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Cytokine Status of Patients with Chronic Viral Hepatitis

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Abstract: The study of immunological aspects of the course of chronic viral hepatitis is relevant. The problem of chronic viral hepatitis for many states and its medical and social significance is currently increasing. Early diagnosis and prevention of complications of chronic viral hepatitis can reduce morbidity and disability.

The author has developed recommendations for the early diagnosis and prognosis of complications of chronic viral hepatitis, taking into account the immune status, which contribute to improving the quality of preventive care for infectious diseases, therapists, followed by a decrease in morbidity and disability.

Keywords: viral hepatitis, immune status, prognosis, immunity, liver.

Hepatitis viruses (hepatotropic viruses) predominate among the non-toxic etiological factors leading to the development of chronic liver diseases (CKD) [1,2].

The work in the field of virology, immunology, molecular biology and genetics carried out over the past few decades has made it possible to identify the main epidemiological, clinical and morphological manifestations of infections caused by hepatotropic viruses, as well as to develop etiotropic therapy [3].

However, the problem of viral liver diseases currently remains extremely relevant due to the widespread spread of its main forms and its enormous medical and social significance. In all developed countries, there is a rapid increase in the number of patients with chronic liver disease in different age groups. Currently, antibodies to hepatitis B virus (HBV) antigens are detected in about 2 billion people. people around the world, among them 300-400 million have been diagnosed with chronic hepatitis B [4,5].

The purpose of the study: to develop a method for diagnosing and predicting complications of chronic viral hepatitis, taking into account the immune status.

Materials and methods: A total of 64 patients were included in the study, of which 30 patients (group 1) aged 38 to 79 years with chronic viral hepatitis C (HCV) and 34 patients (group 2) aged 31 to 67 years with chronic viral hepatitis B (HCV). At the same time, the average age of patients was y patients of group 1 54.6± 1.89 years, y patients of group 2 45.4± 1.44 years. The control group consisted of 32 healthy people aged 35 to 54 years (average age 44.3 ± 0.95).

Results and discussion. The immunological parameters of patients' blood, depending on the form of chronic viral hepatitis, differ slightly.

The study found a decrease in IL-6 levels by 2.15 times in y patients of group 1 ($p<0.05$), 1.6 times in y patients of group 2 ($p<0.05$), (Fig.1).

The results of the study of IL-8 levels showed a tendency to increase in y patients of the 1st group and a slight decrease in y patients of the 2nd group. The data obtained indicate the

likelihood of bacterial and/or exacerbation of viral infection against the background of HCV, Fig.1.

