THE DYNAMICS OF PRO-INFLAMMATORY CYTOKINES IN PATIENTS WITH ANEMIA AND CHRONIC HEART FAILURE

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Recent studies indicate that patients with chronic heart failure experience a range of neurohumoral responses, including the activation of inflammatory cytokines such as tumor necrosis factor-alpha, interleukin-1, and interleukin-6. These inflammatory processes contribute to the development of anemia of chronic disease. The identification of hepcidin, a key regulator of iron homeostasis in chronic heart failure, has significantly advanced our understanding of the pathophysiological link between inflammation and anemia. Elucidating the interactions between inflammatory mediators and targeting the suppression of pathogenic pathways are essential steps in the development of more effective therapeutic strategies.

Purpose of the study

To assess the role of inflammatory cytokines in the pathogenesis of anemia associated with chronic diseases in patients with chronic heart failure.

Materials and methods

The research included 115 patients with chronic heart failure of the II and III functional class of stabil angina pectoris, based on ischemic heart disease. Their ages ranged from 50 to 70 years, with an average age of 64.6 ± 4.9 years. All patients were treated in a hospital setting and later observed on an outpatient basis, being divided into two: main and control groups. The main group consisted of 35 patients with chronic heart failure complicated by chronic disease-related anemia and 40 patients with iron deficiency anemia, while the control group included 40 patients with chronic heart failure but without anemia. Each group was further subdivided based on the functional class of chronic heart failure into two smaller groups (II, III functional classes). The main group received 200 mg of iron III hydroxide sucrose complex (Venofer) intravenously as an antianemic treatment and standard chronic heart failure treatment, while the control group received only the standard chronic heart failure treatment.

Results

The results of the study showed that in the control group, the patients with chronic heart failure without anemia had the following cytokine levels in their blood plasma for II-III functional classes: interleukin-1 levels ranged from 16.4 ± 0.72 to 18.6 ± 1.93 ng/ml, interleukin-6 levels ranged from 17.2 ± 1.78 to 18.9 ± 1.3 ng/ml, and alpha- tumor necrosis factor levels ranged from 15.9 ± 0.72 to 17.4 ± 1.93 ng/ml.

In the main group, patients with chronic heart failure complicated by chronic disease-related anemia had the following cytokine levels: interleukin-1 levels ranged from 18.2 ± 0.72 to 21.1 ± 1.93 ng/ml, interleukin-6 levels ranged from 26.6 ± 1.7 to 29.7 ± 1.3 ng/ml, and α - tumor necrosis factor levels ranged from 20.2 ± 0.72 to 24.7 ± 1.93 ng/ml. In the main group of patients with chronic disease-related anemia and chronic heart failure (II functional class), interleukin-1 levels decreased from 18.2 to 16.2 ng/ml ($P > 0.05$), interleukin-6 levels decreased from 26.6 to 19.7 ng/ml ($P < 0.05$), and α - tumor necrosis factor levels decreased from 20.2 to 15.6 ng/ml ($P < 0.05$). In the III functional class group, interleukin-1 levels decreased from 21.1 to 18.3 ng/ml ($P > 0.05$), interleukin-6 levels decreased from 29.7 to 22.8 ng/ml ($P > 0.05$), and α - tumor necrosis factor levels decreased from 24.7 to 19.3 ng/ml ($P > 0.05$).

Conclusion

As mentioned above, the pro-inflammatory cytokin levels in the blood plasma were higher in both groups of patients with and without anemia in chronic heart failure. These cytokine levels were especially higher in patients with severe functional classes (II and III) of chronic heart failure, certainly in those with chronic disease-related anemia. This may be attributed to the activation of local hormonal pathways triggered by systemic hypoxia within the body.