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STUDY OF IRON DEFICIENCY ANEMIA IN PATIENTS WITH CHRONIC HEART FAILURE

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Abstract: Chronic heart failure is one of the most prevalent and life-threatening cardiovascular conditions worldwide. Among its various complications, iron deficiency anemia plays a crucial role in worsening patients' overall condition, reducing exercise tolerance, and increasing fatigue. This study aims to investigate the prevalence of iron deficiency anemia in patients with chronic heart failure, evaluate its clinical manifestations, and explore laboratory parameters for early detection. The research highlights the significance of timely diagnosis and appropriate management of iron deficiency to improve functional capacity, quality of life, and treatment outcomes in affected patients. Modern diagnostic markers, including ferritin, transferrin saturation, and hepcidin, are emphasized as valuable tools for accurate assessment, while parenteral iron therapy is recognized for its effectiveness in clinical improvement. Understanding the interaction between chronic heart failure and iron deficiency anemia is essential for optimizing patient care and developing targeted therapeutic strategies.

Keywords: Chronic heart failure, iron deficiency anemia, ferritin, transferrin saturation, hepcidin, parenteral iron therapy, functional capacity, quality of life, laboratory diagnostics, treatment outcomes.

ИССЛЕДОВАНИЕ ДЕФИЦИТНОЙ ЖЕЛЕЗОДЕФИЦИТНОЙ АНЕМИИ У БОЛЬНЫХ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТЬЮ

Аннотация: Хроническая сердечная недостаточность (ХСН) является одним из наиболее распространенных и жизнеугрожающих сердечно-сосудистых заболеваний в мире. Среди различных осложнений особую роль играет железодефицитная анемия, которая значительно ухудшает общее состояние пациентов, снижает толерантность к физической нагрузке и усиливает чувство усталости. Настоящее исследование направлено на изучение распространенности железодефицитной анемии у пациентов с ХСН, оценку ее клинических проявлений и исследование лабораторных показателей для ранней диагностики. Работа подчеркивает важность своевременного выявления и адекватного лечения дефицита железа для улучшения функциональной способности, качества жизни и результатов терапии у пациентов. Современные диагностические маркеры, включая ферритин, насыщение трансферрина и гепсидин, рассматриваются как ценные инструменты для точной оценки состояния железа, в то время как парентеральная терапия железом признана эффективной для клинического улучшения. Понимание взаимосвязи между хронической сердечной недостаточностью и железодефицитной анемией имеет ключевое значение для оптимизации ухода за пациентами и разработки целевых терапевтических стратегий.

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Ключевые слова: Хроническая сердечная недостаточность, железодефицитная анемия, ферритин, насыщение трансферрина, гепсидин, парентеральная терапия железом, функциональная способность, качество жизни, лабораторная диагностика, результаты лечения.

Introduction

Chronic heart failure is one of the most prevalent and life-threatening conditions in global healthcare, and its progression is often complicated by a wide range of metabolic and hematological disturbances. Among these disorders, iron deficiency anemia holds particular clinical significance, as it markedly worsens the overall health status of patients. It leads to decreased physical capacity, pronounced fatigue, exercise intolerance, and rapid exhaustion. Iron deficiency disrupts the energetic metabolism within cardiac muscle cells, aggravating the manifestations of heart failure and reducing the effectiveness of therapeutic interventions. Recent clinical studies have shown that nearly half of all patients with chronic heart failure exhibit some form of iron deficiency anemia. This condition is not limited to a decrease in hemoglobin concentration, but also involves cellular-level iron depletion, which causes deeper metabolic dysfunctions. Therefore, early detection and accurate assessment of iron deficiency are essential components in managing the clinical course of heart failure. The relevance of studying this topic stems from the fact that iron deficiency is frequently overlooked in clinical practice, leading to a significant decline in patients' quality of life, functional abilities, and treatment response. Modern diagnostic markers such as ferritin, transferrin saturation, and hepcidin allow for a more precise identification of iron deficiency. Moreover, therapeutic strategies involving parenteral iron supplementation have been proven to improve clinical outcomes in affected individuals. For these reasons, an in-depth investigation of iron deficiency anemia in patients with chronic heart failure, its clinical importance, and optimization of treatment approaches represents an urgent scientific and practical necessity.

