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Research Article

CURRENT VIEWS ON IRON DEFICIENCY ANAEMIA IN PATIENTS WITH CARDIOVASCULAR DISEASE

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ABSTRACT

Currently, cardiovascular diseases (CVDs) are leading not only in prevalence but also in mortality worldwide. It is known that old age is characterized by polymorbidity, and all concomitant conditions change the clinical picture, course and prognosis of CVDs. One of the exacerbating factors of CVD is the anaemic syndrome, particularly in the case of iron deficiency. Anaemia is also an important public health problem, as it affects quality of life, mortality and is a frequent reason for seeking medical attention. A review of the literature has demonstrated that anaemia is an independent predictor of cardiovascular disease and adverse outcomes. Because the incidence of anaemia increases with age, this makes it a frequent co-morbidity of cardiovascular events. A possible mechanism seems to be an increase in sympathetic activity and cardiac output due to prolonged hypoxia-induced vasodilation, leading to left ventricular hypertrophy and increased heart size and thus increased oxygen consumption. Also myocardial tolerance to low haemoglobin levels is reduced in patients with CHD compared to healthy controls. Patients should therefore be given a full examination, including obligatory determination and monitoring of haemoglobin levels. Primary measures should be directed at the etiological factor causing anaemia of varying severity. Oral and intravenous iron, as well as erythropoietin, are used as the main therapy. Despite the development of effective diagnostic and treatment regimens and a wide range of effective medicines, problems in this area are still among the most pressing.

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KEYWORDS

Polymorbidity, cardiovascular disease, iron deficiency anaemia, anaemic syndrome, sideropenic syndrome, iron-containing drugs.

INTRODUCTION

Nowadays, polymorbidity is one of the most pressing problems of modern medicine, especially in geriatric practice. At the clinical examination of elderly and senile patients at least 4-5 diseases and manifestations of pathological processes are diagnosed. According to G.B. Aksamentov the elderly patients in geriatric hospitals had 4.1 diseases per one person, and 4.6 diseases per one (only clinically expressed nosological forms, which manifested themselves for several years were taken into account). According to VandenAkkeretaL., 78% of persons aged 80 years or older have two or more chronic diseases, whereas among children and adolescents under 19 years the percentage of polymorbidity does not exceed 10. L.B. Lazebnik et al., analyzed the number of diseases in patients in gerontological hospital therapy departments as a function of age. The authors obtained data on the number of diseases per patient aged 60-65 years - 5.2 ± 1.7; 66-70 years - 5.4 ± 1.4; 71-75 years -7.6 ± 1.7; 76-80 years - 5.8 ± 1.6; 81-85 years - 5.8 ± 1.8; 86-90 years -4.4 ± 1.6; in long-livers 91-95 years - 3.2 ± 0.5.

The onset of polymorbidity-forming diseases and their chronicity are predominantly in middle age, but the result of their cumulative accumulation, i.e. the period of vivid demonstration, begins to manifest itself in old age. The interplay of diseases changes their classic clinical picture, the nature of the course, increases the number of complications and their severity, worsens the quality of life and prognosis.

The most common polymorbid pathology in the elderly is cardiac, which is often combined with the anaemic syndrome of varying degrees of iron deficiency anaemia (IDA).

Cardiovascular disease is the leading cause of death worldwide. The WHO estimates that 17.9 million people died from CVDs in 2016, accounting for 31% of all deaths. Many patients with this pathology are anaemic due to acute or chronic comorbidities. Anaemia affects 1.62 billion people, representing 24.8% of the global population. The average prevalence of anaemia in the elderly is 23.9%. In hospitalised patients it ranges from 36% to 80%, in 25-48% of patients with chronic heart failure (CHF) and 10-20% in patients with IHD1. Anaemia is a clinical and haematological syndrome based on tissue hypoxia due to a reduction in haemoglobin levels as a result of blood loss, impaired red cell production, increased red cell destruction or a combination of these causes.

