ANIUM AND ITS SPECIFIC CHARACTERISTICS IN PATIENTS WITH CARDIOVASCULAR DISEASES

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ANNOTATION: Today, inflammatory processes occur in various organs as well as the heart, affecting the heart's 3-layer histological systems, affecting myocardial function. In addition, blood rheology and hemoglobin factors are considered to be one of the main causes of myocardial infarction.

Keywords: erythropoiesis, anemia, hemoglobin, C-reactive protein.

Relevance. Anemia is a well-known and independent predictor of adverse cardiovascular prognosis. Anemia can be both a manifestation of an independent disease due to various reasons (erythropoiesis disorders, deficiency of vitamin B12, folic acid, iron, etc.), and with a syndrome associated with the underlying disease. In both cases, anemia can be regarded as a comorbid condition that worsens life prognosis and considered as a new object for correction in HF.

According to the WHO definition, anemia is a decrease in hemoglobin (Hb) levels to <13 g / l in men and to <12 g / l in women. The relationship between mortality and Hb levels is nonlinear; most; increased risk is expressed, with a low level of IIb [4]. In the Val-HeFT study, the risk of death was almost the same in the upper 2 quartiles of level IIb (13.7 to <14.7 g / L) and decreased in the lower 2 quartiles (<12.8 and 12.8 to < 13.7 g / l). Several studies have reported a J-shaped relationship between 1 lb; and mortality in the population of persons without concomitant diseases, with ischemic heart disease, acute coronary syndromes [7] and chronic heart failure [1,2]. The risk of death was lowest with Hb levels from 13 to 16 g%, it increased with values outside of the designated range. These results explain fears that excessive increases in Hb levels may be associated with increased mortality.

While the predictive value of anemia in chronic heart failure and acute heart failure is clear enough, there are no clear recommendations for treatment that improves outcomes. In the presence of obvious deficiencies (low levels of iron, folic acid, vitamin B12), replacement therapy is necessary. Identification of anemia of "chronic conditions" in chronic renal failure and chronic heart failure associated with erythropoietin deficiency or resistance to it, it is possible to prescribe erythropoietin with the achievement of target Hb values not higher than 13.5 g / l.

One of the mechanisms of anemia in acute coronary syndrome with acute heart failure, in its absence before the onset of an acute condition, is blood loss, like. complication of the use of antithrombotics. With significant acute blood loss, blood transfusion is indicated.

Contrary to the prevailing opinion about the worsening prognosis in chronic heart failure during blood transfusion due to anemia, in the Heart Failure Survey in Israel (HFSIS) study of 2335 patients hospitalized due to acute heart failure due to various diseases, including acute coronary syndrome, encouraging results were obtained. In 166 cases, blood transfusion was performed, and when analyzing the outcomes, mortality in patients who received the transfusion procedure during the hospital period was lower (8.7% vs 14.6%, p = 0.20) and remained lower after 30 days (9.7% vs 18.4%, p = 0.08), after a year (38.8% vs 42.7%, p = 0.59) and after 4 years (72.8% vs 76.7).

Thus, there is no final unambiguous answer to the question of whether anemia, which can affect the prognosis of patients with heart failure, is a cause or a marker of an unfavorable clinical outcome. In this regard, the problem of anemia in acute coronary syndrome with acute heart failure remains relevant and requires further study. The importance of markers of inflammation in various types of myocardial dysfunction. It is now well known that the main pathogenetic mechanism leading to the development of acute coronary syndrome is atherothrombosis [1]. The role of coronary artery thrombosis in the pathogenesis of myocardial infarction has been noted by many Russian and foreign authors [3].
In addition, the inflammatory theory of atherosclerosis has become widespread. The data of numerous studies support the direct involvement of local and systemic inflammation in the formation and progression of atherosclerosis and its complications. It was found that this disease, as a rule, is accompanied by signs of a chronic inflammatory reaction (an increase in the serum of patients in the level of C-reactive protein, fibrinogen, plasminogen, proinflammatory cytokines, total leukocyte count) [6].

In clinical practice, to assess the activity of inflammatory processes, the determination of the number of leukocytes in the blood is widely used; in the plasma, the level of C-reactive protein (CPB), fibrinogen, and amyloid cytokines is determined recently. With the use of highly sensitive methods for determining SRV, a stable relationship between the initial level of SRV and cardiovascular events has been demonstrated [3]. The level of C-reactive protein and fibrinogen is increased in patients with coronary artery disease [4] and peripheral vascular diseases compared with healthy individuals. The degree of their increase correlates with the severity of the disease [1]. Large epidemiological studies have shown that a small increase in the concentration of C-reactive protein in healthy individuals is associated with the risk of MI and acute cerebrovascular accident [2]. By the concentration of C-reactive protein, one can indirectly judge the level of interleukin-6 (IL-6), which is the main mediator of the synthesis of acute-phase proteins. [6] found that the level of C-reactive protein increased in patients with unstable angina pectoris regardless of damage to cardiomyocytes, and a high level of C-reactive protein on admission to the hospital (> 3.0 mg / L) was a predictor of a poor prognosis in these patients. The determination of C-reactive protein after acute infection should be avoided, since its level remains elevated for 2-3 weeks, and it should not be used as a marker of cardiovascular diseases in patients with chronic inflammatory diseases. The results of various studies show that the inflammatory process persists despite the stabilization and relief of clinical symptoms. A persistent increase in the concentration of C-reactive protein after an episode of unstable angina pectoris is associated with readmission for recurrence of the disease [7].

Thus, at present it can be argued that inflammation, local and systemic, plays an important role in the pathogenesis of myocardial infarction. The intensity of the inflammatory response determines the clinical outcome, and a high concentration of inflammatory markers may have an independent prognostic value in patients with myocardial infarction. Moreover, a persistent inflammatory reaction persists for a long time after the ischemic event has subsided and causes subsequent cardiovascular complications. However, some questions related to the role of inflammation in the development of myocardial dysfunction in myocardial infarction have not been resolved to date.

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