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THE COURSE OF CHRONIC VIRAL HEPATITIS C IN IRON DEFICIENCY ANEMIA.

Abstract: *This article reflects on the causes on chronic virus hepatitis C in iron deficiency anemia.*

Keywords: *Chronic hepatitis C, combined antiviral therapy, Iron deficiency anemia, Erythrocytometry*

Chronic hepatitis C is one of the most common human infectious diseases. The use of modern combined antiviral therapy for Chronic hepatitis C makes it possible to achieve complete elimination of the pathogen in many patients, however, the development of undesirable phenomena remains an urgent problem, among which hematological complications occupy a special place. One of the most serious hematological syndromes in patients with Chronic hepatitis C is combined antiviral therapy-associated anemia, the development of which significantly increases the risk of life-threatening conditions in patients. It is assumed that the main cause of anemia, in this case, is the development of hemolysis due to the accumulation of ribavirin metabolites inside erythrocytes, which is known to be one of the components of combined antiviral therapy [3]. Unfortunately, the pathological changes that occur with red blood cells, as well as the features of hemolysis itself, remain practically unexplored.

Iron deficiency anemia accounts for more than 80% of all anemia in adults and more than 90% in children. This disease is manifested by a decrease in the level of hemoglobin and red blood cells in the blood due to a lack of iron in the body. Pregnant women are at risk: 40% of whom suffer from iron deficiency

anemia. The disease not only causes unpleasant symptoms in the form of weakness and decreased performance, but is also accompanied by severe disorders of tissue oxygenation.

The direct cause of iron deficiency anemia is called iron deficiency. This trace element is necessary for the formation of hemoglobin, an oxygen carrier protein contained in red blood cells. At first, the iron deficiency is compensated by the depot, but its own reserves are rapidly depleted. As a result, the formation of red blood cells is disrupted, they become small and change their shape.

All causes of iron deficiency can be divided into 3 groups:

● chronic blood loss in nasal, uterine, gastrointestinal, hemorrhoidal bleeding, as well as in women with copious menstruation

° disturbances in the intake, absorption and transport of iron, which is observed during malabsorption, a decrease in blood protein levels, poor nutrition

● increased need for iron, characteristic of young children and adolescents, pregnant women, cancer patients

The study included 22 patients with chronic hepatitis C who have indications for combined antiviral therapy. HCV 1a genotype was found in 3.1%, 1b - 55.3%, 3a - 28.6% and 2a - 16.1% of people. The level of viral load in 29.9% of patients was <3×105 IU/ml, in 46.9% - 3×105-6×105 IU/ml and in 23.2% >6×105 IU/ml. All subjects were prescribed combined antiviral therapy according to modern international standards. 51.8% of patients received ribavirin in combination with pegylated α-interferons (peg-INF-α), and 48.2% - with "short". combined antiviral therapy was discontinued in the absence of an early virological response and/or the development of severe adverse events in patients. In the course of the work, a number of highly specialized studies were performed on the observed persons. The determination of hemogram parameters was carried out by the method of automatic hematological analysis (Advia 2120i (Siemens)) immediately before the start of

combined antiviral therapy, after 4, 8, 12, 24 and 48 (persons infected with HCV genotype 1) weeks of combined antiviral therapy. Morphological characterization of erythrocytes was performed in a monolayer of peripheral blood smears using the program "Erythrocytometry" and the hardware-software complex "Mekos-C1". The intracellular ultrastructure of PC erythrocytes was studied using transmission electron microscopy on a transmission electron microscope "Tecnai G2 Spirit BioTWIN" (Philips, the Netherlands). To study the role of lipid peroxidation in the pathogenesis of combined antiviral therapy-associated anemia, the observed patients underwent a study of the activity of superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase, as well as the content of malondialdehyde and extraerythrocytic hemoglobin in blood plasma. The serum concentration of endogenous erythropoietin was determined by automatic chemiluminescent immunoassay (IMMULITE 2000, Siemens Healthcare Diagnostics, USA-Germany).