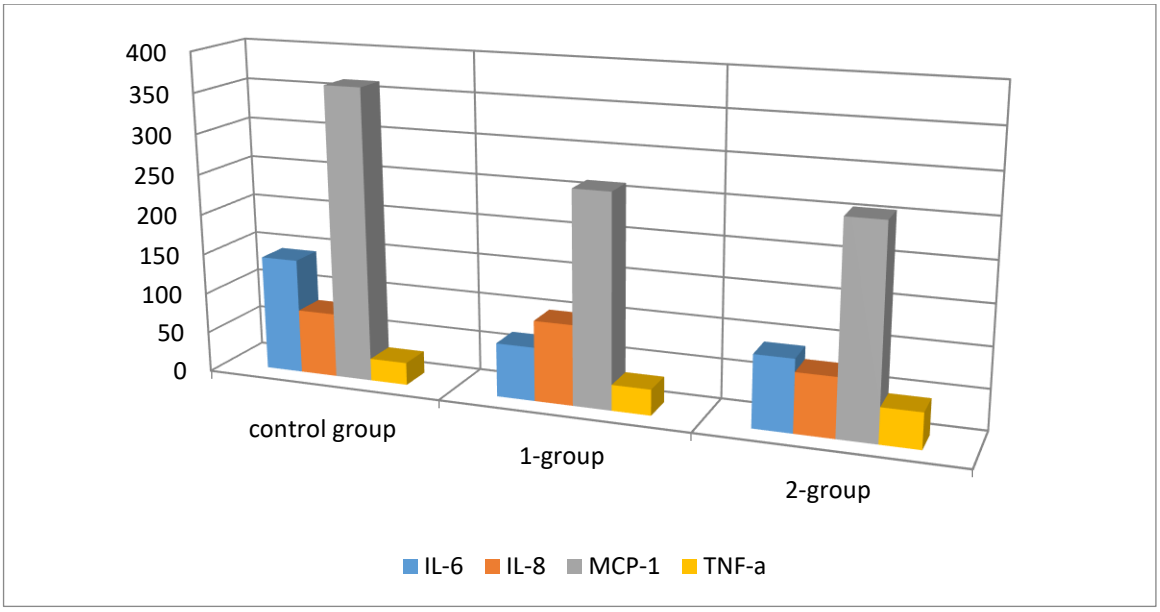

Figure 1. Cytokines in chronic hepatitis

One of the main chemokines for monocytes/macrophages and activated T-lymphocytes is monocyte chemotactic protein-1 (MCP-1). MCP-1 was first identified as a product of secretion of monocytic leukemic cells stimulated by lipopolysaccharide, as well as mononuclear cells of peripheral blood. MCP-1 belongs to the class of CC chemokines and is a powerful chemoattractant of monocytes/macrophages. MCP-1 is not only a chemoattractant that ensures the migration and extravasation of mononuclear cells into the focus of inflammation, but also a mediator of inflammation, while activating resident cells. Human MCP-1 is a protein consisting of 76 amino acids. MCP-1 is produced by many cell types, including mononuclear cells, mast cells, T cells, osteoblasts, fibroblasts, endothelial cells, bone marrow cells, epithelial cells, astrocytes. The synthesis of MCP-1 is induced by IL-1β, α-TNF, γ-INF, IL-6, IL-4. Under the influence of MCP-1, proliferation of vascular smooth muscle cells also occurs with their secretion of proinflammatory cytokines, which contribute to the progression of the disease due to vascular damage [1].

As a result, a statistically significant decrease in MCP-1 was found to 262.1±15.2 pkg/ml ($p<0.05$) y large 1st group, to 255.8±17.9 pkg/ml ($p<0.05$) y large 2nd group, against the control values - 362.9± 25.3pkg/ml, Fig.2.

Taking into account the data from the literature sources, the decrease in MCP-1 in patients with CVH against the background of comorbidity (especially when combined with CVH with CVD) testifies from one position to the chronization of the pathological process, from the other position it testifies to a high risk of complications and fatal outcomes, which requires correction of therapy for both the underlying and concomitant diseases and shows the importance of adherence to the principle of continuity in the administration of such patients together with infectious disease specialists, cardiologists and therapists.

The study of the interferon status of the patients selected for the study showed a statistically significant increase in the level of IFNa to 14.34 ± 0.65 pg/ml in patients of the 2nd group versus the control-12.24 ± 0.64 pg/ml ($p<0.05$). At the same time, the given interferon of y patients of group 1 was at the level of control values, Fig.5.