Iron deficiency is the cause of anaemia in half of all cases. Iron deficiency not only impairs erythrocyte formation, but also impairs cellular functions related to muscle metabolism and affects mitochondrial function, neurotransmitters, DNA synthesis and the immune system. Hypoxia in anemia is compensated by a cascade of hemodynamic and hemodynamically unassociated mechanisms, such as activation of erythropoietin production and increased tissue oxygen utilization. Realisation of basic haemodynamic factors is achieved by increasing myocardial contractility,

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decreasing post-load, increasing preload, and realising positive and chronotropic effects. Increased production of nitric oxide, hypoxia-induced vasodilatation and decreased blood viscosity cause vascular resistance reduction and result in decreased post-load. Chronic anaemia stimulates angiogenesis and the formation of new small vessels. The development of collaterals and the microcirculatory bed contributes to the reduction of the afterload. An increase in venous return (preload) and left ventricular filling contributes to an increase in left ventricular end-diastolic volume and ejection fraction. In short-term anaemia these changes are reversible, but with chronicity they lead to remodelling with the formation of eccentric left ventricular myocardial hypertrophy, as in other forms of volume overload.

Increased cardiac output, in turn, contributes to arterial remodelling of central elastic vessels, such as the aorta and common carotid arteries, by increasing the lumen and compensatory thickening of the intima-media complex. As a consequence, systolic pressure and inertia increase, and a larger mass of blood enters the dilated arterial system. Activation of the sympathetic nervous system increases the contractility of the left ventricle and increases the heart rate. In the presence of chronic heart disease these additional effects, mediated by anaemia, contribute to an increased incidence of cardiovascular complications.

According to European and American guidelines for the management of patients with stable angina pectoris, it is assumed that haemoglobin levels should be determined in all patients and anaemia is considered to be a contributing factor to coronary heart disease. In recent guidelines on the diagnosis and treatment of patients with CKD, American and European experts have noted that anaemia not only increases the symptoms of CKD, impairs quality of life, reduces exercise tolerance, can cause acute decompensation of CKD and increase the frequency of hospital admissions, but is also an independent negative predictor of prognosis. The risk of death in CKD patients with anaemia is twice as high as that without anaemia, even when additional variables (renal dysfunction, severity of CKD, etc) are taken into account. Latent iron deficiency may have a negative impact on prognosis in CKD, making it reasonable to determine its markers in all patients with CKD. The Bo_UE retrospective study showed that a 1% reduction in haematocrit increased overall mortality in patients with CKD by 2.7%. The oRT1ME study showed a 12% increased risk of death or rehospitalisation with a haemoglobin level of less than 12g/dl. A more severe functional class of heart failure (FC HF) according to YHRL was associated with lower haemoglobin and higher creatinine. There is evidence of a worse prognosis of cardiovascular mortality in LDA compared with other types of anaemia.

In acute coronary syndrome (ACS), the presence of anaemia can increase the likelihood of death by up to fourfold and is considered as an independent predictor of the risk of adverse clinical outcomes. Even chest pain syndrome in women is more prognostically poor (doubling the risk of death) if anaemia is associated with it. Meneveau et al., in addition to recognising anaemia as an independent risk factor for death in ACS, propose to include it along with other factors in the GRACE (Global Registry of Acute Coronary Events) risk scale for a more accurate prognosis. Studies at the population level and in patients with CHD confirm a U-shaped relationship between haemoglobin levels and cardiovascular morbidity and mortality, i.e. high haemoglobin levels (>13 g/dL) are associated with a poor prognosis, along with low levels.

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Daily mean diastolic blood pressure. Patients with isolated systolic hypertension and left ventricular hypertrophy with associated decreased haemoglobin levels have increased cardiovascular mortality and the incidence of acute cerebrovascular events. It has also been found that haemoglobin levels correlate strongly with electrocardiogram changes. Electrocardiographic repolarisation changes (depressed BT, inversion of the T wave, prolongation of the OT interval) are common in anaemic patients both at rest and during exercise.

Patients with AH and anaemia have been reported in the literature to have higher mean daily and nocturnal systolic blood pressure and a poorer reduction in nocturnal systolic blood pressure than those with normal haemoglobin levels. Anaemic patients also tended to have higher mean daily diastolic blood pressure. Patients with isolated systolic hypertension and left ventricular hypertrophy with concomitant haemoglobin reduction have increased cardiovascular mortality and acute cerebrovascular events.

It has also been found that haemoglobin levels correlate strongly with electrocardiogram changes. Electrocardiographic repolarisation changes (BT segment depression, inversion of the T wave, prolongation of the OT interval) are common in anaemic patients both at rest and during exercise.