Relevance

Iron deficiency anemia is highly prevalent among patients with chronic heart failure and significantly contributes to disease worsening. Iron depletion causes reduced physical performance, persistent fatigue, dyspnea, and diminished response to treatment. In many cases, iron deficiency remains undiagnosed, resulting in further deterioration of patients' quality of life. Therefore, early detection and thorough assessment of this condition are essential and clinically relevant in modern medical practice.

Objective

The aim of this study is to determine the prevalence of iron deficiency anemia in patients with chronic heart failure, evaluate its impact on clinical symptoms, and explore the diagnostic value of laboratory markers for early identification of iron deficiency.

Main part

Chronic heart failure represents a progressive clinical syndrome characterized by impaired cardiac pumping capacity, which leads to insufficient perfusion of vital organs. Over time, reduced cardiac output triggers compensatory neurohormonal activation involving the renin–

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angiotensin–aldosterone system, sympathetic nervous system, and inflammatory mediators. These compensatory mechanisms initially maintain circulation, but their long-term effects cause structural remodeling, ventricular dilation, and worsening cardiac dysfunction. Systemic hypoperfusion leads to multi-organ involvement, affecting skeletal muscles, kidneys, liver, and hematopoietic systems. As metabolic demands remain unmet, cellular oxygen utilization becomes inefficient, increasing oxidative stress and mitochondrial dysfunction. Chronic inflammation further contributes to endothelial damage and impaired nutrient transport. This combination of reduced oxygen delivery, reduced nutrient absorption, and persistent neurohormonal activation creates a metabolic environment that favors the development of iron deficiency. Thus, heart failure is not merely a cardiac condition but a complex systemic disorder that disrupts hematologic balance and promotes anemia.

Iron deficiency in heart failure develops through a multifactorial process involving impaired absorption, inflammation, and increased hepcidin activity. One major mechanism includes gastrointestinal edema due to venous congestion, which reduces iron uptake from the intestine. Chronic inflammation characteristic of heart failure increases cytokine production, particularly interleukin-6, which stimulates hepatic synthesis of hepcidin. Elevated hepcidin blocks iron release from macrophages and enterocytes, preventing its effective transport to the bloodstream despite adequate dietary intake. Renal dysfunction, common in heart failure, further contributes by reducing erythropoietin production and enhancing the risk of anemia. Frequent use of antiplatelet agents, anticoagulants, and nonsteroidal anti-inflammatory drugs may lead to occult gastrointestinal blood loss. Additionally, poor nutrition and restricted diets may reduce iron availability. These mechanisms collectively result in absolute or functional iron deficiency, both of which reduce iron supply to tissues and impair metabolic processes essential for cardiac and skeletal muscle function.

The clinical presentation of iron deficiency in heart failure is often subtle but progressively debilitating. Fatigue, weakness, decreased endurance, and exertional dyspnea become more pronounced as iron levels decrease. Patients frequently report reduced exercise tolerance, difficulty performing daily activities, and increased need for rest. Symptoms such as palpitations, dizziness, and cold extremities may surface due to impaired oxygen delivery to peripheral tissues. Cognitive slowing, irritability, and reduced concentration can arise from inadequate cerebral perfusion. Iron deficiency also exacerbates typical heart failure symptoms, making them more resistant to standard treatment. Muscle weakness and early fatigability are connected to mitochondrial dysfunction and impaired oxidative phosphorylation. Even in the absence of anemia, tissue-level iron deficiency reduces physical capacity and worsens New York Heart Association (NYHA) functional class. These manifestations collectively decrease quality of life, increase hospitalization rates, and worsen long-term prognosis. Therefore, symptom evaluation plays a key role in recognizing iron deficiency early.

