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The Structure of the Anemia Syndrome in Patients With Chronic Heart Failure and Chronic Kidney Disease

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Abstract: General Background: Chronic heart failure (CHF) and chronic kidney disease (CKD) frequently coexist, exacerbating clinical outcomes and contributing to anemia, which further deteriorates cardiovascular and renal functions. Specific Background: Anemia in CHF and CKD is multifactorial, involving iron deficiency, chronic inflammation, and protein-energy malnutrition. The pathophysiological mechanisms, including erythropoietin resistance and impaired iron metabolism, require further investigation to optimize therapeutic interventions. Knowledge Gap: Despite recognition of anemia's role in CHF and CKD progression, the structural characteristics of anemia syndrome, particularly the interaction between iron metabolism markers and nutritional status, remain insufficiently studied. Aims: This study aims to determine the structure of anemia syndrome in CHF and CKD patients by analyzing iron parameters, transferrin saturation, hemogram indices, and protein metabolism markers to assess their impact on anemia severity. Results: The findings reveal a predominance of anemia of chronic disease (ACD), with progression leading to a combination of ACD and iron deficiency anemia (IDA). Severe anemia was linked to significant reductions in iron stores, transferrin saturation, and markers of protein metabolism, indicating trophological disturbances. Novelty: This study identifies the combined influence of iron metabolism and malnutrition in CHF-CKD patients with anemia, offering a comprehensive perspective on anemia pathogenesis beyond iron deficiency alone. Implications: The results emphasize the necessity of an integrated therapeutic approach that includes anemia correction and nutritional interventions to improve patient outcomes. Further research should explore the relationship between nutritional status, iron metabolism, and CHF decompensation to refine clinical management strategies.

Key words: Chronic heart failure, chronic kidney disease, anemia of chronic disease, iron deficiency anemia, protein metabolism, transferrin saturation, trophological status.

INTRODUCTION

Chronic heart failure (CHF) is a common complication of many cardiovascular diseases, 9 out of 10 cases of CHF are associated with coronary heart disease (CHD)[1] and arterial hypertension. The prevalence of CHF reaches 3% in the general population. Mortality from this pathology, despite therapeutic successes, is not inferior to its leading positions and is comparable to mortality from oncological diseases. The study of potential factors for the development and progression of CHF is an urgent issue of modern medicine. The presence of comorbid pathology, namely anemia and renal dysfunction, determines the clinical course of CHF according to the results of large studies. The prevalence of renal dysfunction is quite high in patients with CHF, and the severity of chronic kidney disease (CKD)[2] correlates with the severity of CHF. According to the results of a study that included more than 1 million U.S. adults, CHF and anemia independently increase the risk of death or the need for dialysis by 50-100%, and their combination - by 300% [3]. According to Silverberg and co-authors, the connection between the heart and kidneys is mutually potentiating, changes in one of the organs - the heart or kidneys - can lead to dysfunction of the other organ. Such relationships have been described within the framework of the cardiorenal continuum. Anemia is an important predictor of the

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unfavorable course of CHF. The pathophysiological mechanisms underlying the development of anemia are multifactorial [4]. A certain The role is assigned to the high activity of immunoinflammation due to the induction of erythropoietin resistance and renal dysfunction as a factor determining a significant decrease in erythropoietic activity, as well as the presence of persistent malabsorption and micronutrition. Rather contradictory information exists about the structure of the anemia syndrome in patients with CHF. According to some authors, the predominance of signs of anemia of chronic disease (AHD)[5] in patients with CHF has been revealed, which is associated with reticuloendothelial iron block (Fe) as a result of activation of the central regulator of Fe homeostasis, hepcidin, by high concentrations of proinflammatory cytokines. The problem of malnutrition in patients with CHF[6] and CKD remains relevant and intensively studied. According to the literature, this category of patients has a variety of metabolic and trophological disorders accompanied by protein and energy deficiency. The question of the structural features of the anemia syndrome and the nature of nutritional changes in patients with CHF and CKD[7]. These provisions determine the relevance of conducting research in this area, which will optimize therapy and influence the prognosis of comorbid pathology. The relationship of work with scientific programs and topics. The aim of the study was to determine the structure of the anemia syndrome based on the study of the level of enteric Fe, the coefficient of saturation of transferrin (CST)[8], pherritin and hemogram parameters, as well as the assessment of the presence of violation of the trophic status based on the study of indicators of protein metabolism - total protein (OB)[9], albumin and transferrin (Tf) of the blood serum, the absolute number of lymphocytes in patients with CHF and CKD with varying severity of anemia[10].