Lengthened OT interval is a predictor of ventricular arrhythmias and sudden death. Hypoxia and impaired oxygen delivery in anaemic patients may cause myocardial repolarisation. Studies have suggested that anaemia, macrocytosis and anisocytosis correlate with prolongation of the OT interval in patients with arterial hypertension and may be considered in the risk of sudden death.

Thus, anaemia is an independent predictor of cardiovascular disease and associated adverse outcomes.

Anaemia is clinically manifested by anaemic and sideropenic syndromes.

Non-specific anemic syndrome caused by hemic hypoxia of organs and tissues is characterized by unmotivated weakness, rapid fatigability, dizziness, syncope and pre-syncope, dyspnea and palpitations at light physical activity, increased irritability and tearfulness. Objective examination reveals pale skin and visible mucous membranes, tendency to lower blood pressure, tachycardia, functional systolic murmur over heart.

Sideropenic syndrome due to tissue iron deficiency, often detectable even when hemoglobin levels are normal, leads to a decrease in the activity of many enzymes that comprise iron (cytochrome oxidase, peroxidase, succinate dehydrogenase, etc.). This leads to the very characteristic symptoms of perversion of taste (picachLorotica) and smell. Persons with iron deficiency have a craving for chalk, charcoal, clay, sand, raw dough, minced meat, grits, ice and a taste for unpleasant smells (petrol, acetone, varnish, paint, shoe polish, etc.). In addition, patients with iron deficiency show marked muscle weakness and fatigue, muscle atrophy and reduced muscle strength due to deficiency of myoglobin and tissue respiratory enzymes. Objective examination reveals dry skin, thinning, brittleness and transverse striation of nails, koilonychia, angular stomatitis, glossitis ("varnished" tongue), as well as atrophic changes in the mucosa of the esophagus (sideropenic dysphagia), stomach and intestine (atrophic gastritis, enteritis). There may be an increase in body temperature to subfebrile levels ("sideropenic subfebrileitis")

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TREATMENT

The aim of the treatment of WHD is to eliminate iron deficiency and restore iron stores in the body. This can only be done by eliminating the underlying cause of AF and at the same time compensating for the iron deficiency in the body.

The basic principles of treating GIHD are:

1. It is not possible to compensate for iron deficiency without iron medication.
2. Treatment of ASD should be predominantly with oral iron preparations.
3. Treatment of ASD should not be discontinued once haemoglobin levels are normalized.
4. Haemotransfusions for ARF should be administered strictly on a life-saving basis.

Diet for iron deficiency

From the current understanding of the mechanisms of iron absorption in food, the administration of a diet cannot be considered a valid method of correcting iron deficiency. The bulk of iron (~90%) is absorbed in the duodenum and the rest in the upper jejunum. In iron deficiency conditions the absorptive surface of the small intestine is increased. Iron is absorbed in two forms:

- Iron in haemic form (10%), whose sources are haemoglobin and myoglobin in animal products such as meat, fish, poultry, liver and blood;

Non-heme iron (90%), which is found in plant foods such as vegetables, fruit, cereals and milk.

The majority of dietary iron intake is in the non-heme form. The bioavailability of iron from cereals, legumes, tubers, vegetables and fruits is much lower than that from heme compounds and depends largely on factors that inhibit or potentiate intestinal ferroabsorption.

It should also be noted that consumption of meat, liver and fish products increases iron absorption from fruit and vegetables. However, in general, a complete and balanced diet can only 'cover' the physiological need for iron, but not eliminate iron deficiency.

CONCLUSION

Anaemia correlates with worsening prognosis in patients with cardiovascular pathology and is an urgent therapeutic problem of modern medicine.

Cardiology patients require timely diagnosis and treatment of anaemic syndrome, given that up to 95% of patients do not receive adequate therapy. This syndrome requires adequate correction of the identified abnormalities in the specific clinical situation and with the use of iron-containing drugs. The question of individual therapy of anaemia in long-term cardiovascular disease remains open. The accumulated evidence base in these patients on the treatment of anaemia with intravenous iron and/or erythropoietins does not allow an unequivocal determination of the appropriateness and safety of this use.

Until results of large studies are available, oral iron supplementation of patients with mild to moderate GAD is considered to be the best approach, whereas a combination of intravenous iron and erythropoietin may be considered in patients with severe anaemia to allow dose reduction and side-effect reduction.

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