ANEMIA IN THE PRACTICE OF A GENERAL PRACTITIONER: A NEW LOOK AT AN OLD PROBLEM

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Abstract: *The general practitioner usually has to deal with comorbid diseases in patients. Anaemia and iron deficiency commonly cause congestive heart failure (CHF) in the elderly. Iron deficiency in combination with anaemia and individually can worsen the course of CHF and diseases underlying its development, reduce the life quality, physical activity of patients and increase the risk of adverse events. Fundamentally important is to conduct iron deficiency screening in all patients with CHF, especially during the first occurrence, and laboratory studies are crucial when diagnosing iron deficiency anaemia. To date, there is no unequivocal evidence for the treatment of elderly patients with CHF and anaemia. Therefore, it is necessary to identify and correct the causes of iron deficiency in patients with CHF, prescribe neuromodulators in recommended doses, in particular, the main groups of drugs (angiotensin-converting enzyme inhibitors and angiotensin I I receptor antagonists, β-blockers, mineralocorticoid receptor antagonists) and a reasonable approach to the treatment of the edema syndrome.*

Keywords: *anaemia, iron deficiency, congestive heart failure, iron supplements, treatment.*

Anemia and chronic heart failure

Most of the problems associated with the diagnosis, treatment and rehabilitation of elderly patients with chronic heart failure (CHF) still have to be solved by a general practitioner and a general practitioner. To date, CHF can be considered as an elderly disease, since the "phenotype" of CHF patients has changed significantly over the past two decades: the number of older patients, the number of patients with preserved and intermediate left ventricular ejection fraction (LVEF), the number of concomitant diseases and medications taken has increased [1, 2]. Concomitant diseases can mutually provoke the progression of each other, including decompensation of cardiac activity, leading to hospitalizations or death. Among the concomitant diseases, anemia and iron deficiency (iron deficiency) are of particular interest.

When detecting anemia in an elderly patient with CHF, a number of questions arise before the doctor, especially in the primary link:

What is the cause of anemia?

What is the minimum of diagnostic measures required to identify the nature of anemia?

How to prescribe the right treatment?

Pathogenetic basis of comorbidity

The pathogenesis of anemic syndrome in the elderly with CHF is multifactorial, including deficiency of iron, folic acid, vitamin B12 and other factors . Almost every 3rd elderly patient develops anemia without an obvious cause (hypoproliferative anemia against the background of low erythropoietin activity) [4]. Regulation of erythropoiesis is a complex and complex process involving different representatives of cytokines. For the final differentiation of erythroid cells, erythropoietin is needed — a glycoprotein, the primary mediator of the normal physiological response to hypoxia, stimulating mitosis and being a differentiation hormone that promotes the formation of erythrocytes from stem cells. Erythropoietin is produced mainly in the kidneys (mainly in peritubular interstitial fibroblasts of the cortex and the outer region of the medulla) and to a lesser extent (up to 15%) — in the liver (by hepatocytes and fibroblast—like liver cells - Ito cells).

CHF promotes hypoperfusion of the kidneys, redistribution of blood flow in them, vasoconstriction, which leads to impaired renal function in almost every 2nd elderly patientч. Anemia developing in patients with CHF and kidney pathology is determined by a deficiency of endogenous erythropoietin production due to an irreversible decrease in the number of kidney glomeruli or their relative ischemia due to a decrease in cardiac output. As the functional activity of the kidneys decreases, the structures producing erythropoietin are replaced by fibrous tissue and lose their hormone-producing properties. As a result of renal hypoperfusion, ischemia of endotheliocytes of peritubular capillaries and fibroblasts localized in the tubulointerstition increases, and erythropoietin production decreases. In addition, proteinuria in CHF increases the loss of erythropoietin, transferrin, ionized iron in the urine and can lead to the development of an iron deficiency condition.

