IJACT, Volume 3, Issue 1, 2025 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT

INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Modern Methods for Diagnosing the Impact of Ischemic Heart Disease on the Pathogenesis of Anemia

Uzoqova Oyjamol Narzullayevna
Samarkand State Medical University, Assistant Professor, Department of Hematology

Jalolov Sohib Aslitdinovich, O'sarov Shohruh Abduvahob o‘g‘li
Therapeutists of the Samarkand branch of the Republican Scientific Center for Emergency Medical Care

Abstract: Purpose of the review: To analyze modern views on the problem of anemia in chronic heart failure (CHF), the main pathogenetic mechanisms of its formation, and the possibilities of drug correction.

Basic principles. The problem of CHF has a number of interdisciplinary aspects. One of the frequent pathologies in CHF is anemia, which exacerbates hemodynamic disorders and worsens the prognosis of patients. Understanding the mechanisms of anemia development in CHF is of great importance in choosing treatment tactics. The review discusses views on the pathogenetic mechanisms of the formation of anemia syndrome in patients with CHF. Approaches to its treatment are considered from the point of view of pathogenesis.

Conclusion: Anemia in CHF is associated with an increased risk of mortality, worsening of comorbidities, and increased functional class of heart failure, but is a potentially reversible condition.

Keywords: chronic heart failure, anemia, cardiorenal anemia syndrome.

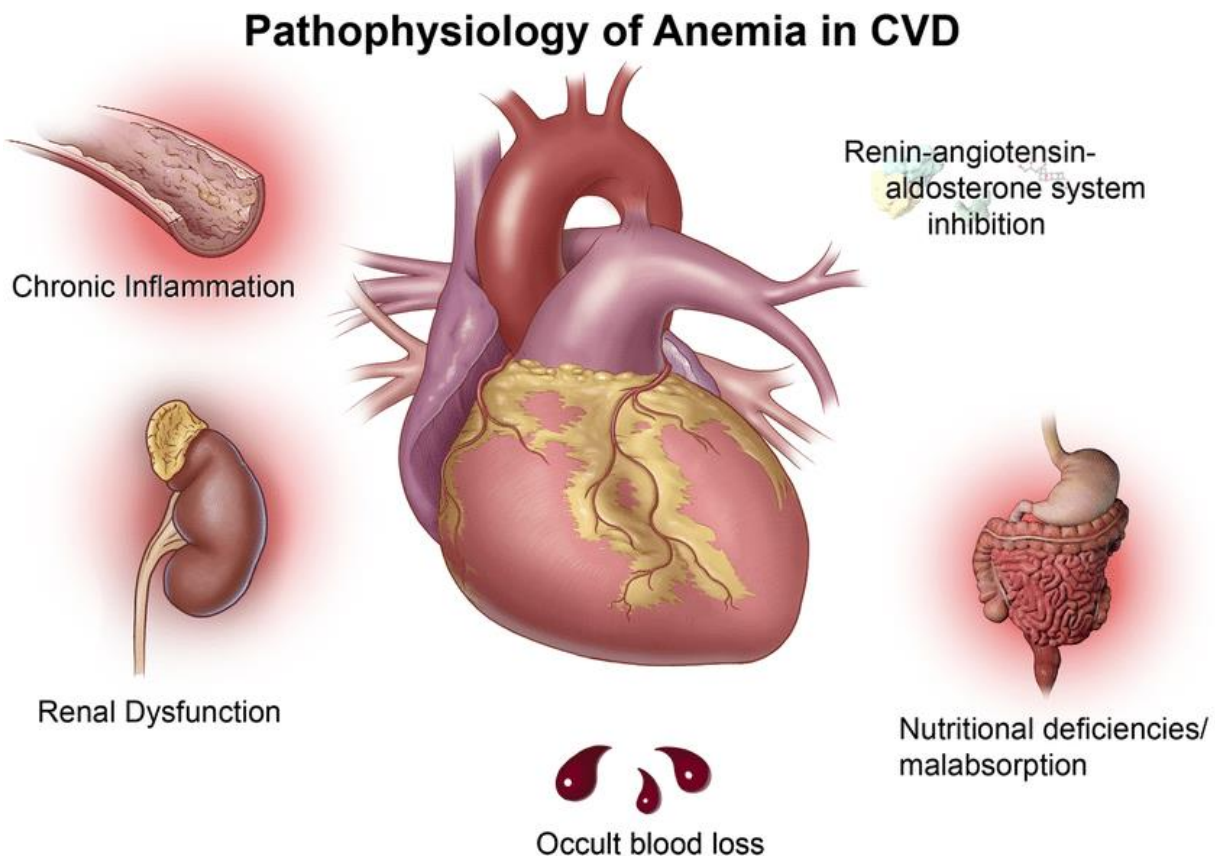
Introduction

The number of people with chronic heart failure (CHF) has been steadily increasing in recent decades [1]. This is due to the increasing proportion of elderly people in the population, as well as the increased survival rate of patients with cardiovascular diseases due to improved medical care [1].

CHF is not just a heart problem, this disease has a number of interdisciplinary aspects; therefore, timely diagnosis and correction of conditions that aggravate the severity of heart failure are an important task in the treatment of such patients. One of them is anemia.

The purpose of this review is to analyze modern views on the problem of anemia in chronic heart failure, the main pathogenetic mechanisms of its formation, and the possibilities of drug correction.

Anemia syndrome is very common in patients with CHF. According to many clinical studies (SOLVD, ELITE II, ValHeFT, COPERNICUS, COMET), anemia occurs in 7-79% of people with heart failure [2]. The significant variation in its prevalence is explained by the lack of a single approach to the diagnosis of anemia, the heterogeneity of their causes, differences in the severity of CHF, demographic data of patients, as well as comorbidities of the studied subjects. . The prevalence of anemia has been noted to increase with the severity of heart failure [3, 4]. It is more often detected in elderly patients [5]. There is evidence of a higher incidence of anemia in women with CHF [6]. It is noted that among young people it develops more often in women, and in those over 85 years of age - in men. Thus, in the age category over 85 years of age, anemia is noted in 27-40% of cases in men and only in 16-21% of women [7].



