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IMMUNOREACTIVITY DISORDER IN CHRONIC HEART FAILURE OF VARIOUS ETIOLOGIES

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Abstract
Chronic heart failure (CHF) is a complex clinical syndrome. CHF is the outcome of many cardiovascular diseases, both inflammatory and non-inflammatory in nature, and in most cases leads to permanent disability of patients.
Statistics show that over the past 20 years, the prevalence of CHF in developed countries has increased significantly, reaching epidemic levels in some of them, more than 5 million people suffer from heart failure in the United States, 1.5 million of them have its high (III-IV) functional class according to the NYHA classification.
Epidemiological data of recent years have revealed such an alarming fact that decompensation of CHF caused hospitalizations to hospitals with cardiology departments in almost every second patient (49%), and CHF appeared in the diagnosis in 92% of patients hospitalized in such hospitals.

Keywords: angiotensin converting enzyme inhibitors, coronary heart disease, left ventricle, flow-dependent vasodilation, C-reactive protein, ejection fraction, functional class.

Introduction

However, the mechanisms of inflammation development involving cytokines have not been disclosed in detail, and the features of the relationship of inflammatory mediators with other parts of the immune system in CHF of various etiologies have not been established. The most common cause of CHF is coronary heart disease. The study of the immune mechanisms of CHF in coronary heart disease requires comparison with other diseases with pronounced immune disorders that lead to the development of heart failure. Such a disease is dilated cardiomyopathy (DCMP). In DCMP, most of the cases of which are included in the group of inflammatory cardiomyopathy, the autoimmune mechanism is the main one in the pathogenesis of the disease. Inflammatory myocardial lesions in DCMP in most cases result in a violation of the contractile function of the heart and the development of cardiomyopathy.
Thus, due to the presence of changes in the immune mechanisms of inflammation in these nosologies leading to the development of CHF, it seems relevant to give a comparative

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description of the systemic inflammatory and autoimmune response in patients with CHF with coronary heart disease and DCMP, as well as the possibility of using statins to correct the inflammatory process.

Despite the development of special recommendations for the treatment of patients with CHF, the total number of hospitalizations and the frequency of early readmissions in this condition has not decreased, but tends to increase. All this leads to the search for new methods of diagnosis and therapy of CHF. According to a number of researchers [Mustafina D.M., 2001, El Sherif W.T., El Tooney L.F., Meki A.R., Abdel Moneim A., 2005] immune changes can play a key role in the pathogenesis of CHF. But in order to understand the contribution of immune mechanisms to the development of complications in coronary heart disease, it is necessary to compare this nosology with each other, where immune disorders play a key role.

The available data suggest that cytokines play an important role in the pathogenesis of inflammation in CHF of ischemic and non-ischemic etiology. However, the mechanisms of inflammation development involving cytokines have not been disclosed, and the features of the relationship of inflammatory mediators with other parts of the immune system in CHF of various etiologies have not been established.

A number of authors have proven the association of elevated IL-18 levels with a severe clinical course of the disease: increased CHF and decompensation in coronary heart disease [Mustafina D.M., 2001]. With an increase in the functional class (FC) of CHF (NYHA) [Koller-Strametz J., Pacher R., Frey B., Kos T. et al., 2003], as well as with impaired diastolic heart function [Mann D.L., 2001], an increase in the level of IL-1, sIL-2R, TNF-a was observed IL-6, and high levels of TNF-a were associated with the progression of CHF and a deterioration in the quality of life [El Sherif W.T., El Tooney L.F., Meki A.R., Abdel Moneim A., 2005]. A high concentration of IL-6 was found to be associated with impaired contractile function of the heart in CHF [Satoh M, Nakamura M, Akatsu T et al., 2005]. It was found that cytokines IL-1, TNF-a enhance the production of the MCP-1 protein, which causes the migration of monocytes into the intima of blood vessels, and thereby contribute to the development of atherosclerosis. It was proved that high levels of IL-12, IL-18, and IFN-y in experimental animals contributed to the development of atherosclerosis, and blockade of these cytokines reduced the severity of atherosclerosis by 15-69%.

Thus, due to the presence of changes in the immune mechanisms of inflammation in these nosologies leading to the development of CHF, it seems relevant to give a comparative description of the systemic inflammatory and autoimmune response in patients with CHF of various etiologies, as well as the possibility of using statins to correct the inflammatory process.

The purpose of the work. To study the role of humoral factors of inflammation and endothelial dysfunction in the development of chronic heart failure of various etiologies.

