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PRINCIPLES OF TREATMENT OF ANEMIA WITH CHRONIC HEART FAILURE

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ABSTRACT: The effectiveness of the treatment of patients with chronic heart failure also depends on the elimination of associated conditions that can lead to the progression and complications of the disease. Treatment of anemia, first of all, should be based on its etiological factor. The review presents data on the current approach to the treatment of anemia in patients with chronic heart failure.
Keywords: chronic heart failure, anemia.

INTRODUCTION

The effectiveness of treatment of patients with chronic heart failure (CHF) also depends on the elimination of comorbid conditions that can lead to exacerbation and complications of this disease. In them, the treatment of anemia should be carried out primarily by taking into account its etiological factor. To achieve this goal, it is necessary to first thoroughly examine the patients and determine the cause of anemia. In 2013, the first recommendation was developed by the American Association of Cardiologists for the treatment of anemia in patients with cardiovascular disease [4].

The recommendations of the American Association of Cardiologists for the treatment of anemia in patients with cardiovascular disease set out the principles for the management of patients with cardiological diseases and anemia regardless of the etiology of anemia. Three treatment strategies have been proposed: enhanced erythropoietin production; erythromass infusion - filling iron deficiency; intravenous administration of iron supplements. Given that erythrocyte mass transfusion may worsen the condition of patients, this procedure can be performed only in a hospital setting in patients with severe ischemic heart disease (CHD). The use of erythropoietin, a stimulant, is also not recommended in the treatment of patients with YuIK and SYuE with mild to moderate anemia due to the high probability of developing thromboembolic complications and a positive effect on the outcome of the disease and the number of hospitalizations. There are currently positive reports that the elimination of iron deficiency in the body by intravenous administration of iron carboximaltose leads to increased endurance of patients to physical exertion, improved quality of life, re-hospitalization, and reduced mortality [1,5]. An analytical article entitled "Treatment of anemia in patients with cardiovascular disease" [1] provides a review of some articles devoted to the treatment of iron deficiency with drugs. In one of them, iron carboxy-maltose (ferinect) was administered 200mg per week for 8–12 weeks, then 200mg per month for 24–26 weeks [5], and in two other studies, venofer was administered 200 mg per week for 5 weeks as an iron hydroxide sucrose complex. reported a positive effect on the outcome of the disease [1,3]. According to the results of these studies, intravenous administration of iron preparations led to a decrease in functional classes of the disease, increased resistance to physical exertion,

and improved renal function and quality of life in patients with SLE. There was also a significant increase in hemoglobin, left ventricular hemorrhage fraction (CHQOF), natriuretic peptides, decreased S-reactivity protein (S-RO), and a decrease in the number of hospitalizations associated with SYuE decompensation. However, there are insufficient data on the effect of monotherapy on iron-on mortality and other adverse effects in SWE, although results are confirming an improvement in disease prognosis [3].According to the recommendations of the European Society of Cardiology, the use of iron supplements in conditions of proven iron deficiency can lead to an improvement in the course of SUE, and these drugs can be recommended before the use of erythropoietin drugs [8]. Some authors advocate the use of erythropoietin in combination with iron [9]. The effectiveness of a combination of erythropoietin and iron supplements cannot be achieved with oral iron supplements [10]. It is also decided which patients should or cannot take iron supplements orally by determining the amount of hepsidin in their serum. Intravenous iron preparations - iron gluconate (Ferrlecit, Ron-Pulenk, 62.5mg) and iron sucrose (venofer, Uriach, 100mg), practically do not cause any side effects. When the dosage is compared, especially during a blood transfusion, 250 mg of iron is introduced into the body and it decreases with the breakdown of the transfused erythrocytes. In this case, there is no iron deficiency, but there will be anemia. Iron deficiency anemia is a major pathogenetic factor in some patients, in which case iron therapy is considered sufficient and erythropoietin should not be recommended [7]. This is also confirmed by the study of IRON-HF [9], which studied cases of absolute and relative iron deficiency caused by secondary blood loss, antithrombocytic-anticoagulant therapy, and causes such as aspirin gastritis [8]. In addition, iron deficiency may occur due to insufficient intake of vitamins B12, folic acid, and iron, which are necessary for the maturation of erythroid cells in SYuE, or their inability to be absorbed in the gastrointestinal tract [7]. Recent studies do not provide sufficient data on the occurrence of vitamin deficiency in SLE, and these pathogenetic elements of anemia are not considered an important factor in determining the outcome of the disease. Long-term treatment of patients with iron deficiency anemia with low CQOF-proven iron deficiency anemia has been shown to reduce YuE symptoms, increase endurance to physical exertion, and improve quality of life [4]. The European Association of Cardiologists' 2012 recommendation for the diagnosis and treatment of heart failure reported that intravenous iron and recombinant erythropoietin (rEPO) were effective [3]. With this in mind, in recent years, extensive research has been conducted on the treatment of anemia with erythropoietin in patients with SYuE [6]. The first multicenter randomized clinical trial, called RED-HF, showed positive results in the treatment of patients with SLE with erythropoietin, improving clinical condition, reducing shortness of breath, and increasing endurance to physical activity [10]. Unfortunately, the data obtained at the end of the study did not prove the primary theory. The use of erythropoietin (darbepoetin-alpha) did not have a positive effect on the number of hospitalizations and deaths in patients with SYuE with a hemoglobin level below 120 mg / l [10]. At the same time, the number of thromboembolic complications in this group of patients increased by 3.5 times, and stroke by 1.7 times. Similar cardiovascular complications have been reported with the use of erythropoietin in the treatment of patients with chronic kidney disease (CHD) and