Researchers recognize the negative contribution of anemia to the clinical picture, course and rate of progression of CHF and even consider it to be an independent predictor of mortality. The clinical picture of CHF patients with anemia is characterized by worsening of systolic and diastolic dysfunction of the heart, worsening of the functional class (FC) of CHF, a rapid decline in renal function, worsening of quality of life and heart failure. low BMI [8, 9]. However, some authors still cannot find a reliable relationship between anemia and the state of ejection fraction of the heart [10].

The Framingham study was one of the first to demonstrate that anemia is a significant risk factor in people with CHF [11]. And the results of the SOLVD study showed a negative inverse relationship between hematocrit level and mortality in CHF. Thus, at 33 months of follow-up, the mortality of patients was 22%, 27% and 34% for hematocrits of 40-44%, 35-39% and less than 35%, respectively [12].

The work of D. Silverberg noted that with a decrease in hematocrit by 1%, the risk of death in patients with CHF class III-IV increases by 11% [13]. According to 3-year follow-up by Italian researchers, mortality from cardiac causes in people with CHF and anemia exceeded that in patients without anemia and often led to the development of severe coronary events (39% and 27%, respectively) [14]. A relationship was found between the presence of anemia in patients with CHF, the frequency of hospitalization for its decompensation, and the cost of treatment. According to the results of an analysis of medical records of 91,316 people hospitalized for decompensated heart failure, anemia was a stronger predictor of the need for surgical repair of the coronary arteries or early readmission than for ischemic heart disease with arterial hypertension [15].

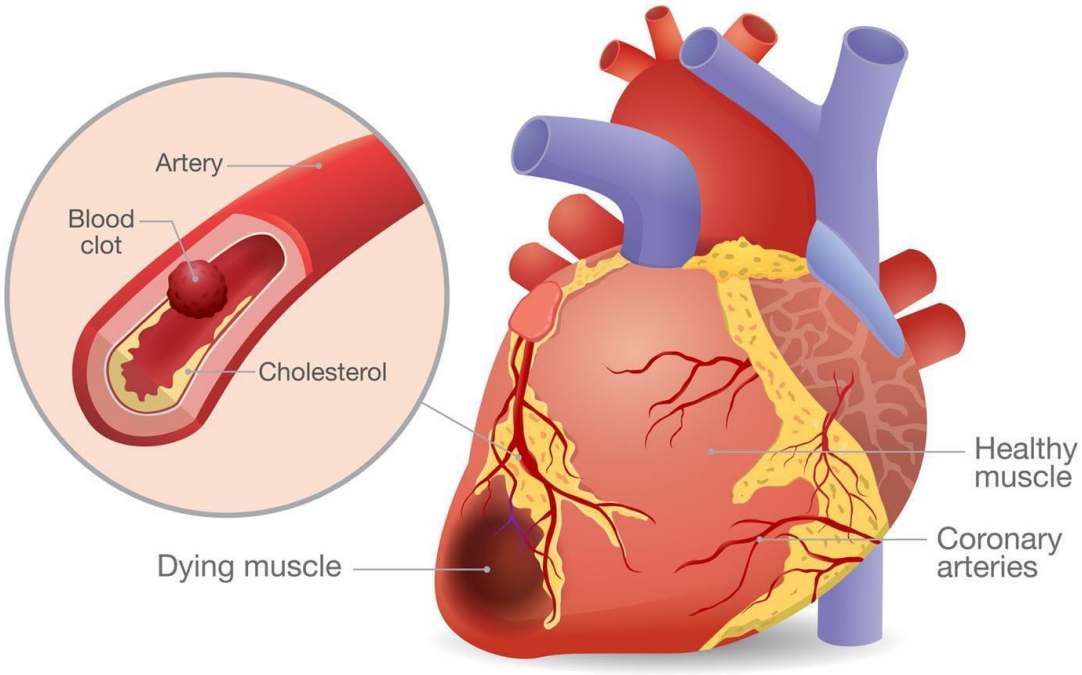
Most patients with CHF are characterized by mild anemia. Given the unity of some pathogenetic mechanisms of anemia and CHF, it is advisable to assume the development of anemia with an increase in the degree of CHF decompensation. This pattern has been shown in a number of studies. At the same time, anemia rarely reaches a moderate or severe degree, more often we are talking about a greater prevalence of mild anemia with increasing severity of CHF and FC.

According to the literature, this is a pathogenetic anemia that negatively affects the development of heart failure. The PR Karla study (n = 552), which included patients with recent-onset heart failure and

not yet anemic, showed that the prognosis of the disease was not dependent on the decrease in hemoglobin [17].

To date, the pathogenesis of anemia syndrome in patients with CHF is not fully understood. The mechanisms of anemia development include impaired renal function, hemodilution, the influence of iatrogenic factors (angiotensin-converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), beta-blockers (BABs), acetylsalicylic acid), proinflammatory cytokines, and malabsorption syndrome. In addition, when the pumping function of the heart is impaired, there is a direct inhibition of bone marrow function due to its hypoperfusion [10].

