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PREDICTORS OF ANEMIA OF CHRONIC DISEASE IN PATIENTS WITH VIRAL HEPATITIS B AND C

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Abstract. Many chronic human diseases are accompanied by the so-called anemia of chronic disease (ACD), the key mediator of which is hepcidin, an iron-regulatory polypeptide hormone of the liver. ACB is considered as one of the fundamental reactions of innate immunity directed at increasing the overall resistance of the organism. Recall also that hepcidin has a direct antimicrobial effect by destroying bacterial membranes.

Keywords: Hepcidin, liver hemosiderosis, anemia of chronic diseases, viral hepatitis B and C

Relevance. Anemia is one of the most common syndromes in clinical practice. It is registered in every fourth inhabitant of the planet and almost always has a secondary character [1]. Multifactorial regulation of erythropoiesis makes it difficult to determine the causes of anemia and methods for its correction. this is especially true of infectious diseases, the pathogenesis of anemia in which is still not always clear. In chronic viral liver diseases, anemia is observed in approximately half of patients [2]. The supposed causes of anemia in these patients are the myelosuppressive effect of a viral infection and alcohol, deficiency of iron, vitamin B12 and folic acid, hemodilution, chronic blood loss, etc. [3].

The European School of Hematology, 2006, divides the most common types of anemia into 2 groups - hyporegenerative (with a reticulocyte count less than $50*10^9$) and regenerative (more than $100*10^9$ reticulocytes). hyporegenerative include aplastic anemia, pure red cell aplasia and myelodysplastic syndromes, deficient conditions (iron and vitamin deficiency), bone marrow infiltration (fibrosis), anemia of chronic diseases (with inflammation, etc.), decreased production of erythropoietin.

regenerative anemias are associated with bleeding and hemolysis. This group includes hereditary hemolytic anemia, anemia due to hypersplenism and microangiopathies. Aplastic anemias are rare, but are the most severe diseases with a poor prognosis. Viral infections are capable of inducing a special variant of aplastic anemia, the so-called pure red cell aplasia (RCC). Parvovirus B19 is a typical etiological agent of hkkA; the role of cytomegalovirus, HIV, hepatitis C and B viruses has been noted [1]. Myelosuppressive effects in HCV and HBV infection are known. Thus, 2–5% of cases of aplastic anemia in Europe and 4–10% in the East are associated with acute viral hepatitis. Deep pancytopenias develop both in the icteric period and in early convalescence with normal biochemical parameters [4,5]. however, the pathogenetic mechanisms leading to these reactions are unclear. T-lymphocytes and bone marrow macrophages play an important role in the regulation of hematopoiesis. It is assumed that in the presence of fHoα [2–6] and interferon-γ [4,5] secreted by them, apoptosis of hematopoietic cells is activated. the so-called pure red cell aplasia (PPC). Parvovirus B19 is a typical etiological agent of hkkA; the role of cytomegalovirus, HIV, hepatitis C and B viruses has been noted [1]. Myelosuppressive effects in HCV and HBV infection are known. Thus, 2–5% of cases of aplastic anemia in Europe and 4–10% in the East are associated with acute viral hepatitis. Deep pancytopenias develop both in the icteric period and in early convalescence with normal biochemical parameters [4,5]. however, the pathogenetic mechanisms leading to these reactions are unclear. T-lymphocytes and bone marrow macrophages play an important role in the regulation of hematopoiesis. It is assumed that in the presence of fHoα [2–6] and interferon-γ [4,5] secreted by them, apoptosis of hematopoietic cells is activated. the so-called pure red cell aplasia (PPC). Parvovirus B19 is a typical etiological agent of hkkA; the role of cytomegalovirus, HIV, hepatitis C and B viruses has been noted [1]. Myelosuppressive effects in HCV and HBV infection are known. Thus, 2–5% of cases of aplastic anemia in Europe and

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Iron deficiency is observed in chronic blood loss from erosions of the gastrointestinal tract and varicose veins of the esophagus. however, "deficiency" anemia may be more common with chronic HCV infection than we realize. The spectrum of lesions in hepatitis C is not limited to the liver, its extrahepatic manifestations such as autoimmune thyroiditis, chronic pancreatitis and diabetes mellitus are well known, which can cause vitamin and microelement deficiencies.