Suppression of bone marrow function with the formation of anemia in CHF can also be realized through inflammatory reactions, because with this disease, the level and activity of proinflammatory cytokines, in particular TNF-α and IL-6, increases. An increase in cytokines is associated with inhibition of proliferation and differentiation of erythroid progenitor cells, with inadequate hypoxia, production of endogenous erythropoietin and increased synthesis of hepcidin. The latter is released in the liver in response to inflammation and inhibits the absorption of iron in the small intestine and the release of iron from the reticuloendothelial macrophage system due to a decrease in ferroportin activity, which leads to ineffective erythropoiesis. It was shown that the low level of hepcidin, along with the normal values of the inflammatory response indicators and the absence of a link between them, as well as the lack of correlation between the level of hepcidin and the concentration of hemoglobin, indicates that hepcidin does not play a role in the development of anemia in older patients with CHF and IDA. Conversely, high levels of hepcidin, indicators of inflammation, positive correlations

between them and a negative correlation between hepcidin levels and hemoglobin concentrations indicate inflammation as the cause of an increase in hepcidin levels, which causes the development of anemia in CHF in the elderly and senile age.

Principles of differential diagnosis

Early manifestations of CHF in old age may be fatigue, weakness, heaviness in the legs, often manifested during daily physical activity and not always associated with the severity of shortness of breath and edematous syndrome. Patients may complain of dizziness, presyncopal and syncopal states, irritability, sleep disturbance. As a rule, these symptoms are not very specific, since they can also be present in other diseases, in particular in anemia. The clinical picture of anemic syndrome includes 3 subjective symptoms: fatigue, shortness of breath and palpitation. When examining a patient with CHF, one should pay attention not only to these symptoms, which are an integral part of the clinical picture of heart failure, but also remember about the anemia syndrome and not ignore the role of hemodilution, especially in the elderly.

Difficulties in the differential diagnosis of CHF arise, as a rule, due to multimorbidity. In elderly patients with anemia, it is also often necessary to exclude or confirm the presence of CHF. Diagnostic criteria in this case are a set of anamnestic data (in particular, coronary heart disease, myocardial infarction), clinical symptoms and signs (displacement of the apical push to the left, increased heart rate), data from objective research methods.

With anemia that has developed against the background of blood loss, an increase in the number of reticulocytes is noted immediately after bleeding. The number of leukocytes tends to decrease, but the leukocyte formula does not change. The number of platelets is usually normal, with a tendency to increase with bleeding. There is a decrease in serum iron levels and an increase in the total iron-binding capacity of blood plasma. About 33% of the total amount of serum transferrin is associated with iron (an indicator of transferrin saturation with iron). The remaining amount of transferrin remains free and characterizes the latent iron binding capacity of the blood serum. With iron deficiency, the level of transferrin decreases to 10-20 mg / l, but at the same time the latent iron-binding capacity of plasma increases.

Basic principles of treatment of IDA, including in elderly people with CHF

1. To determine the cause of blood loss and to treat the underlying disease.
2. It is impossible to cure IDA with just one diet!
3. It is impossible to compensate for iron deficiency without iron-containing medicines!
4. Therapy with iron preparations must be carried out for a long time.
5. IDA therapy should not stop after the normalization of hemoglobin levels and the number of red blood cells.
6. Hemotransfusion with IDA should be carried out strictly according to vital indications.

7. Parenteral administration of drugs is indicated only under certain circumstances.

In this regard, the experts of the Society of Specialists in Heart Failure focus on the need to identify and correct the causes that lead to the development of iron deficiency in patients with CHF (alimentary disorders, gastrointestinal bleeding, taking unwanted medications, etc.). Oral iron-containing drugs can be the drugs of choice for IDA in the complex treatment of patients with CHF due to their sufficiently high efficiency and low cost. Iron preparations can be in the form of ionized (Fe2+) as part of organic or inorganic salts (fumarate, lactate, sulfate, chloride) and nonionic — as part of complexes containing iron oxide (Fe3+) - protein succinylate, polymaltosate or sucrose complex. Preparations of ionized iron are used only orally, preparations of iron oxide complexes are used parenterally. The effectiveness of treatment of patients with IDA is determined by the daily dose and the form of elemental iron that is part of the drug. At the same time, the rate of increase in hemoglobin level depends on the severity of the anemia syndrome. The choice between iron preparations — Fe2+ or Fe3+ — is determined primarily by the clinical goal: rapid achievement of the effect (preference is given to Fe2+) or good tolerability against the background of long-term use (preference is given to Fe3+).