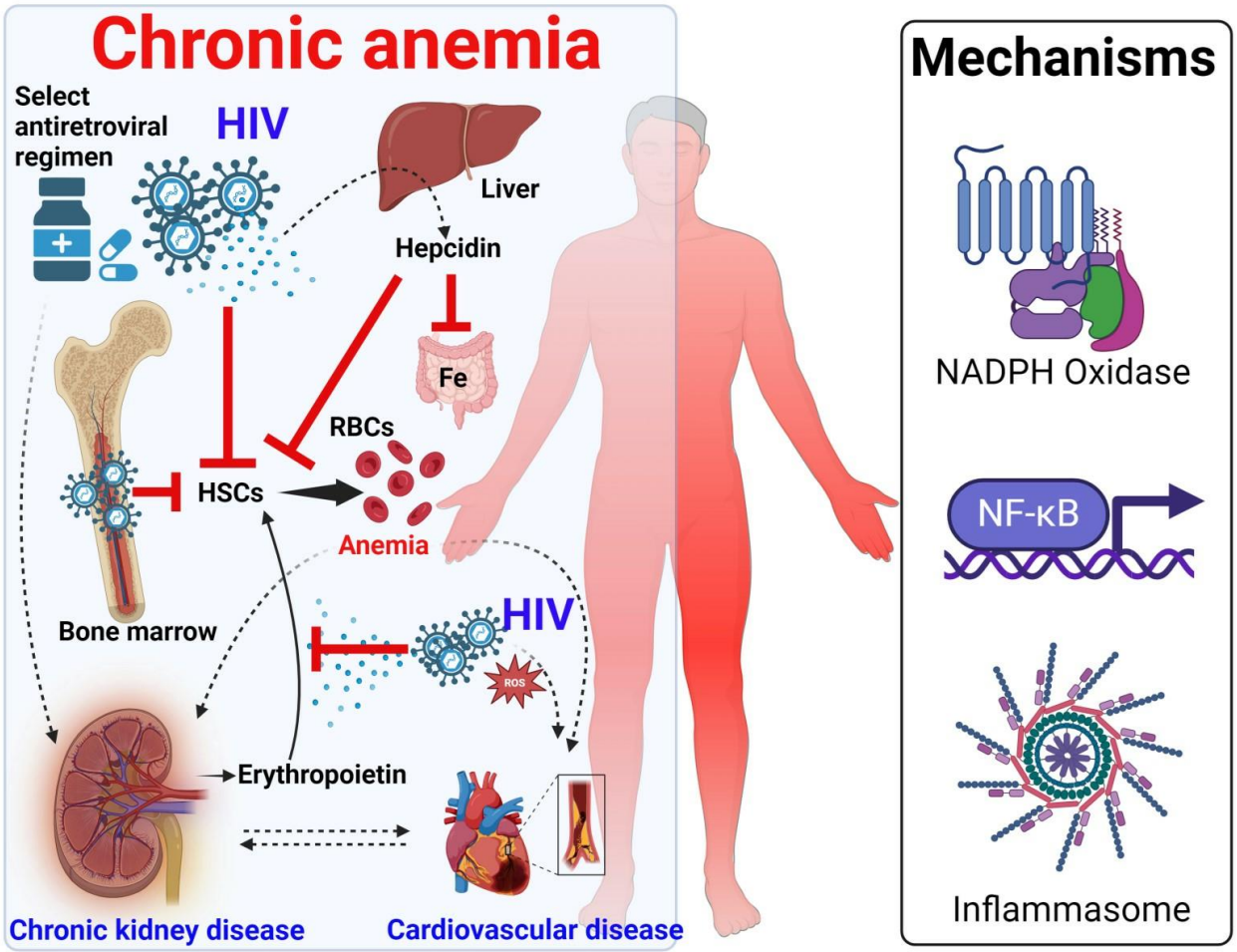
The mechanisms of hemoglobin depletion in patients with CHF can be divided into two categories: those leading to anemia of chronic disease (ACD) (cardiorenal anemia syndrome, effects of pro-inflammatory cytokines) and those contributing to the development of iron deficiency (drug exposure, malabsorption syndrome, cardiac cachexia). The proportion of these mechanisms is different in patients with CHF.



When considering the causes of anemia syndrome in patients with CHF, the authors note a different structure of anemia. A large Canadian study on the epidemiology of anemia syndrome in CHF (n = 12,065) showed a prevalence of iron deficiency anemia (IDA) - 58%. In 27% of cases, B12 deficiency anemia was detected, in 8% - folate deficiency, and only in 7% of cases, signs of ACD without iron deficiency were detected [18].

J. Ezekovitz et al. reported iron deficiency as the cause of anemia in 21% of patients, and deficiency of other hematopoietic factors in 8%; ACD and other established forms of anemia were included in the diagnosis in 58% and 13% of patients, respectively [18].

According to domestic and Ukrainian scientists, 24-40% of patients had iron deficiency anemia, 4-7% had B12 deficiency, and 4-11% had other specific causes of anemia. In 46-69% of patients, anemia was not detected, but met the criteria for ACD [5, 19]. According to GP. Arutyunov, with CHF, approximately 50% of anemia can be attributed to AKD [20].



ACD is most often normochromic normocytic, less often moderately hypochromic or hypochromic-normocytic. The level of reticulocytes is normal or reduced. The bone marrow is characterized by a normal or reduced number of erythrokaryocytes, an abundance of macrophages and sideroblasts with hemosiderin inclusions. In addition, there is an average or normal concentration of iron in the blood serum (10-18 mg / L), a decrease in the total iron-binding capacity of the blood serum, transferrin, transferrin iron saturation (<20%), and an increased or normal level of ferritin (40-300 μg / L).), that is, there is a redistributive (functional) iron deficiency. In other words, in ACD, the parameters of iron metabolism can vary within a very wide range [21].

According to the literature, in patients with ACD, hepcidin levels are often elevated [21]. Hepcidin is an acute-phase protein with the properties of a universal humoral negative regulator of iron metabolism in the body, blocking the expression of the ferroportin protein on the membrane of iron-storing cells (enterocytes, hepatocytes, macrophages) [22]. Ferroportin is responsible for the transport of iron from cells into the blood, a decrease in its content reduces the release of iron from the depot and the development of hypoferremia. Hepcidin synthesis is increased during inflammation and in conditions of iron overload. Thus, the overall biological effect of hepcidin in ACD is aimed at reducing iron absorption in the small intestine and reducing iron content due to its sequestration in macrophages and hepatocytes [23].