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It would be logical to consider anemia in HCV infection as anemia of chronic disease (ACD). The model for the study of ACP are infections lasting more than 1 month - tuberculosis, bacterial endocarditis, osteomyelitis, chronic fungal diseases, as well as cancer and autoimmune diseases - rheumatoid arthritis, systemic lupus erythematosus, systemic vasculitis. It has been determined that chronic inflammation leads to anemia in three different ways: iron deficiency, erythropoietin deficiency, and progenitor cell alteration. it was initially noted that, unlike classical iron deficiency anemia, ACP can be normocytic, characterized by a decrease in serum iron concentration and an increase in ferritin levels, normal or slightly reduced serum iron-binding capacity, and, most importantly, a lack of response to iron therapy [4]. The discovery of the central regulator of iron homeostasis, hepcidin, clarified the pathogenesis of ACP. Hepcidin synthesis occurs in the liver and is induced by inflammatory cytokines. Hepcidin inhibits iron absorption in the duodenum, iron release from macrophages, and transport via ferroportin, which mimics true iron deficiency and reduces its availability to progenitor cells [1]. In the study of ACHB, pro-inflammatory cytokines were identified that can suppress the expression of erythropoietin and its receptor genes [1,3]. Chronic renal failure is also accompanied by inhibition of erythropoietin synthesis, but this occurs only with a decrease in creatinine clearance of less than 30 ml / min. In chronic HCV infection, kidney damage is possible due to cryoglobulinemia or with the development of hepatorenal syndrome. however, cirrhosis is characterized by an increase in the level of erythropoietin and a decrease in the therapeutic effect of exogenous erythropoietin [2]. According to A.G. Rakhmanova [2], anemia in cirrhosis is not fully explained by the deposition of blood in the spleen, since portal decompression or splenectomy do not have a significant effect on the level of red blood cells; an increase in the capture of labeled erythrocytes by the spleen is observed in no more than 20% of patients. But anemia in viral cirrhosis of the liver is not ACHB, which is confirmed by studies

of bone marrow punctate, which reveal an increase in cellularity and the number of reticulocytes [2]. V.G. Radchenko [3] also indicates that cirrhosis is accompanied by anemia of the regenerative type. anemia in cirrhosis is not fully explained by the deposition of blood in the spleen, since portal decompression or splenectomy does not significantly affect the level of red blood cells; an increase in the capture of labeled erythrocytes by the spleen is observed in no more than 20% of patients. But anemia in viral cirrhosis of the liver is not ACHB, which is confirmed by studies of bone marrow punctate, which reveal an increase in cellularity and the number of reticulocytes [2]. V.G. Radchenko [3] also indicates that cirrhosis is accompanied by anemia of the regenerative type. anemia in cirrhosis is not fully explained by the deposition of blood in the spleen, since portal decompression or splenectomy does not significantly affect the level of red blood cells; an increase in the capture of labeled erythrocytes by the spleen is observed in no more than 20% of patients. But anemia in viral cirrhosis of the liver is not ACHB, which is confirmed by studies of bone marrow punctate, which reveal an increase in cellularity and the number of reticulocytes [2]. V.G. Radchenko [3] also indicates that cirrhosis is accompanied by anemia of the regenerative type. which is confirmed by studies of bone marrow punctate, which reveal an increase in cellularity and the number of reticulocytes [2]. V.G. Radchenko [3] also indicates that cirrhosis is accompanied by anemia of the regenerative type. which is confirmed by studies of bone marrow punctate, which reveal an increase in cellularity and the number of reticulocytes [2]. V.G. Radchenko [3] also indicates that cirrhosis is accompanied by anemia of the regenerative type.

Hepcidin mRNA expression in hepatocytes is induced by cytokines (IL-6, IL-1α, TNF-α.) of macrophages and stellate reticuloendotheliocytes exposed to liposaccharides. Hepcidin binds ferroportin (an iron carrier protein from enterocytes, macrophages and hepatocytes into the blood plasma), which leads to the redistribution of iron from a functional to a stable fund with a delay in the release of

the microelement. At the same time, iron accumulates in hepatocytes, macrophages of the bone marrow, spleen and liver - stellate reticuloendotheliocytes. Oversaturation of ferritin (a cellular metal storage protein) is accompanied by the formation of hemosiderin pigment (a morphological marker of iron overload in cells), the presence of deposits of which in hepatocytes and stellate reticuloendotheliocytes is referred to as liver hemosiderosis (HSP). Due to a decrease in the delivery of a microelement to the bone marrow, a relative iron deficiency develops with normal or slightly increased iron reserves. The biological meaning of the process is to limit the availability of iron for the growth of microorganisms and the replication of viruses.[1] Therefore, ACB is considered as one of the fundamental reactions of innate immunity aimed at increasing the overall resistance of the organism. Recall also that hepcidin has a direct antimicrobial effect by destroying bacterial membranes.[2] [1] Therefore, ACB is considered as one of the fundamental reactions of innate immunity aimed at increasing the overall resistance of the organism. Recall also that hepcidin has a direct antimicrobial effect by destroying bacterial membranes.[2] [1] Therefore, ACB is considered as one of the fundamental reactions of innate immunity aimed at increasing the overall resistance of the organism. Recall also that hepcidin has a direct antimicrobial effect by destroying bacterial membranes.[2]

Purpose of the study. To study the most important clinical, laboratory and morphological manifestations of ACHB in chronic viral hepatitis B and C, to evaluate the importance of HSP in the diagnosis and prognostic evaluation of the disease.