Materials and methods of research. A screening examination of 50 patients was conducted. As a result of the screening, 27 patients with CHF-IV FC (NYHA), which complicated the course of coronary heart disease and DCMP, were included in the study.

Markers of endothelial and left ventricular dysfunction and immune activation were studied in 23 patients with CHD stage I-IIB and I-IV FC according to NYHA.

Of these, 21 (95%) men and 2 (5%) women aged 23-64 years. The average age is 48±8 years.

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FC I had 10 (23%) people, FC II - 10 (23%), FC III - 11 (26%), FC IV - 12 (28%).

The study also included 44 patients with DCMP stages I-IIB and I-I FC CHF. Among them, 42 men (95%) and 2 women (5%) aged 22 to 61 years old. The average age is 43±10 years. There were 10 (23%) people with FC I, 9 (20%) with FC II, 13 (30%) with FC III, 12 (27%) with FC IV.

General clinical examination of patients with coronary heart disease+PIX and CHD+AH with heart failure FKI-IV (NYHA). Determination of markers of immune inflammation of proinflammatory cytokines-IL-6, TNF α and anti-inflammatory cytokine IL-10 in blood serum by solid-phase enzyme immunoassay in patients with coronary heart disease+PEAKS and CHD+AH complicated by heart failure FC I-IV (NYHA). A comparative analysis of the relationship of inflammatory markers in patients with CHF of various etiologies (CHD + PIC, CHD + AH).

Investigation of the dynamics of humoral inflammatory and autoimmune factors in patients with coronary heart disease+PEAKS and CHD+AH complicated by heart failure FC I-IV (NYHA) on the background of standard therapy and on the background of statin treatment. Statistical processing of the received material.

The results of the study. In patients with coronary heart disease, a violation of the vasoregulatory function of the endothelium, a decrease in the contractile function of the heart was revealed.

In the CHD group, 32 (74%) patients had sinus rhythm, 11 (26%) patients had rhythm disturbances of the type of atrial fibrillation. Higher levels of sIL-2R, CRP, and IL-18 were recorded in patients with atrial fibrillation, which allows us to assume the involvement of inflammation in the genesis of atrial fibrillation.

The analysis of CHF FC (NYHA) showed a statistically significant increase in the level of NT-proBNP, CRP and a decrease in the ejection fraction in patients III-IV CHF FC, which indicates the relationship of the inflammatory process with the severity of heart failure.

In the CHD group, there was a direct correlation of CHF FC with NT-proBNP (p=0.77), with CRP (p=0.72) and the reverse - with PV (p=-0.49). The level of CRP in patients with CHD was 1.98 (1.02; 4.54) g/L. The rate of CRP < 3 g/l is accepted as the norm. Patients with coronary heart disease were divided into 2 subgroups: with a level of CRP < 3 g/l and CRP > 3 g/l (Figure 2). CRP was elevated in 17 (39.5%) patients and had a value of 5.60 (4.08; 7.96) g/L. The analysis revealed that the groups with the level of CRP < 3 g/l and CRP > 3 g/l had statistically significant differences in the severity of CHF (p=0.008).

It was found that the groups with CRP levels > 3 g/l and CRP < 3 g/l differed statistically significantly in terms of sIL-2R levels, as well as in some echocardiographic parameters.

As can be seen from, in patients with CRP greater than 3 g/l, the sIL-2R content was statistically significantly higher and there was a more pronounced violation of systolic myocardial function. And also, as a result of the correlation analysis, it was revealed that in the group of coronary heart disease the level of

CRP was statistically significantly correlated with the severity of heart failure according to FC (NYHA) (p=0.72) and the level of NT-proBNP (p=0.45), with sIL-2R (p=0.36), with IL-8 (p=0.31), with IL-6 (p=0.46), with the level of NO metabolites (p=0.39) and with systolic heart function: CSR (p=0.48), CSR (p=0.41), CDR (p=0.38). An inverse correlation

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was found with the ejection fraction (p=-0.43). Correlations of IL-8 with the level of blood leukocytes (p=0.31), with NYHA FC severity (p=0.32), with diastolic heart function Decel time (p=-0.37). In coronary heart disease, elevated sIL-2R levels directly correlated with IgM levels (p=0.32), with CRP (p=0.36). IL-18 was associated with IFN-y concentration (p=0.36), with flow-dependent vasodilation of blood vessels (p=-0.31) and with Decel time (p=-0.39) The level of nitric oxide metabolites was directly correlated with the level of CRP (p=0.39), with NT-proBNP (p=0.38), with the severity of heart failure according to CHF FC (NYHA) (p=0.38), with systolic heart function: CSR (p=0.31), CSR (p=0.40), BWW (p=0.39).