IDA is characterized by microcytic hypochromic features, decreased iron metabolites (serum iron ≤ 10 μmol/l, serum ferritin ≤ 14 μg/l), increased transferrin levels, decreased transferrin iron saturation, and increased serum concentration of soluble transferrin receptors. Hepcidin levels in IDA are often reduced to 20–25 pg/ml [24].

Patients with CHF often have a combination of ACD and IDA. In such patients, there is a moderate decrease in serum iron, transferrin and transferrin saturation, serum ferritin levels are moderately reduced or within normal limits, and the concentration of soluble receptors is moderately increased or

normal. Differential diagnosis of these conditions is of fundamental practical importance: it determines adequate therapeutic tactics and allows avoiding the appointment of iron supplements in patients at risk of iron overload and secondary hemosiderosis [25].

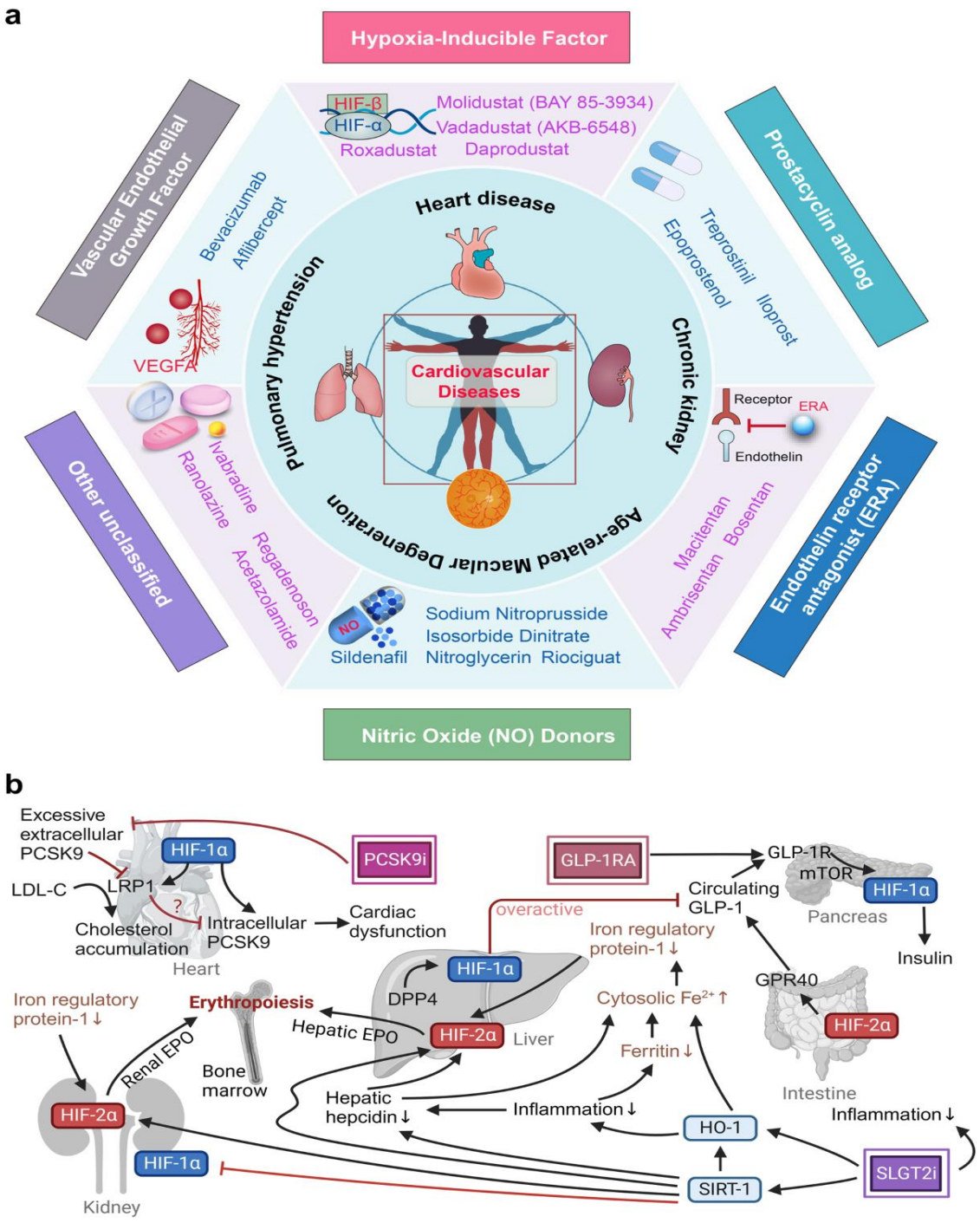
According to various sources, iron deficiency is detected in 5-42% of patients with CHF [2-7]. Pathophysiological conditions for this include malabsorption syndrome, cardiac cachexia associated with compensatory physical inactivity and malnutrition, as well as occult gastrointestinal bleeding (GIB) caused by taking acetylsalicylic acid and even proteinuria [16].

In patients with CHF and anemia, absolute and relative (functional, redistributive) iron deficiency occurs. With functional deficiency, iron is not available for erythropoiesis even at normal levels, since it is located in the macrophage depot; This condition is typical for ACD [2]. As the disease progresses, bone marrow cells continue to use iron for their own needs. As a result, the plasma iron pool is depleted, which leads to hypoferremia against the background of impaired absorption in enterocytes [26]. This explains the widespread combination of ACD and IDA with increasing severity of anemia in CHF, and the emergence of a category of patients with isolated IDA [3, 27].