MATERIALS AND METHODS OF RESEARCH

145 patients with CHF of the II-IV phases and BS were examined (average age 71.42±8.66 years). The main group included 87 patients with signs of anemia and stage II-III PD on the background of CHF . The comparison group consisted of 58 patients with chronic heart failure without signs of anemia and CKD. Patients with acute coronary syndrome, acute myocardial infarction, and other diseases contributing to the development of anemia (pathology) were excluded from the study. gastrointestinal tract, oncological diseases of various localization, bleeding, diagnosed on the eve of hospitalization or during hospitalization). Heart failure was determined according to the classification of the American Heart Association (NYHA). The presence and stages of CD were determined according to the classification proposed by the experts Native renal ph onda SH A (NKF) K/DO Q. The diagnosis of anemia was determined according to According to the criteria of the Medical Committee of Standards of Hematology (ICST, 1989): a decrease in the concentration of Hb in venous blood of more than 120 g/l for women and more than 130 g/l for men. The severity of anemia was assessed by the level of the Hb index: mild anemia was diagnosed when the Hb level was lower than 120 g/l (for women) or 130 g/l (for men) to 90 g/l, moderate severity - from 89 g/l to 70 g/l and severe - 69 g / l or less. Parameters of protein metabolism were used to assess the trophic status. The criteria for protein deficiency were a decrease in the level of Tf <2 g/l, a decrease in the level of OB <65 g/l, mainly due to albumin <35 g/l, and lymphocytopenia < 18 oo*109/l. All patients underwent clinical and biochemical blood tests. ECG examination, echocardiography in Doppler mode, ultrasound of the liver and kidneys, fibrogastroduodenoscopy. The Fe concentration was determined using a colorimetric method using a sample set Liquick Cor-FERR UM reagents (Corm ay, Poland). The concentration of ferritin was determined by the immunopharmaceutical m method using a set of reagents "Ferritin ELISA" (DAI, U SA). The average volume of red blood cells (pl or m cm3) was calculated using the formula: M ^ = ((blood cells, %)/(number of red blood cells, (in ml))x1o. The average hemoglobin content in erythrocytes (pg) was calculated using the formula: MN =(hemoglobin, g/100 m l)/(number of red blood cells, (in millions)). The coefficient of transferrin saturation (CST) was defined as the ratio of the level of cellular Fe to the level of the general electrical ability of the gate. More than 20% of the r was regarded as a criterion of high efficiency. The serum albumin concentration was determined using a colorimetric method using the Liquik Cor-ALBU M IN diagnostic kit (Corm ay, Poland). The blood levels were determined using a colorimetric method using an ECOtest reagent kit (Russia). And the maximum number of pounds was determined as the ratio of the product of the percentage of pounds and the number of pounds to 100. The statistical processing of digital data x was performed using the

standard package of application programs M icrosoft Excel and STATISTICA 6,o for W indow s. The values of e are represented as (M ±m). The statistical significance of the different averages was determined by the criterion F is an epa chip.