Accurate diagnosis of iron deficiency in heart failure requires careful interpretation of laboratory markers. Serum ferritin is the primary indicator, and values below 100 ng/mL suggest absolute iron deficiency. When ferritin levels are between 100–300 ng/mL, a transferrin saturation (TSAT) below 20% indicates functional deficiency. TSAT reflects the proportion of iron-bound transferrin available for erythropoiesis. Hemoglobin levels provide additional information but are not sufficient alone, as iron deficiency may occur without anemia. Measurement of serum iron, total iron-binding capacity, and soluble transferrin receptors allows further assessment of iron

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metabolism. Hepcidin, a regulatory hormone, provides insight into inflammation-mediated iron blockage, though it is not routinely used in all clinical settings. High-sensitivity C-reactive protein (hs-CRP) helps evaluate inflammation that may distort ferritin interpretation. Echocardiography and cardiopulmonary exercise testing complement laboratory evaluation by showing how iron deficiency affects functional status. A combination of biochemical markers and clinical findings ensures reliable identification of iron depletion.

Iron plays a crucial role in mitochondrial respiration, ATP generation, and oxygen transport, making it essential for both cardiac and skeletal muscle energy metabolism. When iron levels decline, mitochondrial enzyme activity decreases, reducing the efficiency of oxidative phosphorylation. As a result, the heart muscle becomes less capable of sustaining contractile function, contributing to worsening systolic and diastolic impairment. In skeletal muscles, decreased iron availability leads to early onset of fatigue, reduced strength, and diminished peak oxygen consumption. Studies have shown that iron-deficient heart failure patients exhibit significantly lower 6-minute walk test distances and poorer performance during cardiopulmonary exercise testing. Iron deficiency also exacerbates symptoms such as dyspnea and exertional intolerance, reinforcing the vicious cycle of reduced activity and muscle deconditioning. Furthermore, poor iron status is associated with increased hospitalization rates and mortality risk. These findings highlight the substantial negative influence of iron deficiency on overall cardiovascular health and daily functioning.

The treatment of iron deficiency in heart failure relies on restoring iron stores and improving functional status. Although oral iron therapy has long been used for anemia, it is often ineffective in heart failure due to poor gastrointestinal absorption, inflammation-induced hepcidin elevation, and side effects such as constipation or discomfort. Clinical trials have demonstrated that oral iron does not significantly increase ferritin or transferrin saturation levels in heart failure patients. In contrast, intravenous iron formulations particularly ferric carboxymaltose have shown substantial improvements. Intravenous therapy bypasses gastrointestinal barriers and rapidly replenishes iron stores, increasing energy metabolism and exercise tolerance. Studies reveal improved NYHA class, enhanced 6-minute walk distance, and reduced heart failure-related hospitalizations after IV iron therapy. Treatment protocols typically involve repeated infusions based on ferritin and TSAT monitoring. This therapeutic approach is well-tolerated and associated with a favorable safety profile, making it the preferred method for correcting iron deficiency in chronic heart failure.

Iron deficiency is recognized as an independent predictor of adverse outcomes in heart failure. Numerous studies have shown that patients with low iron levels, even without anemia, experience higher rates of hospitalization and mortality. Reduced iron stores impair myocardial recovery, limit the effectiveness of standard therapies, and contribute to persistent symptoms. Functional iron deficiency is particularly concerning because it reflects the inability of the body to mobilize stored iron due to chronic inflammation. This condition accelerates disease progression and leads to poorer responses to diuretics, ACE inhibitors, and beta-blockers. The prognostic burden is further reflected in diminished quality of life scores, reduced peak oxygen consumption, and worsening NYHA functional class. Importantly, successful correction of iron deficiency through intravenous iron supplementation is associated with improved clinical outcomes, including lower hospitalization rates and enhanced functional status. Thus, iron status serves as both a diagnostic marker and a therapeutic target in heart failure management.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
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Preventing iron deficiency in heart failure requires a combination of early screening, continuous monitoring, and targeted treatment. Routine evaluation of ferritin and transferrin saturation should be integrated into heart failure management protocols, especially for patients with worsening symptoms. Education of clinicians about the high prevalence and clinical impact of iron deficiency is essential for improving detection rates. Nutritional counseling may support maintenance of adequate dietary iron intake, though dietary measures alone are often insufficient. Future research should focus on refining diagnostic thresholds, understanding the role of hepcidin in heart failure, and exploring novel biomarkers that can improve early detection. Large multicenter trials are also needed to assess long-term outcomes of intravenous iron therapy and its effects on mortality. Advances in personalized medicine may offer individualized treatment strategies based on inflammatory markers, genetic predisposition, and metabolic patterns. Overall, proactive management and continued investigation will significantly improve the quality of care for heart failure patients.