Material and methods. We studied 30 cases of chronic hepatitis of viral etiology, including CG-B (16) and CG-C (4). The etiology of the disease was confirmed using serum tests (HBsAg, HBeAg, HBcAb, HBV-DNA, HCV-Ab IgM, HCV-RNA). The state of iron metabolism was assessed by the concentration of hemoglobin (Hb), serum iron (SI), serum ferritin (SF) and the degree of saturation of transferrin with

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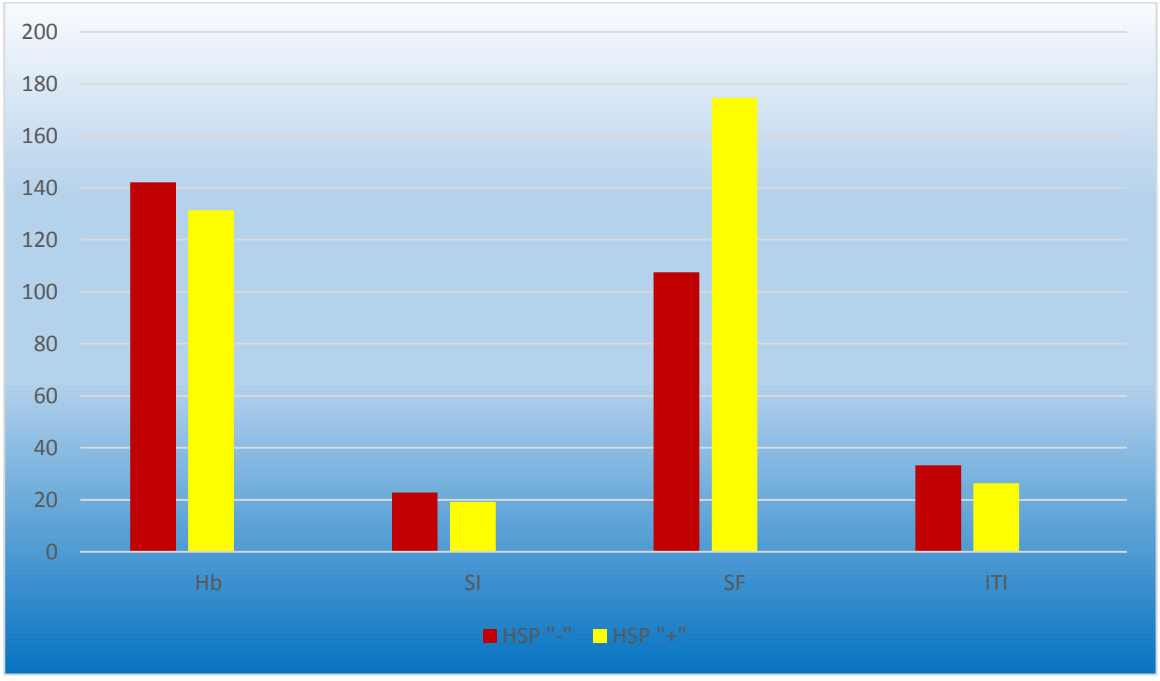
iron (ITI). Patients underwent puncture liver biopsy. Histological sections were stained with hematoxylin and eosin, picrofuchsin, orcein, and for iron using the Perls method. During the morphological study, the activity of the disease and its stage, as well as the presence of HSP, were assessed. To study the association of HSP with clinical and laboratory manifestations of ACP, all cases were divided into 2 groups. The first group (HSP "–") included 13 cases without morphological manifestations of liver iron overload, the second (HSP "+") included 17 cases with this phenomenon. Carried out standard statistical processing of the obtained data.

Results and its discussion. In the first group of observations (HSP "-") the state of iron metabolism was characterized by normal indicators: Hb - 142.1±1.4 g/l; SI - 22.8±0.3 μmol/l; SF - 107.5±9.3 μg/l; ITI – 33.2±0.9%.

In patients of the second group (HSP "+"), a significant ($p<0.05$) decrease in the functional fund of iron was noted for all (except for SF) indicators: Hb - 131.4±1.9 g/l; SI - 19.2±0.2 μmol/l; ITI - 26.3±0.7%.



It was also established that HSP was clearly associated with a higher activity of chronic hepatitis B and C and the degree of fibrosis of the organ tissue. It is in such

cases of the disease, based on information about the pathogenesis of ACHB, that it can be assumed

development and more severe course. This can also explain the significant ($p<0.05$) increase in PS concentration (174.6±9.3 μg/l) in patients of the second group. It is known that this indicator in liver diseases mainly reflects the activity of the process and the severity of the cytolytic syndrome, and not the amount of iron reserves in the body of patients.[5]

Conclusions. Thus, the state of the functional fund of iron, which fully meets the criteria for anemia, was relatively rare in our observations in patients with chronic hepatitis B and C. At the same time, the majority of patients showed a decrease in this fund, which was clearly combined with HSP. Analysis of the obtained results and information about the pathogenesis of ACHB allows us to reasonably suggest that the decrease in the functional fund of iron and the emerging overload of the liver with a microelement in chronic hepatitis B and C are pathogenetically associated mainly with ACHB. Therefore, HSP can be considered as a morphological marker of ACHB in chronic hepatitis B and C. Since the presence of this marker was associated with higher disease activity and the degree of liver fibrosis,

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