Methodology

The methodology of this study centers around a comprehensive review of existing literature regarding the impact of ischemic heart disease on the development of anemia, particularly in the context of chronic heart failure (CHF). Initially, the authors conducted an extensive search of peer-reviewed articles, clinical studies, and reviews published in the past decade, using prominent medical databases. The focus was on understanding the pathophysiological mechanisms underlying the development of anemia in CHF patients and identifying potential pharmacological interventions. Criteria for inclusion in the review included studies that addressed both the diagnostic challenges and therapeutic strategies for managing anemia in CHF. Emphasis was placed on research that examined the relationship between hemodynamic disturbances, impaired renal function, and the exacerbation of anemia within CHF. A particular interest was given to studies that explore cardiorenal anemia syndrome, given its relevance to the topic. Data were synthesized and analyzed to compare the efficacy of different treatment modalities, including both drug therapy and non-pharmacological approaches. The review aimed to highlight areas where further research is needed, especially in terms of targeted drug therapies that address the underlying mechanisms of anemia in CHF patients. Additionally, the study considered the clinical outcomes associated with anemia correction in CHF, assessing its impact on patient prognosis and quality of life. The overall aim was to provide a thorough overview of modern diagnostic methods and treatment options, which could contribute to improved patient outcomes in this growing patient population.

Results and Discussion



In addition, there is a group of CHF patients with reduced serum iron but normal hemoglobin levels (latent iron deficiency). According to the literature, this group accounts for approximately 32% [28]. Multivariate regression studies have shown that patients with normal hemoglobin levels but reduced serum iron levels have a lower quality of life on the HRQoL scale (Health-Related Quality of Life scale) than a comparable group of patients without iron deficiency [29]. Reduced serum iron in the absence of anemia is an independent factor determining the level of submaximal workload in patients with CHF [30]. Thus, a normal hemoglobin level does not exclude iron deficiency, which should be identified in all patients with CHF for timely correction of medication;

It is known that renal dysfunction often develops in CHF. Currently, much attention is paid to the contribution of kidney damage as a target organ in CHF to the development of anemia [31]. Understanding the strong interrelationship between CHF, renal failure and anemia allows us to combine all three conditions into the concept of cardiorenal anemia syndrome proposed by DS

Silverberg et al. in 2003 [13, 31]. Heart failure contributes to the development of renal dysfunction, primarily due to a decrease in ejection fraction and deterioration of renal tissue perfusion, which is subsequently associated with the development of anemia [32]. Increased ischemia of endothelial cells and fibroblasts of peritubular capillaries localized in the tubulointerstitium leads to their fibrosis and a decrease in the synthesis of erythropoietin (EPO) [3, 33]. However, data have been presented that in patients with CHF, increased EPO synthesis is considered as a renal response to hypoxia, and the existing anemia syndrome is considered as a resistance to EPO [20].

In patients with CHF, the functional reserve of the kidneys decreases long before the contractile function of the heart decreases. In the early stages of heart failure, the kidneys are unable to maintain an adequate balance when sodium levels increase. Then renal perfusion and glomerular filtration rate decrease, and then severe renal dysfunction develops in parallel with the development of heart failure [14]. Today, there is an opinion that the state of the kidneys can be a sensitive marker of cardiac function, and anemia is an indicator of renal function in patients with CHF [2, 14].

One possible cause of anemia is the synthesis of large amounts of pathological pro-inflammatory cytokines (IL-1 and -6, tumor necrosis factor, C-reactive protein, fibrinogen) in CHF, which leads to ACD [15].

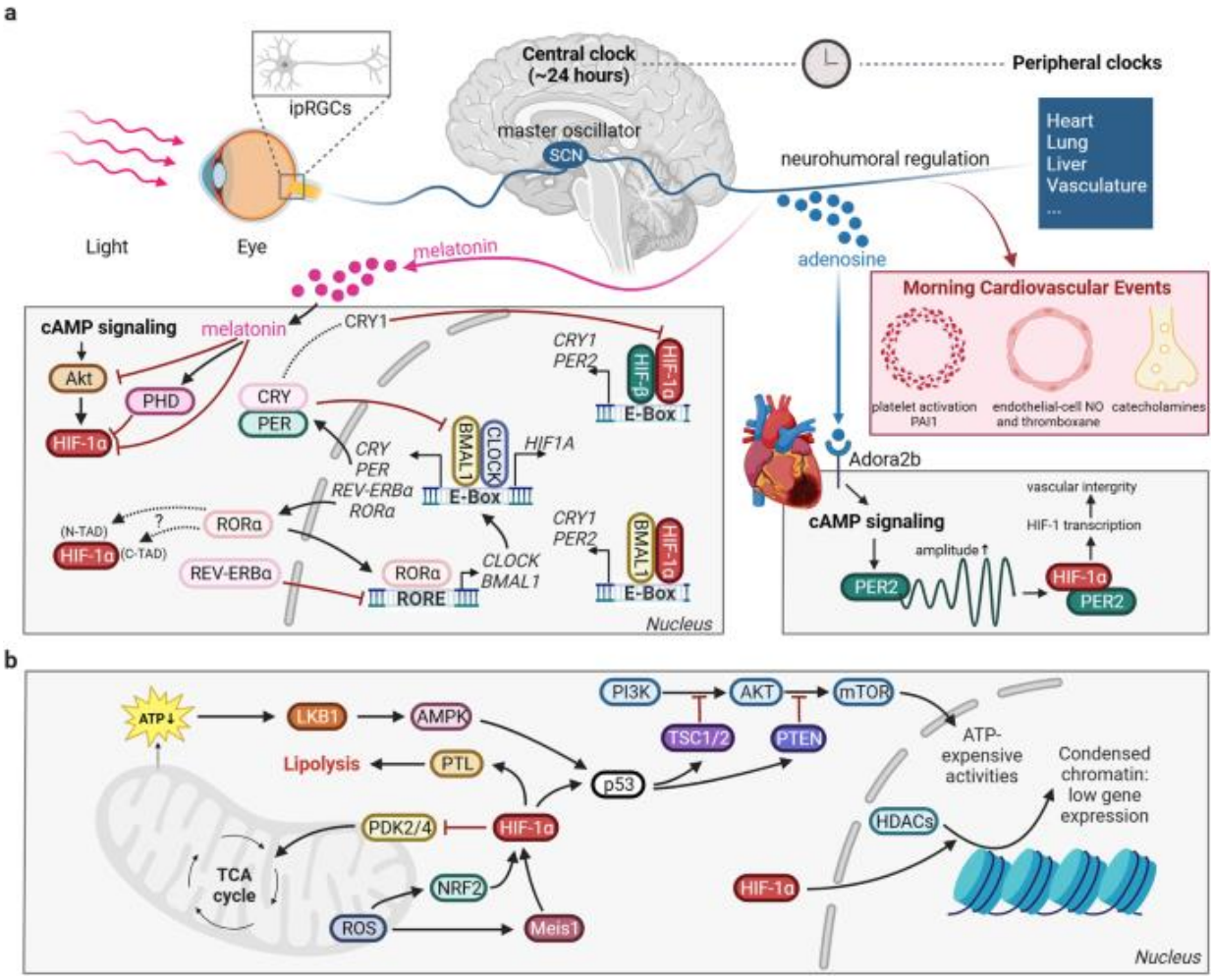
ACD in CHF is considered by a number of authors as an inflammatory autoimmune process with immunological disorders that are one of the causative agents of the disease or occur against the background of severe atheromatous destructive damage to the vascular endothelium. The role of infection (cytomegalovirus, herpes simplex virus, Helicobacter pylori, Chlamydia pneumoniae) in the development of atherosclerosis and the formation of CHF is assumed [16]. In general, inflammation and the resulting cytokine cascade may play an important role in the development of anemia syndrome in CHF, but have not yet been sufficiently studied. The true contribution of proinflammatory cytokines and hepcidin to the development of ACD in patients with CHF needs to be assessed. There are no studies on the role of cytokine aggression and hepcidin in the pathogenesis of cardiorenal anemia syndrome. Hepcidin may become a marker that determines the tactics of management and treatment of these patients.