Iron (III) hydroxide polymaltosate is indicated for the treatment of latent iron deficiency and IDA. This drug has several dosage forms, in particular the form of chewable tablets containing 400 mg of iron-polymaltose complex (100 mg of elemental iron), which allows the patient to take the drug 1 tablet 2 p. / day with a good clinical effect. This effect of the drug is due, firstly, to the structure of the complex, which has an iron (III) hydroxide core and a carbohydrate shell, which resembles an iron reserve protein — ferritin, and, secondly, controlled dissociation of iron ions from this drug complex. In addition to the above, the polymaltose shell provides stability and solubility of the complex in a fairly wide pH range, which determines its therapeutic efficacy and good tolerability, followed by an improvement in the clinical condition and quality of life, which is important in old age, especially if it is impossible to eliminate the underlying cause of the disease.

An adequate response to therapy with iron-containing drugs includes:

1. increase in the number of reticulocytes after 7 days;

2. an increase in the level of hemoglobin by ≈2 g / dl in 1-2 weeks after the start of taking iron preparations;

3. normalization of hemoglobin content after 6-8 weeks.

It is necessary to pay attention to the dynamics of erythrocyte indices, the normalization of which reflects the adequacy of therapy: the average volume of erythrocytes, the average hemoglobin content in erythrocytes, the average concentration of hemoglobin in erythrocytes. Peripheral blood tests are carried out 1 time every 1-2 weeks. at the beginning of treatment, and then 1 time in 2-4 weeks.

Since it is not quite correct to regard any decrease in hemoglobin below normal in CHF patients as anemia, in addition to true anemia, hemodilution can also be diagnosed in most CHF patients, the treatment of which should focus on dose adjustment of diuretics. It is obvious that the treatment strategy of the age category of patients, in addition to pathogenetic therapy of anemia, provides for the effect on neurohumoral activation — the use of ACE inhibitors and angiotensin II receptor blockers, mineralocorticoid receptor antagonists, beta-blockers, as well as loop diuretics. Since complex neurohumoral mechanisms are involved in the development of edematous syndrome, thoughtless dehydration causes side effects and rebound fluid retention, which can lead to unjustified therapy. Blocking hepcidin may be an effective therapeutic strategy, especially in relation to functional iron deficiency. Direct blockade of hepcidin expression using anti-hepcidin-1-oligoribonucleotide (lexaptepid), suppression of hepcidin activity using human antibody preparations to hepcidin, blocking of signal transmission from hepcidin using a low molecular weight inhibitor (LDN-193189) or non-anticoagulant heparins can be considered as promising measures. Spironolactone, commonly used as the main drug in patients with heart failure, suppresses hepcidin expression in mice.

Conclusion

Thus, anemia and iron deficiency are common concomitant CHF conditions in the elderly. Iron deficiency both on the background of anemia and independently of anemia can worsen the course of CHF and the diseases underlying its development, reduce the quality of life, physical activity of patients and increase the risk of adverse events. Screening of iron deficiency in all patients with CHF is of fundamental importance, especially when it first occurs, and when diagnosing IDA, laboratory data are crucial — confirmation of the fact of anemia and iron deficiency. To date, there are no unambiguous recommendations for the treatment of elderly and senile patients with CHF and anemia. In this regard, it is necessary to identify and correct the causes of iron deficiency in patients with CHF, influence neurohumoral activation and reasonably approach the treatment of edematous syndrome. Close cooperation of nephrologists, cardiologists, therapists is required for effective correction of CHF and anemia therapy in accordance with modern achievements and requirements. Only in conditions of active regular medical supervision is it possible to improve the quality and increase the life expectancy of patients with CHF.

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