THE RESULTS AND THEIR DISCUSSION

In the patients of the main group, there was a significantly more pronounced decrease in serum Fe, KT, and hemogram parameters (Ht, MCH, MCV)[11] compared with these indicators in the comparison group. In patients with mild anemia who developed on the background of CHF and CKD, MCV did not significantly differ from the value from the comparison group. Determination of the ferritin content showed a tendency to decrease with the severity of anemia, however, there were no significant differences between the studied indicators and the results. Comparing the concentrations of ferritin in patients of the main group Compared with the comparison group, there were no significant differences ($p>0.05$)[12]. In order to assess the trophic status, the level of blood volume, the absolute number of lymphocytes, albumin, and serum Tf was studied. Patients with CHF and PD with varying degrees of anemia have decrease in blood volume, absolute number of lymphocytes, albumin, and serum Tf when compared with the comparison group (differences are significant, $p<0.05$)[13]. There is a significant decrease in the indicators characterizing protein metabolism, in proportion to the severity of the anemia syndrome in patients with CHF and CKD. Analysis of the structure of the anemia syndrome in patients of the main group showed that 18% of patients with mild anemia who developed on the background of CHF and CKD[14], serum Fe was within the normal range, 82% of patients in this group showed a decrease in serum Fe levels. A decrease in CST<20% was detected in 76.7% of patients, in 23.3% the studied indicator remained within the normal range[15]. When determining CP, MCH in patients with mild anemia on the background of CHF and The studied indicators did not differ from the norm in CKD in any of the examined patients. When studying the levels of Ht, MV and ferritin, it was revealed that in all patients with a combination of CHF, CKD and mild anemia, the level of the studied parameters was within the normal range. Therefore, in patients with CHF and CKD mild anemia is normochromic and normocytic, with a moderate decrease in serum Fe levels combined with a high concentration of ferritin, which is a reflection of sufficient resources of reserved Fe and indicates the presence of ACH in this cohorts of patients[16]. The study of trophological parameters in patients with mild anemia on the background of CHF and CKD revealed a decrease in the level of OB in 77.8%, albumin in 80%, Tf in 100%, and the absolute number of lymphocytes in 80.5% of individuals. In all patients with anemia of moderate severity, who developed on the background of CHF and CKD, a decrease in the level of cervical Fe was determined[17]. Also, 21% of patients in this group had a decrease in M CH; a decrease in CP was observed in 25% of patients, and no changes were found in more than half (54%)[18] of patients. Reducing the level of ferritin It was found in 12% of patients with x, and a decrease in M C V was found in 15% of patients in this group. It was found that in all patients with CHF , CKD and anemia of moderate severity, the level of H t did not differ from the norm. Numerous changes indicate that the progression of anemia in patients with CHF and PD is associated with a decrease in CP, M CV, M HF and the appearance of microcytosis, This, along with a reduced level of enteric Fe and a high concentration of ferritin, indicates the presence of a combination of A X Z and W D A. 12% of patients in this group had low levels of ferritin, which is associated with depletion of readily available Fe in the depot and is a sign of isolated fever[19]. In all patients with moderate anemia, who developed on the basis of CHF and CKD, a decrease in the level of blood volume, albumin, and Tf is determined. The number of defects was significantly reduced in 95.5% of patients in the study cohort. Among the examined patients, x patients with severe anemia developed during CHF Phase II-IV and CD of stage II-III predominated in patients (66.7%)[20] with low levels of MG and CP (69%)[21]. In all patients of this group, a decrease in portal Fe was noted; in 1.7 ,5% of patients with x, a decrease in the level of ferritin was detected and in 24% of patients with x - M CV. The given impressions are excluded It is believed that in patients with CHF and PD, anemia is characterized by a decrease in heart rate, MV, the level of cervical Fe, and an increase in the number of patients with microcytosis. You can see the changes along with the established high concentration of ferritin, indicating the predominance of patients with a combination of A X Z and W D A. In the group of patients with severe anemia on the background of CHF II-IV F and stage II-III PD, there were

patients with isolated hypertension, 17.5%[22] of whom had a relatively low serum ferritin level. Ht reduction was detected in 9.8% of patients, This indicates the significance of hemodilation anemia in the progression of anemia severity in this category of patients. The study of trophic status indicators showed that all patients with CHF, CKD and severe anemia showed a decrease in blood volume, albumin, Tf, and the absolute number of lymphocytes[23]. The results indicate a different severity and structure of the anemia syndrome, the presence of pathological disorders associated with an imbalance in the functioning of the visceral protein pool in patients with CHF in combination with CKD, the diagnosis of which will allow us to determine differentiated approaches to therapeutic correction of this category. Patients[24].