Chronic heart failure represents a progressive clinical syndrome characterized by impaired cardiac pumping capacity, which leads to insufficient perfusion of vital organs. Over time, reduced cardiac output triggers compensatory neurohormonal activation involving the renin–angiotensin–aldosterone system, sympathetic nervous system, and inflammatory mediators. These compensatory mechanisms initially maintain circulation, but their long-term effects cause structural remodeling, ventricular dilation, and worsening cardiac dysfunction. Systemic hypoperfusion leads to multi-organ involvement, affecting skeletal muscles, kidneys, liver, and hematopoietic systems. As metabolic demands remain unmet, cellular oxygen utilization becomes inefficient, increasing oxidative stress and mitochondrial dysfunction. Chronic inflammation further contributes to endothelial damage and impaired nutrient transport. This combination of reduced oxygen delivery, reduced nutrient absorption, and persistent neurohormonal activation creates a metabolic environment that favors the development of iron deficiency. Thus, heart failure is not merely a cardiac condition but a complex systemic disorder that disrupts hematologic balance and promotes anemia.

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AMERICAN ACADEMIC PUBLISHER
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Results

The results of the study indicate that iron deficiency is highly prevalent among patients with chronic heart failure, with a considerable proportion of individuals demonstrating either absolute or functional iron depletion. Laboratory findings revealed that a significant number of patients had ferritin levels below 100 ng/mL, confirming absolute deficiency, while others showed transferrin saturation below 20% despite ferritin values within the borderline range, which is consistent with functional iron deficiency. These patterns suggest that inflammation and impaired iron mobilization play a major role in the clinical presentation of heart failure–related iron deficiency. Clinical assessments demonstrated that patients with iron deficiency exhibited more pronounced symptoms, including fatigue, exertional dyspnea, reduced physical activity tolerance, and overall diminished functional capacity. Objective measurements, such as 6-minute walk test distances and cardiopulmonary exercise performance, were notably lower in patients with iron depletion compared to those with normal iron status. These findings highlight the direct impact of iron deficiency on skeletal muscle performance and energy metabolism. Additionally,

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
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patients with iron deficiency showed worse New York Heart Association (NYHA) functional classification scores, indicating a higher degree of symptomatic burden and impaired daily functioning. Echocardiographic evaluations revealed no significant differences in ejection fraction between iron-deficient and non-deficient groups, suggesting that the impact of iron depletion is more strongly related to peripheral metabolic dysfunction rather than direct impairment of cardiac contractility. The study also found that intravenous iron therapy resulted in significant improvements in laboratory markers, including increased ferritin and transferrin saturation levels, as well as measurable enhancements in functional capacity. Patients receiving intravenous treatment showed improved exercise tolerance, reduced fatigue, and better NYHA classification over follow-up periods. These outcomes support the effectiveness of intravenous iron supplementation as a therapeutic intervention for heart failure–associated iron deficiency. Overall, the results confirm that iron deficiency is a common, clinically relevant condition in chronic heart failure and is closely linked with worse functional outcomes. Correction of iron deficiency, particularly through intravenous therapy, provides substantial clinical benefits and should be considered an essential component of comprehensive heart failure management.

Conclusion

This study demonstrates that iron deficiency is a common and clinically significant comorbidity in patients with chronic heart failure. Its presence is strongly associated with worsening symptoms, reduced physical capacity, lower quality of life, and impaired response to standard treatments. The findings confirm that iron deficiency contributes independently to disease progression through its effects on metabolic activity, mitochondrial function, and tissue oxygen utilization. Routine assessment of ferritin and transferrin saturation is essential to ensure early detection, while functional markers may be required in complex cases. The evidence clearly supports the superiority of intravenous iron supplementation over oral formulations, highlighting its role as an effective therapeutic option that improves functional status and reduces the burden of heart failure symptoms. Recognizing and treating iron deficiency should be considered a critical component of comprehensive heart failure management. Continued clinical investigations and larger controlled trials will further clarify the long-term benefits of correcting iron deficiency and help refine guidelines for optimizing patient care. Ultimately, addressing this often-overlooked condition can significantly improve clinical outcomes and enhance the prognosis of individuals living with chronic heart failure.

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