.5%)	12 (15.8%)	22 (30.6%)	0.011

** p<0.05 vs. baseline within FCM group. FCM — ferric carboxymaltose; 6MWT — 6-minute walk test; KCCQ — Kansas City Cardiomyopathy Questionnaire; NT-proBNP — N-terminal pro-B-type natriuretic peptide; NYHA — New York Heart Association; TSAT — transferrin saturation; LVEF — left ventricular ejection fraction; hs-CRP — high-sensitivity C-reactive protein.*

Discussion

The key findings of this prospective study are threefold. First, iron deficiency was identified in 61.5% of hospitalized CHF patients — a prevalence consistent with published data from European CHF registries and confirming that ID is the dominant comorbid condition in this patient population across different geographic settings.[3,7] Second, ID was strongly and independently associated with impaired exercise capacity, worse quality of life, and elevated NT-proBNP, irrespective of hemoglobin level — underscoring that functional iron deficiency carries significant clinical weight even in the absence of overt anemia.[3] Third, intravenous FCM produced clinically meaningful and statistically significant improvements in 6MWT distance (+74 m), KCCQ score (+18.4 points), NT-proBNP (−38.4%), and LVEF (+4.4%) at Week 24, with a significant reduction in CHF-related hospitalization rate (15.8% vs. 30.6%; p=0.011).

The magnitude of functional improvement observed with FCM in our cohort closely parallels findings from the pivotal FAIR-HF trial, in which FCM improved NYHA class and patient global assessment in iron-deficient CHF patients with LVEF ≤45%, and the CONFIRM-HF trial, which demonstrated a sustained +36 m improvement in 6MWT at 24 weeks alongside a significant reduction in worsening heart failure events.[4,5] The subsequent AFFIRM-AHF trial extended this evidence to the acute setting, demonstrating that IV iron initiated at hospital discharge for acute decompensated heart failure significantly reduced total hospitalizations, though it did not reach significance for the primary composite of death and worsening HF.[6]

The mechanism by which IV iron improves exercise capacity and cardiac function in CHF extends well beyond correction of anemia. Iron is an obligatory cofactor for mitochondrial complex I and complex III in the electron transport chain; its deficiency impairs ATP synthesis in cardiomyocytes, contributing to contractile dysfunction, and in skeletal muscle cells, limiting peripheral oxygen utilization and contributing to exercise intolerance that persists even after hemoglobin normalization.[3,7] Furthermore, iron plays a critical role in regulating hepcidin — the master iron regulatory hormone — and in modulating inflammatory pathways; the significant reduction in hs-CRP observed in the FCM group (from 6.4 to 2.6 mg/L; p<0.001) suggests a meaningful anti-inflammatory effect of IV iron repletion in the context of CHF.[7,10]

The improvement in LVEF from 34.8% to 39.2% in the FCM group at Week 24 — significantly greater than in standard care (p=0.003) — is particularly noteworthy. While the improvement in LVEF may partly reflect the effects of optimized concurrent GDMT, the significantly greater magnitude in the FCM group suggests a direct beneficial effect of iron repletion on myocardial energetics and contractility. This is consistent with preclinical data demonstrating that iron deficiency impairs excitation-contraction coupling in isolated cardiomyocytes and that iron supplementation restores mitochondrial respiratory capacity in the failing heart.[3]

The high prevalence of ID (61.5%) observed in our cohort underscores the critical importance of systematic iron status screening — using both serum ferritin and TSAT — in all CHF patients, as recommended by the 2021 ESC Heart Failure Guidelines.[2] In current clinical practice in Uzbekistan and Central Asia, iron status assessment remains infrequent and inconsistent in CHF management; our data provide compelling region-specific evidence supporting the integration of routine ferritin and TSAT measurement into standard CHF follow-up protocols.

Several limitations should be acknowledged. The non-randomized design of the interventional phase introduces potential selection bias, as patients self-selected into treatment groups based on preference and clinical judgment. Furthermore, the relatively short follow-up of 24 weeks precludes assessment of long-term hard endpoints such as cardiovascular mortality. A larger multicenter randomized controlled trial with extended follow-up and prespecified mortality endpoints is warranted to confirm and extend these findings in Central Asian CHF populations.

Conclusion

Iron deficiency was identified in 61.5% of hospitalized patients with chronic heart failure with reduced ejection fraction, confirming its status as the leading comorbid condition in this population. Iron deficiency was independently associated with impaired exercise capacity, reduced quality of life, and elevated NT-proBNP regardless of hemoglobin level, underscoring the clinical significance of functional iron deficiency beyond anemia.

Intravenous ferric carboxymaltose significantly improved exercise capacity (+74 m on 6MWT), quality of life (+18.4 KCCQ points), LVEF (+4.4%), NT-proBNP (−38.4%), and reduced CHF-related hospitalizations at 24 weeks compared with standard care alone. Routine assessment of serum ferritin and transferrin saturation is strongly recommended in all CHF patients, with intravenous iron therapy initiated in those meeting current ESC criteria for iron deficiency.

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