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Characteristics of Anemia in Patients with Chronic Hepatitis C and Ways of Their Correction

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Abstract:
Objective – to characterize changes in the blood system that occur during antiviral therapy for chronic hepatitis C (HCV) and modern methods for their correction. The presented scientific review provides information about the main hematological side effects and methods for their correction in modern antiviral therapy for chronic HCV infection. Conclusion. Thus, modern highly effective antiviral therapy for chronic HCV infection can be accompanied by a wide range of hematological disorders caused by various interactions of the hepatitis C virus and antiviral drugs with the bone marrow and the immune system.

Keywords: chronic HCV infection, anemia, antiviral therapy.

Introduction

Objective – to characterize changes in the blood system that occur during antiviral therapy for chronic hepatitis C (HCV) and modern methods for their correction. The most common hematological complications of antiviral therapy for chronic HCV infection are anemia, neutropenia, and thrombocytopenia, which can affect adherence and outcome of therapy. Preparations of hematopoietic growth factors contribute to the full implementation of the antiviral therapy program without dose reduction or drug withdrawal, which ensures the achievement of a sustainable virological response.

Viral hepatitis C (HCV) is highly prevalent among young people, which determines the importance and urgency of the problem of its detection and subsequent treatment. When examining 6433 apparently healthy young men aged 18–19 years, overall seropositivity (presence of HBsAg, anti-HCV) was found in 10.53% [1]. According to the World Health Organization (WHO), chronic viral hepatitis C (CHC) occurs in a significant part of the world's population [2]. Hepatitis C virus (HCV) is very virulent, the course of HCV, especially CHC, is accompanied by various complications both from the liver and from other organs and systems. In addition, no vaccine against HCV has been developed. HCV infection has an increased incidence among patients with diseases of the blood system [3], which is important for a hematologist, since it can significantly complicate the implementation of specific therapy. At the same time, it should be noted that CHC is often accompanied by various complications from the blood system [4]. The causes of hematological disorders in chronic hepatitis are most

often associated with ongoing antiviral therapy (adverse events with the development of anemia, leukopenia and thrombocytopenia), as well as with the course of hepatitis. Disease of the blood system can also be concomitant with CHC, and vice versa.

The basis of CHC therapy is the appointment of a combination of various antiviral drugs: interferon alfa (IFN-α), ribavirin, as well as modern direct antiviral drugs (boceprevir, telaprevir, simeprevir, sofosbuvir, dasabuvir, daclatasvir, paritaprevir, ombitasvir, etc.). Therapy may be accompanied by unequal tolerability, which leads to a deterioration in adherence to treatment, reduction in doses of drugs (or even their withdrawal) and, as a result, worsens the outcome of therapy. Direct-acting antiviral drugs are most acceptable in HCV-infected hematological patients due to their good tolerability, the achievement of an antiviral effect in most patients, and ease of use, which allows chemotherapy to be carried out with little or no delay.

The combination of antiviral drugs IFN-α with ribavirin, according to various studies, can reduce the level of hemoglobin up to 26% of the original in 7-32% of patients in the first 8 weeks of therapy [8]. According to the ADVANCE and ILLUMINATE clinical trials, 37% and 41% of patients, respectively, developed anemia during antiviral therapy with telaprevir, compared with 19% of patients who received the combination of pegylated interferon alfa (peg-IFN-α) with ribavirin [9, 10]. Boceprevir was also more likely than conventional therapy to develop anemia, which was reported in 49% of participants (SPRINT-2 study) compared to 29% treated with pegIFN-α + ribavirin [11].

The main mechanism of anemia in combination therapy is the accumulation of ribavirin metabolites in erythrocytes, which leads to oxidative stress, mitochondrial toxicity and, as a result, to hemolysis (dose-dependent hemolytic anemia develops) [12]. Other mechanisms include the myelosuppressive effect of IFN-α on the bone marrow, activation of apoptosis of erythroid precursors, provocation of immune hemolysis, decreased expression of erythropoietin and its receptor (in response to ribavirin), as well as impaired renal function [13, 14].