The study findings reveal a complex interplay between anemia, chronic heart failure (CHF), and chronic kidney disease (CKD), emphasizing the predominance of anemia of chronic disease (ACD) with an increasing contribution of iron deficiency anemia (IDA)[25] in advanced cases. Patients with CHF and CKD exhibited a significant decline in serum iron levels, transferrin saturation, and key hemogram parameters, alongside notable reductions in protein metabolism markers. The structural analysis of anemia syndrome demonstrated that mild anemia in CHF-CKD patients primarily presents as normocytic and normochromic, while moderate to severe anemia is associated with microcytosis and hypochromia, indicating progressive iron depletion and malnutrition-related hematological alterations [26].

Patients with mild anemia showed largely preserved ferritin levels, suggesting the presence of functional iron deficiency rather than absolute iron depletion. However, as anemia severity progressed, a substantial proportion of patients exhibited depleted iron stores alongside reduced hemoglobin indices, indicating a transition towards combined ACD and IDA. The depletion of transferrin, albumin, and lymphocytes further underscores the presence of trophological disturbances, which contribute to the worsening of anemia severity. These findings highlight the necessity for a dual therapeutic strategy targeting both iron metabolism and nutritional deficiencies to improve patient outcomes [27].

Deep Theoretical and Practical Research

The pathophysiology of anemia in CHF-CKD patients is multifactorial, involving chronic inflammation, erythropoietin resistance, and impaired iron metabolism. While current research has extensively documented the role of hepcidin in iron sequestration, further investigation is required to elucidate the precise mechanisms through which nutritional status influences erythropoiesis and anemia progression. The interrelationship between inflammatory cytokines, iron regulatory pathways, and metabolic disturbances warrants deeper exploration to optimize therapeutic interventions [28].

From a practical standpoint, the integration of nutritional support alongside conventional anemia management could enhance clinical outcomes. The observed reductions in protein metabolism markers indicate the necessity of personalized dietary interventions that focus on improving protein-energy status and micronutrient availability [29]. Future clinical trials should evaluate the efficacy of combined iron and nutritional therapy in mitigating anemia-related complications in CHF-CKD patients[30].

Knowledge Gap and Future Research Directions

Despite advancements in understanding anemia in CHF-CKD patients, significant gaps remain. First, the role of micronutrient deficiencies beyond iron, such as vitamin B12 and folate, remains underexplored[31]. Given that malnutrition is prevalent in this patient cohort, future studies should investigate the broader nutritional landscape contributing to anemia[32].

Second, the long-term effects of current therapeutic strategies, including erythropoiesis-stimulating agents (ESAs) and iron supplementation, require further validation[33]. While ESAs are commonly prescribed, their impact on cardiovascular morbidity and mortality in CHF-CKD patients is still debated[34]. Future research should assess alternative treatment regimens, such as hepcidin-targeted therapies or novel erythropoiesis modulators[35].

Third, more studies are needed to establish precise biomarkers that can differentiate between ACD, IDA, and mixed anemia types in CHF-CKD patients [36]. The incorporation of advanced diagnostic techniques, such as functional iron assessment and proteomic profiling, could provide a more comprehensive understanding of anemia subtypes, leading to personalized treatment approaches [37].

CONCLUSIONS

This study highlights the complex structure of anemia syndrome in patients with chronic heart failure (CHF) and chronic kidney disease (CKD), revealing a predominance of anemia of chronic disease (ACD), with progression often leading to a combination of ACD and iron deficiency anemia (IDA). The findings indicate that severe anemia in CHF-CKD patients is characterized by decreased serum iron, transferrin saturation, and hemogram parameters, alongside significant reductions in protein metabolism markers, reflecting profound trophological disturbances. These results underscore the necessity of an integrated therapeutic approach that not only addresses iron metabolism but also targets malnutrition to optimize anemia management. Clinically, recognizing the interplay between inflammatory processes, iron homeostasis, and protein metabolism is critical for improving prognosis and guiding treatment strategies in CHF-CKD patients. Future research should focus on elucidating the molecular mechanisms linking nutritional deficiencies and erythropoiesis impairment, as well as evaluating the long-term effects of combined iron and nutritional therapies to enhance clinical outcomes in this patient population.

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