Inhibition of erythropoiesis plays a special role in the treatment of CHF with drugs: ACE inhibitors and ARBs. It is known that the renin-angiotensin system plays an important role in the regulation of red blood cell count and plasma volume [37]. An increase in the concentration of angiotensin II (AT-II) in the blood plasma leads to a decrease in the peritubular partial pressure of oxygen in renal cortical fibroblasts. This contributes to an increase in the concentration of intracellular reactive oxygen species, which activates the hypoxia-inducible factor HIF-1, increasing the expression of the EPO gene. A direct stimulating effect of AT-II on the erythroid lineage of the bone marrow has also been noted [38]. The contribution of ACE inhibitors to the development of anemia was demonstrated in a study by A. Ishani et al. They confirmed that in patients with CHF with initially normal hematocrit values, taking enalapril for a year, the incidence of anemia increased. However, the survival rate of patients taking enalapril was higher even with the development of anemia, so despite their ability to cause anemia, ACEIs remain first-line drugs in the treatment of CHF [19].

Prophylactic use of acetylsalicylic acid is one of the pathophysiological conditions for the formation of iron deficiency and the subsequent development of anemia in patients with CHF. Gastrointestinal diseases, which can lead to gastrointestinal diseases, are present in 18.5-62% of patients with CHF, and the development of gastrointestinal diseases in CHF is closely related to the presence of chronic pathology of the gastrointestinal tract and, as a result, decompensation. diabetes mellitus or myocardial infarction [10].

It is impossible not to note the data on the effect of beta-blockers on erythropoiesis. As noted in the work of V.Yu. Akhmatova et al. [11], as shown in the COMET (Carvedilol or Metoprolol European Trial) study, a significant decrease in hemoglobin levels with the use of carvedilol was explained by the blockade of β 2 - adrenergic receptors of erythroid cells and β 2 - adrenergic adrenergic receptors of the apparatus cells, which led to a decrease in EPO production.

Along with a decrease in the number of red blood cells (true anemia), patients with CHF often have hemodilutional (relative) anemia, in which a decrease in hemoglobin and hematocrit occurs due to an increase in plasma volume.



There is evidence of an increase in plasma volume in all patients with systolic heart failure and in 71% of patients with diastolic heart failure. At the same time, 59% and 88% of patients with anemia had a true deficiency of erythrocytes in systolic and diastolic CHF, respectively, while the remaining patients had hemodilutional anemia [4]; in studies by AS Androne et al. true anemia was detected only in 54% of patients with CHF. It is important that the survival rate of the group of patients with hemodilution was worse. Interestingly, the hematocrit level in true anemia was significantly lower than in relative anemia [4,13].

It is known that in many patients with CHF, the absorption of nutrients (proteins, fats, carbohydrates and trace elements, including vitamins and iron) in the intestine is significantly impaired due to severe hemodynamic changes, which leads to a decrease in their secretory and absorptive activity. digestive tract. Therefore, with an equivalent energy value and physical activity in patients with CHF, the "bioavailability" of food calories is significantly reduced (by 40% or more) compared with people without CHF, which leads to a negative energy and nitrogen balance [4].]. Iron and protein deficiency due to malabsorption also aggravates anemia.

In patients with CHF and chronic kidney disease, a violation of the trophic state, manifested in the form of a decrease in the level of markers of protein metabolism, which is associated with the severity of anemia, has been proven. Studies have found a pattern: the greater the proportion of protein loss in the small intestine, the lower the hemoglobin level. Thus, in patients with hemoglobin less than 100 g / l, protein loss was 2 times higher than in patients with hemoglobin 120-130 g / l, which also indicates the important role of malabsorption in the formation of anemia syndrome [7].