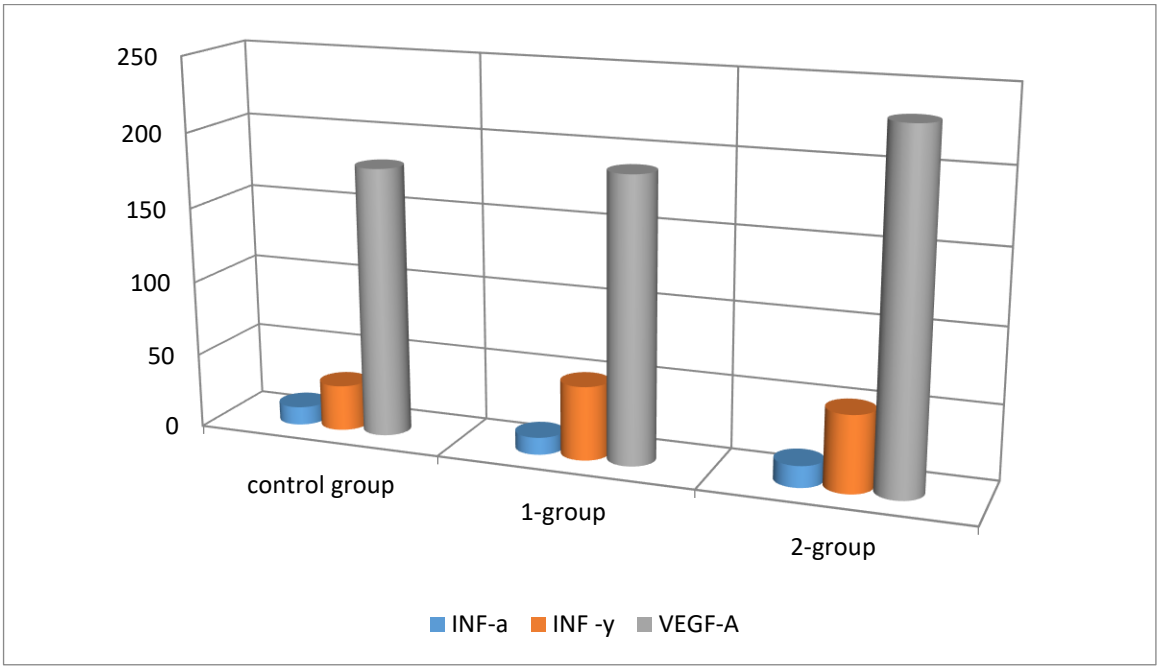

Figure 2. Interferons and vascular endothelin factor in chronic hepatitis

The analysis of IFN synthesis showed a significant increase in its effect in patients of both groups 1 and 2: up to 49.43± 3.73 pg/ml and 51.16± 3.48 pg/ml, respectively, against the control-30.62± 2.62 pg/ml, $p<0.05$. Consequently, an increase in the synthesis of interferons shows the activation of the protective mechanisms of the body in CVD.

The study found a significant increase in TNFa y levels in patients of group 2 by 1.58 times ($p<0.05$), Fig.2.

The obtained result shows viremia and a higher risk of thrombosis in HCV.

Taking into account the pathogenetic mechanisms of the development of HCV, the level of vascular endothelial growth factor (VEGF) in patients of this category was determined. At the same time, there was no significant increase in the level of VEGF in both study groups to 189.1±24.9 pg/ml in y patients of group 1, to 231.2±38.54 pg/ml in y patients of group 2, with respect to control indicators - 180.26±35.9pg/ml, Fig.2.

As a result, data are obtained, indicating an increase in the process of inflammation of the vascular wall in CVI and a direct dependence of the activity of inflammation on the form of CVI.

Thus, the mechanism of development of CVH in comorbidity is closely interrelated with the synthesis of cytokines and interferons. The degree and severity of HCV directly depends on the degree of damage to liver tissue and the state of metabolism in the body.

For early detection of the risk of developing CVI in patients with comorbid conditions, regular examination and study of proinflammatory markers and interferences for the prevention of complications of CVI are necessary.

Conclusions

1. A 2.15-fold decrease in IL-6 levels was found in patients with chronic viral hepatitis C ($p<0.05$), 1.6-fold in patients with chronic viral hepatitis B ($p<0.05$).
2. A statistically significant decrease in MCP-1 was found in patients with chronic viral hepatitis.
3. An increase in the synthesis of IFNy in chronic viral hepatitis has been established, which shows the activation of the body's defense mechanisms in chronic viral hepatitis.

4. A significant increase in TNFa levels in patients with chronic viral hepatitis B was found to be 1.58 times ($p<0.05$), which confirms viremia and a high risk of thrombosis.

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