The data obtained indicate that the severity of heart failure, impaired systolic and diastolic heart function, and endothelial dysfunction are associated with the inflammatory process. In the IHD group, detectable levels of IL-10 were observed in 3 (7%) people (0.62 pg/ml, 1.57 pg/ml, 11.36 pg/ml). Patients with coronary heart disease according to the median NT-proBNP (725 pg/ml) were divided into subgroups.

In subgroup 2 (NT-proBNP > 725 pg/ml), patients had statistically high levels of CRP, IL-6 (Figure 3), NO, an increase in CSR, a decrease in PV ($p<0.05$), a change in the rate of transmittal flows in early and late diastole (E, A), and their ratios (E/A), compared with 1 subgroup (NT-proBNP < 725 pg/ml).

Thus, the data indicate a connection between the severity of the inflammatory process and the severity of heart failure. In the first subgroup of patients, direct correlations were found between IL-18 and IFN-y (p=0.49). IL-18 levels negatively correlated with flow-dependent vasodilation (p=-0.44) and diastolic heart function Decel time (p=0.48). The increased IgA level was directly correlated with NT-proBNP (p=0.42) and IgG (p=0.50). The content of CRP correlated directly with the severity of CHF FC (p=0.59), CSR (p=0.47), and negatively with the level of PV (p=-0.51). IgM level correlated with the level of nitric oxide metabolites (p=-0.47) and with the content of IL-6 (p=0.72).

Endothelin concentration negatively correlated with the level of IL-8 (p=-0.56). High IL-8 content directly correlated with the content of leukocytes (p=0.49). In the second subgroup of patients Correlations were found between elevated IL-6 levels and IgG concentrations (p=0.72). A direct correlation between the level of CRP and severity has been revealed CHF FC (NYHA) (p=0.55), with systolic heart function CSR (p=0.46).

The level of IFN-y was directly correlated with BWW (p=0.55), with Decel time (p=-.53). IgM concentration was directly correlated with sIL-2R (p=0.54). IL-8 levels correlated with the severity of CHF FC (NYHA) (p=0.55), with diastolic heart function: Decel time (p=-0.59).

The following correlations were found in the CHD group with the absence of autoantibodies. The content of SIL-2R directly correlated with the content of IgM (p=0.40). The level of CRP correlated with PV (p=-0.43), with CSR (p=0.50), with CSR (p=0.41), with CDR (p=0.43), with flow-dependent dilation (p=-0.50). The results obtained indicate the presence of a more pronounced inflammatory process in the presence of an autoimmune component. The severity of heart failure was associated with the severity of the autoimmune component of inflammation.

Thus, in CHD, the association of inflammatory mediators with the severity of heart failure (CRP, IL-8 with NT-proBNP), with endothelial dysfunction, and with impaired heart

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function was revealed. Patients with atrial fibrillation type arrhythmia have a higher level of proinflammatory factors (IL-18, sIL-2R, CRP). The presence of an autoimmune component in coronary heart disease increases the severity of heart failure and the severity of the inflammatory process.

Conclusions

The severity of endothelial dysfunction is associated with a systemic inflammatory response. In patients with coronary heart disease, the level of CRP correlated with the level of nitric oxide metabolites, IL-18 correlated with flow-dependent vascular dilation. In dilated cardiomyopathy, CRP correlated with the level of nitric oxide and endothelin metabolites, IL-18 with endothelin levels.

The activity of the systemic inflammatory response is associated with impaired systolic and diastolic heart function. In patients with coronary heart disease, CRP levels correlated with systolic heart function: CSR, CSR, CDR, PV; IL-8 with diastolic function Decel time; IL-18 correlated with diastolic function Decel time. In DCMP, the level of IFN-y correlated with the diastolic function of Decel time; an increase in IL-6 levels was revealed in the restrictive type of diastolic dysfunction.

In DCMP, the severity of heart failure is associated with the severity of the humoral autoimmune component. Statins improve the clinical course of the disease: they reduce the functional class of CHF, the degree of cardiac decompensation in patients with coronary heart disease and DCMP, and improve systolic heart function in DCMP. At the same time, there was no improvement in endothelial function in CHF and in systolic and diastolic heart function in patients with coronary heart disease.

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