Given the importance of the negative contribution of anemia to the prognosis of CHF described in this review, the need for its correction is undeniable. However, at present there is no single strategy for the treatment of anemia in patients with CHF. The diversity of etiopathogenetic mechanisms of the formation of anemia syndrome in these patients makes it difficult to choose treatment tactics. Existing approaches to the treatment of anemia syndrome in CHF are more related to ACD and IDA, as well as their combination.

The goals of drug correction of anemia syndrome in patients with CHF are to reduce the risk of fatal events, improve prognosis, reduce the need for diuretics, reduce the frequency of hospitalizations, and improve quality of life [12].

Currently, iron preparations (mainly intravenous), EPO preparations, and their combinations are considered as possible methods of treating anemia in patients with CHF. It is also worth remembering about the standard therapy for CHF, which is to level hemodilution to varying degrees and increase hematocrit [5].

When using iron supplements, it is necessary to monitor hemoglobin levels and ferrokinetics to prevent iron overload and iatrogenic hemosiderosis. The dosage of drugs and the duration of their use are individual and depend on the degree of iron deficiency and the severity of anemia. In general, positive experience has been gained in the treatment of anemia syndrome with iron preparations in the complex therapy of CHF. In such patients, hemoglobin levels increased, cardiac pumping function improved, exercise tolerance and survival increased [2, 11].

The effectiveness of treatment with EPO drugs has been confirmed by a sufficiently large clinical experience. In addition, it is known that EPO not only stimulates hematopoiesis, but also has a direct cardioprotective effect, leading to a decrease in the amount of peroxide apoptosis, myocardial necrosis, and helps protect cardiomyocytes from ischemic damage [14]. However, in routine clinical practice, the use of EPO in patients with CHF is very limited. This is primarily due to the increased risk of acute cardiovascular events and death in patients with end-stage cardiovascular disease with an increase in hematocrit of more than 35% [15]. Limiting factors are also high cost and long duration of treatment (12 months or more). As a rule, drugs of this group are prescribed to patients with resistance to iron therapy and the normochromic and normocytic nature of the anemia syndrome, i.e., with ACD, FC III-IV CHF.

CONCLUSION

Summarizing the above, we can say that anemia in chronic heart failure (CHF) is widespread and has significant clinical and prognostic significance. At the same time, the pathogenetic mechanisms of its development are diverse and are not yet fully understood, and the ways of drug correction of anemia syndrome in CHF have not been clearly defined; All this determines the relevance of research in this area.

List of used literature:

1. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS et al. ESC recommendations for the diagnosis and treatment of acute and chronic heart failure 2016. Ross. cardiol. zhurn. 2017; 1(141): 7–81. [Ponikowski P., Voors AA, Anker SD, Bueno H., Cleland JGF, Coats AJS et al. ESC recommendations for the diagnosis and treatment of acute and chronic heart failure 2016. Ros. cardiol. zhurn. 2017; 1(141): 7–81. (in Russian)]
2. Solomakhin NI, Nakhodnova ES, Ershov VI Anemia in chronic heart failure: the role of hepcidin as a universal regulator of iron metabolism. Journal Heart. Failure. 2014; 85(4): 254–60. Solomakhin NI, Nakhodnova ES, Ershov VI Anemia in chronic heart failure: the role of hepcidin as a universal regulator of iron metabolism. Journal Heart. Insufficient. 2014; 85(4): 254–60. (in Russian)]

3. Marazia S., Loderini S., Magliari F., Sarullo FM, Di Mauro M. Sucrose iron supplementation may be a useful adjunct in patients with heart failure and anemia. In: Proceedings of the 5th International Multidisciplinary Course on Iron Anemia. Florence, Italy. 31 March - 1 April 2017.
4. Zohidova KH Selection of optimal therapy for the correction of anemia syndrome in patients with chronic heart failure. Cardiology. 2018; 58(1): 25–31. [Zohidova KX Vybor optimal'noi terapii pri korrektsii anemicheskogo sindrom u bol'nykh s khronicheskoi serdechnoi nedostatochnost'yu. Cardiology. 2018; 58(1): 25–31. (in Russian)]
5. Stuklov NI Iron deficiency and anemia in patients with chronic heart failure. Diet. Pharmacotherapy in cardiology. 2017; 13(5): 651–60. [Stuklov NI. Ratio. Pharmacotherapy in cardiology. 2017; 13(5): 651–60. (in Russian)]
6. Rudakova LE, Belyaeva Yu.B., Fatkabrarova AM, Kozina LE Anemia in chronic heart failure (according to the department of cardiology). theory. and buttocks. modern aspects of science. 2014; 6(2): 128–30. [Rudakova LE, Belyaeva Yu.B., Fatkabrarova AM, Kozina LE. Theory. I am an example. aspekti sovrem. science 2014; 6(2): 128–30. (in Russian)]
7. Andryev S. et al. Experience with the use of memantine in the treatment of cognitive disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 282-288.
8. Antsiborov S. et al. Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases //Science and innovation. – 2023. – T. 2. – №. D11. – C. 29-35.
9. Asanova R. et al. Features of the treatment of patients with mental disorders and cardiovascular pathology //Science and innovation. – 2023. – T. 2. – №. D12. – C. 545-550.
10. Begbudiyev M. et al. Integration of psychiatric care into primary care //Science and innovation. – 2023. – T. 2. – №. D12. – C. 551-557.
11. Bo'Riyev B. et al. Features of clinical and psychopathological examination of young children //Science and innovation. – 2023. – T. 2. – №. D12. – C. 558-563.
12. Borisova Y. et al. Concomitant mental disorders and social functioning of adults with high-functioning autism/asperger syndrome //Science and innovation. – 2023. – T. 2. – №. D11. – C. 36-41.
13. Ivanovich U. A. et al. Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 409-414.
14. Nikolaevich R. A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 898-903.
15. Novikov A. et al. Alcohol dependence and manifestation of autoagressive behavior in patients of different types //Science and innovation. – 2023. – T. 2. – №. D11. – C. 413-419.
16. Pachulia Y. et al. Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 240-244.
17. Pachulia Y. et al. Neurobiological indicators of clinical status and prognosis of therapeutic response in patients with paroxysmal schizophrenia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 385-391.
18. Pogosov A. et al. Multidisciplinary approach to the rehabilitation of patients with somatized personality development //Science and innovation. – 2023. – T. 2. – №. D12. – C. 245-251.
19. Pogosov A. et al. Rational choice of pharmacotherapy for senile dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 230-235.