type 2 diabetes [6]. When the optimal hemoglobin value was 120–140 mg / l, such complications were associated with its elevation above 145 mg / l [8]. However, in the RED-HF test, the hemoglobin level did not exceed an average of 130 mg / l, but the risk of complications increased [2]. It was concluded that erythropoietin should not be used in the treatment of patients with SYuE, even if the hemoglobin level is low (recommended class III, proof level A) [2].

Iron deficiency anemia is more common in patients with SLE. This is evidenced by the fact that most of it are low in hemoglobin (≤ 120 mg / l) and the amount of iron in the serum is less than 10 μmol / l. Determining the amount of ferritin (≤100 ng / ml in the presence of absolute iron deficiency) or transferrin iron saturation (≤20%) allows a reliable assessment of the nature of anemia. A low level of iron saturation of transferrin with a normal amount of ferritin indicates a relative iron deficiency. In these cases, the use of iron supplements is effective in the treatment from a logical point of view [2]. Although oral iron supplements are used, this group of patients refuses to take them due to the presence of gastrointestinal absorption syndrome and side effects of the drug (dyspeptic changes in 10–40% of patients, constipation, etc.). In an article published in the Journal of the American College of Cardiologists, Dr. John Nenas of the Athens Medical School and his colleagues wrote that "intravenous administration of iron supplements should be considered an effective treatment for iron deficiency in the late stages of heart failure." A placebo-controlled randomized FAIR-HF study involving 149 patients with NYHA II – III FS was studied in the SYUE. Iron content was calculated using the Ganzon formula and correlated with hemoglobin index, serum transferrin saturation level, and ferritin content. At the end of the study, the effectiveness of treatment was analyzed by self-assessment of health status (PGA), determination of SYuE resistance to FS and physical load (6-minute walking test), and assessment of the quality of life using a questionnaire. In addition, the efficacy, safety, and well-being of patients with intravenous administration of Ferinect for the study have been scientifically and practically proven [4]. In 50% of patients receiving ferinect, the general condition improved ($r < 0.001$) in only 27% of those in the placebo group. At the same time, in the main group of patients, functional class I and II increased by 17% ($r < 0.001$) at 24 weeks of treatment due to a decrease in SYuE NYHA III FS. At all stages of the study (4, 12, 24 weeks), patients' quality of life, self-health assessment, and 6-minute walking test performance improved significantly ($r < 0.001$), regardless of baseline hemoglobin. Renal function was improved in most patients treated with Ferinect, and the symptoms of SBK were much lower than in the placebo group. The side effects of the drugs did not differ from those in the placebo group in the group undergoing active treatment. Cardiovascular complications were reliably observed in the placebo group ($r < 0.01$) [4, 5]. The FAIR-HF study also suggested the use of iron supplements for prophylactic purposes in patients with SYUE even in the absence of symptoms of anemia [9,10]. At present, scientific research on the effects on the inflammatory system, the process of remodeling of the heart, and the balance of the disease for a long time through the treatment of iron deficiency prolongs the life of patients in the SYUE is required. The available literature does not provide complete information on the treatment of anemia in patients with SYuE, including ERO, ferritin, IL-1 and IL-6, and a-FNO, and there is no clear generally accepted view. Thus, anemia in SYUE