Screening of anti-erythrocyte antibodies was carried out by staging a direct Coombs antiglobulin test using the method of Y. Lapierre et al. (1990), using ID cards "ScanGelTM COOMBS Anti-IgG" (Bio-Rad Laboratories, USA-France). The research methods listed above were carried out twice: before the start and immediately after the completion or forced termination of combined antiviral therapy. In addition, single nucleotide DNA polymorphisms (SNP) of the superoxide dismutase-2 (SOD2) Ala16Val(rs4880) gene, catalase (CAT) -262C/T(rs1001179) gene and glutathione peroxidase-4 (GPX4) 3'UTR,718C/T gene were studied in the subjects by real-time PCR(rs713041). Allele-specific probes of "SNP-Screen" reagent kits (Syntol, RF) were used for SNP genotyping. The final stage was a thorough statistical analysis of the results obtained, which was performed after the complete completion of clinical, laboratory and instrumental monitoring of all patients.

Of the 22 patients with Chronic hepatitis C, 67.9% of people have completely completed the course of CBT. A stable virological effect (SVR) was

achieved in 66.1% of patients, of whom 28.6% were infected with the 1st; 14.3% - with the 2nd and 23.2% - with the 3rd HCV genotypes. Among those receiving peg-IFN-α, the frequency of SVR was 55.2%; those receiving "short" - 77.8%. In patients with Chronic hepatitis C with HCV genotype 1, SVR was observed in 51.6% of cases; with HCV genotypes 2 and 3, in 88.9% and 81.3% of cases, respectively.

When studying the severity of combined antiviral therapy-associated anemia, the classification of the European Society of Medical Oncology was used, according to which mild (Hb 10.0-11.9 g/dl), moderate (Hb 8.0-9.9 g/dl) and severe (Hb<8.0 g/dl) degrees of anemia are distinguished. As a result, it was found that at different stages of combined antiviral therapy, anemia in total developed in 37.5% of patients, with a mild degree noted in 12.5%; moderate - in 19.6% and severe - in 5.4% of patients with Chronic hepatitis C.

Among patients with mild anemia, 85.7% complained of general weakness and fatigue. With the development of moderate anemia, 72.7% of patients with Chronic hepatitis C additionally complained of shortness of breath with little physical exertion. Patients with severe anemia also reported headache, dizziness, tinnitus - 66.7%, palpitations and pain behind the sternum - 33.3%. Objective changes in the form of acrocyanosis, tachycardia, extrasystole, edema of the lower extremities in the evenings were detected exclusively in persons with severe anemia - 66.7 % of patients with Chronic hepatitis C . In 21.4% of patients, starting from week 20 of combined antiviral therapy, the development of splenomegaly was recorded.

Mild anemia after 4 weeks of combined antiviral therapy was recorded in 23.2 %, after 8 weeks - in 30.4 %, after 12 - in 24.1 %, after 24 - in 28.9 % and after 8 (patients with HCV genotype 1) - in 11.8% of patients with chronic hepatitis C. Moderate anemia after 12 weeks of combined antiviral therapy was observed in 5.6% of patients with Chronic hepatitis C, after 24 weeks - in 13.2% and after 48 weeks - in 29.4% of patients. The development of severe anemia was

first detected after 8 weeks of treatment in 1.8% of patients, which was the only reason for the cancellation of combined antiviral therapy in them. In 3.7% of patients, severe anemia was formed 12 weeks after the start of combined antiviral therapy, coinciding with the absence of RVR, as a result of which the reason for discontinuation of therapy was of a "mixed" nature. Correction of anemia was performed with a decrease in Hb <10.0 g/dl. In 2.2% of patients, the dose of ribavirin was gradually reduced to 600 mg/day, 1.3% of patients were treated with recombinant EPO-α as an alternative. In 16.1% of patients with HCV during treatment, the concentration of Hb also decreased <10.0 g/ dl, however, correction of combined antiviral therapy-associated anemia was not carried out, since the corresponding values of Hb were observed in patients either by the time of cancellation of combined antiviral therapy due to the absence of RVR (n=12), or by the time of completion of the full course antiviral therapy (n=24).

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