20. Pogosov S. et al. Gnostic disorders and their compensation in neuropsychological syndrome of vascular cognitive disorders in old age //Science and innovation. – 2023. – T. 2. – №. D12. – C. 258-264.

21. Pogosov S. et al. Prevention of adolescent drug abuse and prevention of yatrogenia during prophylaxis //Science and innovation. – 2023. – T. 2. – №. D12. – C. 392-397.

22. Pogosov S. et al. Psychogenetic properties of drug patients as risk factors for the formation of addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 186-191.

23. Prostyakova N. et al. Changes in the postpsychotic period after acute polymorphic disorder //Science and innovation. – 2023. – T. 2. – №. D12. – C. 356-360.

24. Prostyakova N. et al. Issues of professional ethics in the treatment and management of patients with late dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 158-165.

25. Prostyakova N. et al. Sadness and loss reactions as a risk of forming a relationship together //Science and innovation. – 2023. – T. 2. – №. D12. – C. 252-257.

26. Prostyakova N. et al. Strategy for early diagnosis with cardiovascular diseaseisomatized mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 166-172.

27. Rotanov A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and innovation. – 2023. – T. 2. – №. D12. – C. 267-272.

28. Rotanov A. et al. Diagnosis of depressive and suicidal spectrum disorders in students of a secondary special education institution //Science and innovation. – 2023. – T. 2. – №. D11. – C. 309-315.

29. Rotanov A. et al. Elderly epilepsy: neurophysiological aspects of non-psychotic mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 192-197.

30. Rotanov A. et al. Social, socio-cultural and behavioral risk factors for the spread of hiv infection //Science and innovation. – 2023. – T. 2. – №. D11. – C. 49-55.

31. Rotanov A. et al. Suicide and epidemiology and risk factors in oncological diseases //Science and innovation. – 2023. – T. 2. – №. D12. – C. 398-403.

32. Sedenkov V. et al. Clinical and socio-demographic characteristics of elderly patients with suicide attempts //Science and innovation. – 2023. – T. 2. – №. D12. – C. 273-277.

33. Sedenkov V. et al. Modern methods of diagnosing depressive disorders in neurotic and affective disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 361-366.

34. Andreichev LA, Baleeva LV Anemia of chronic diseases. Rus. asal. journal 2014; 2:50–5. [Andreichev LA, Baleeva LV Anemia of chronic diseases. Rus. med. zhurn. 2014; 2:50–5. (in Russian)]

35. Ebner N., Jankowska EA, Ponikowski P., Lainscak M., Elsner S., Sliziuk V. et al. The impact of iron deficiency and anemia on exercise capacity and outcomes in patients with chronic heart failure. Results of studies investigating comorbidities that exacerbate heart failure. Int. J. Cardiol. 2016; 205:6–12. DOI: 10.1016/j.ijcard.2015.11.178

36. Rukavitsyn OA Anemia of chronic diseases: selected aspects of pathogenesis and methods of correction. Onkogematologiya. 2016; 11(1): 37–46. [Rukavitsyn OA Anemia of chronic diseases: selected aspects of pathogenesis and methods of correction. Onkogematologiya. 2016; 11(1): 37–46. (in Russian)]

37. Caughey MC, Avery CL, Ni H, Solomon SD, Matsushita K, Wruck LM, et al. Outcomes of patients with anemia and acute decompensated heart failure with preserved and reduced ejection fraction (from the ARIC Research Society follow-up). Am. J. Cardiol. 2014; 114(12): 1850–4. DOI: 10.1016/j.amjcard.2014.09.024

38. Sharova EK, Babaeva LA, Padaryan SS, Soselia NN, Lukina OI, Milto AS Chronic heart failure: recommendations and actual clinical practice. Diet. pharmacotherapy in cardiology. 2016; 12(6): 631–7. [Sharova EK, Babaeva LA, Padaryan SS, Soseliya NN, Lukina OI, Mil'to AS Chronic heart failure: recommendations and real clinical practice. Ratio. Pharmacotherapy and cardiology. 2016; 12(6): 631–7. (in Russian)]

39. Yusuf S., Pitt B., Davis CE, Hood WB, Cohn JN. Left ventricular ejection fractions and the effect of enalapril on survival in patients with congestive heart failure. N. Engl. J. Med. 1991; 325(5): 293–302. DOI: 10.1056/NEJM199108013250501

40. Silverberg DS, Wexler D, Blum M, Tchebiner JZ, Sheps D, Keren G, et al. Effect of subcutaneous erythropoietin and intravenous iron on the correction of anemia in diabetic and nondiabetic patients with severe refractory congestive heart failure and chronic renal failure. Nephrol. Dial. Transplant. 2003;18(1):141–6.

41. Martirosyan NV, Tazhenova NN, Demidov AA Dynamics of clinical indicators depending on treatment in elderly patients with chronic heart failure with anemia. Intl. Journal of the. and. main. research. 2015; 12:827–9. [Martirosyan NV, Tazhenova NN, Demidov AA Dynamics of clinical indicators in elderly patients with chronic heart failure with anemia depending on treatment. Intl. Journal of the. and. main. research. 2015; 12:827–9. (in Russian)]

42. Alexander M., Grumbach K., Remy L., Rowell R., Massie BM. Congestive heart failure hospitalization and survival in California: patterns by race/ethnicity. Am. Heart J 1999;137(5):919–27.