To correct drug-induced anemia in CHC, it is often practiced to reduce the doses of drugs (ribavirin, telaprevir, boceprevir), which can lead to a worsening of the antiviral response. In a study by D. Dieterich and J. Spivak (2003), the administration of erythropoietins made it possible to maintain the optimal therapeutic dose of ribavirin, i.e. do not cancel or reduce the dose of the drug in drug-induced anemia in patients with CHC, which improved the results of antiviral therapy [13]. In the correction of drug-induced anemia, in addition, the role of vitamin D is being studied. It has been shown that it can be involved in the regulation of erythropoietin production and expression of its receptor, as well as in the proliferation of erythroid precursors in the bone marrow [15]. Moreover, patients with chronic liver disease are deficient in vitamin D. In a meta-analysis of 11 clinical studies conducted by L. Villar et al., it was shown that among 1575 patients infected with HCV, in 1117 (71%) the level of vitamin D was reduced, and an increase in the frequency of achieving a sustained virological

response (SVR) was associated with higher serum vitamin D levels or with vitamin D replacement therapy [15]. RBC transfusions may also be given to quickly correct severe anemia.

Conclusion

Thus, modern highly effective antiviral therapy for chronic HCV infection can be accompanied by a wide range of hematological disorders caused by various interactions of the hepatitis C virus and antiviral drugs with the bone marrow and the immune system. It is now possible and necessary to support hematopoietic growth factors for all drug-induced hematological adverse events of antiviral therapy instead of reducing doses or discontinuing drugs.

References:

1. Lobzin Yu.V., Kryukov E.V., Zhdanov K.V. et al. The system of detection and medical support for HBV- and HCV-infections in conscripted military personnel (on the model of the Black Sea Fleet) // Hepatology. 2003. No. 1. S. 6–10.
2. Lavanchy D. Evolving epidemiology of hepatitis C virus. Clin. microbiol. Infect. 2011 Vol. 17, No. 2, pp. 107–115.
3. Garmaeva T.Ts. Viral hepatitis B and C in patients with diseases of the blood system: Ph.D. dis. Dr. med. Sciences. M., 2012.
4. Fried MW Side effects of therapy of hepatitis C and their management // Hepatology. 2002 Vol. 36, No. 5. Suppl. 1. P. S237–S244.
5. Rossotti R., Travi G., Pazzi A. et al. Rapid clearance of HCV-related splenic marginal zone lymphoma under an interferon-free, NS3/NS4A inhibitor-based treatment. A case report // J. Hepatol. 2015. Vol. 62, No. 1. p. 234–237.
6. Carrier P., Jaccard A., Jacques J. et al. HCV-associated B-cell non-Hodgkin lymphomas and new direct antiviral agents // Liver Int. 2015. Vol. 35, No. 10. P. 2222–2227.
7. Peveling-Oberhag J., Arcaini L., Bankov K. et al. The anti-lymphoma activity of antiviral therapy in HCV-associated B-cell non-Hodgkin lymphomas: a meta-analysis // J. Viral Hepat. 2016. Vol. 23, No. 7. P. 536–544.
8. Sulkowski MS, Wasserman R., Brooks L. et al. Changes in haemoglobin during interferon alpha-2b plus ribavirin combination therapy for chronic hepatitis C virus infection // J. Viral Hepat. 2004 Vol. 11, No. 3. P. 243–250.
9. Jacobson IM, McHutchison JG, Dusheiko G. et al. Telaprevir for previously untreated chronic hepatitis C virus infection // N. Engl. J. Med. 2011 Vol. 364, No. 25, pp. 2405–2416.
10. Mukhamedjanova MH Anemia in patients with interferences and interpretation of modern therapy. NDM 4(36)2021 P150-152
11. M.Kh. Mukhamedzhanova, N.Sh. Ahmedova. Determination of hepsidin in patients with chronic hepatitis associated with chf as a predictor of the severity of

hepatocardial syndrome.// Art of Medicine International Medical Scientific Journal 10.5281/zenodo.7361300 Vol 2, Issue 3 P.332-336

12. Soota K., Maliakkal B. Ribavirin induced hemolysis: a novel mechanism of action against chronic C virus infection // World J. Gastroenterol. 2014. Vol. 20, No. 43. P. 16 184–16 190.

13. Dieterich DT, Spivak, JL Hematologic disorders associated with hepatitis C virus infection and their management // Clin. Infect. Dis. 2003 Vol. 37, No. 4, pp. 533–541.

14. Milovanova S.Yu., Kozlovskaya L.V., Lopatkina T.N. Anemia in acute and chronic liver diseases. clinical hepatology. 2006; 2(2):16–23.

15. Solovyova, A.E. Prognostic significance of liver density in decompensated heart failure: results of a prospective observational study based on indirect elastometry data / A.E. Solovieva, Zh.D. Kobalava, S.V. Villevalde, M. Bayarsaikhan, I.V. Garmash, M. Fudim // Cardiology. - 2018. - No. 58 (10C). - P.20-32.