is a destructive factor leading to disease aggravation, progression, and mortality, and the results of many studies suggest that this problem needs to be addressed.

In summary, the available data in the literature confirm that among the many comorbid cases, the adverse effects of anemia on the course and outcome as well as renal function have been poorly studied. Studies confirm that its presence can lead to worsening of patients' condition, exacerbation of the disease, increase in hospitalizations, and deaths. Diagnosis of anemia in this group of patients, monand treatment is a little-studied area in medicine, indicating the need for new research in this area.

References

1. Abdullaev T.A., Tsoi I.A., AkhmatovYa.R. New in the treatment of chronic heart failure according to the results of the recommendations of the European Society of Cardiology in 2016 // Uzbekiston Cardiology. - 2017. - Volume 43, No. 1. - S. 51-53.

2. ESC recommendations for the diagnosis and treatment of acute and chronic heart failure // Russian Journal of Cardiology. - 2017. - Volume 141, No. 1.3.Abraham W., Klapholz M., Anand I. Safety and efficacy of darbepoetin alfa treatment in anemic patients with symptomatic heart failure: a pooled analysis of two randomized, double-blind, placebo-controlled trials // Eur Heart J. - 2006, №27. - P. 166-167.

4. Anker S. D, Colet J. iron improves symptoms in chronic heart failure patients with iron deficiency // New Eng J Med. – 2009. – Vol. 361, №11. – P. 2436-2448.

5. Anker S. D., Comin C. J., Filippatos G. et al. Ferric carboxymaltose in patients with heart failure and iron deficiency // New Eng J Med. – 2009. – Vol. 361, №25. – P. 2436-2448.

6. Gadaev A.G., Xalilova F.A., Elmuradov F.X., Tosheva X.B. Structural and functional changes in the kidneys and heart in patients with XSN. Therapy Bulletin of Uzbekistan. 2018. -1 - S. 100-104.

7. Belonje A. M, Voors A. A, van Gilst W. H, van Veldhuisen D. J. Erythropoietin in chronic heart failure // Congest Heart Fail. – 2007. – Vol. 13, №5. – P. 289–292.

8. Bolger A. P., Bartlett F. R., Penston H. S., O'Leary J., Pollock N., Kaprielian R., et al. Intravenous iron alone for the treatment of anemia in patients with chronic heart failure // J Am CollCardiol. – 2006. – Vol. 48. – P. 1225-1227.

9. Ezekowitz J. A., McAlister F. A., Armstrong P. W. Anemia is common in heart failure and is associated with poor outcomes // Circulation. - 2003. - Vol. 21. - P. 223-225.

10. Kodirova, NargizakhonUmarovna MODERN METHODS OF TREATMENT OF CHRONIC LUNG DISEASES // ORIENSS. 2022. № Special Issue 4-2. URL: https://cyberleninka.ru/article/n/modern-methods-of-treatment-of-chronic-lung-diseases).

11. ToshevaKh.B., Xalilova F.A., Gadaev A.G., Erkinova N.E., Djuraeva N.O. Impact Of Chronic Heart Failure On Comorbidities In Hot Climates On The Quality Of Life And Clinical Condition Of Patients // European Journal of Molecular & Clinical Medicine. -2020 – Vol. 7, P. 1080-1089.