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Correction Of Anemia with Red Blood in Patients with Liver Circulosis

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Annotation: Anemia of various etiologies is a common complication of chronic liver disease. Causes of anemia include acute or chronic gastrointestinal bleeding and secondary hypersplenism to portal hypertension. Severe hepatocellular disease is prone to bleeding due to lack of blood clotting factors synthesized by hepatocytes and / or impaired blood clotting caused by thrombocytopenia. After the development of hepatitis, aplastic anemia characterized by pancytopenia and hypocellular bone marrow may occur. Its presentation includes progressive anemia and hemorrhagic manifestations. Hematological complications of complex treatment of chronic viral hepatitis include clinically significant anemia resulting from treatment with ribavirin and / or interferon. This article discusses, data on the correction of anemia when bleeding from the esophagus in patients with cirrhosis of the liver.

Keywords: Anemia, liver disease, liver failure, anemia, pegylated interferon, ribavirin, alcohol, esophageal, anemia correction.

Introduction.

Chronic liver disease is often associated with hematologic abnormalities. Approximately 75% of patients with chronic liver disease experience anemia of various etiologies. The main cause of anemia associated with chronic liver disease is bleeding, especially in the gastrointestinal tract. Patients with severe hepatocellular disease develop endothelial dysfunction, thrombocytopenia, lack of coagulation factors, and blood clotting defects as a result of various diseases. Anemia is especially common in patients with cirrhosis of the liver when bleeding from the esophagus. In patients with chronic liver disease, anemia may be exacerbated by folic acid and / or vitamin B12 deficiency, which may occur as a result of secondary nutrition or malabsorption.

In severe hepatocellular disease, a decrease in the synthesis of plasma proteins produced in the liver leads to a decrease in serum levels of several coagulation factors. Bleeding can occur as a complication of chronic liver disease due to a lack of blood clotting factors produced by one or more livers, thrombocytopenia, and / or impaired platelet function. Bleeding in such patients may also result from secondary portal hypertension from esophageal or gastric varicose veins. The biosynthetic pathways of coagulation factors II, VII, IX, and X are located in hepatocytes and depend on vitamin K [1].

In patients with chronic liver disease, splenomegaly, usually caused by portal hypertension, can lead to secondary hemolysis, increased plasma volume, macrocytosis, and megaloblastic anemia. Alcohol,

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which is a common etiological factor in chronic liver disease, is toxic to bone marrow. Alcoholism often develops secondary malnutrition, the manifestation of which may be anemia caused by folic acid deficiency. Some patients develop bone marrow failure and aplastic anemia after an episode of hepatitis. Finally, anemia is a recognized complication of treatment of chronic hepatitis C with a combination of interferon and ribavirin: in this context, anemia is mainly caused by hemolysis induced by ribavirin [2].

The frequent association of anemia with chronic liver disease and / or hepatocyte insufficiency is the basis for studying the role of the liver in the formation and destruction of erythrocytes. Indeed, the liver itself may be associated with a variety of mechanisms that contribute to the development of anemia in patients with chronic liver disease. The frequent association of anemia with chronic liver disease and / or hepatocellular insufficiency serves as a basis for studying the role of the liver in the formation and destruction of red blood cells. Indeed, patients with liver disease may have different mechanisms in the development of anemia.

Bleeding from the esophagus is a common and potentially serious complication of portal hypertension. It is usually caused by a rupture of the esophageal varices. Bleeding caused by this mechanism is the second most common cause of death in patients with cirrhosis. In such patients, esophageal rupture causes approximately 70% of upper gastrointestinal bleeding episodes. [3]. Acute bleeding can lead to severe hypovolemia and subsequent secondary iron deficiency anemia. The primary goal of treatment is to correct hypovolemia and restore stable hemodynamic function; for average blood pressure and hemoglobin, minimum values for 80 mmHg and 8 g / 100 ml, respectively, should be maintained. Initially, gelatin-based colloids or solutions of human albumin may be introduced to correct hypovolemia. However, packaged infusions of erythrocytes in plasma are very suitable in this regard, as such infusions are able to correct not only hypovolemia but also secondary anemia. Primary treatment includes both medical and endoscopic procedures [4].

In some patients with cirrhosis, chronic bleeding occurs in the gastrointestinal tract. Esophageal and gastric varicose veins and / or portal hypertensive gastropathy may be associated with slow chronic loss of intestinal blood and the development of chronic iron deficiency anemia. The most important method of treatment is to prevent varicose bleeding. The annual frequency of varicose bleeding in patients with cirrhosis is about 4%, but in the group with medium or large vessels this frequency is about 15% The risk of recurrent bleeding in patients who survived the first episode of bleeding> 60%. Accordingly, all patients who survive varicose bleeding should receive active treatment aimed at preventing recurrence. [5].

Additional treatment with oral iron supplements is indicated for iron deficiency anemia resulting from chronic blood loss. In some cases of chronic liver disease, intravenous iron supplements may be given to increase blood plasma concentrations and iron accumulation in tissues. Another mechanism of anemia in patients with chronic liver disease is secondary hypersplenism to portal hypertension. Hypersplenism is associated with splenomegaly. In addition to chronic liver disease, splenic vein thrombosis can also cause an increase in pressure within the portal venous system, which can lead to secondary hypersplenism. The main symptoms of hypersplenism are associated with pancytopenia. Hemolytic anemia is caused by intrasplenic destruction of erythrocytes.

Destruction of megakaryocytes and leukocyte precursors leads to thrombocytopenia and leukopenia [6]. Signs and symptoms of hypersplenism are affected by the underlying disease; they include abdominal pain and / or discomfort, and in advanced cases, secondary esophageal bleeding from portal hypertension. There may be hyperplasia of progenitor cells in the bone marrow. It is important to determine the cause of hypersplenism. The main therapeutic approach for this syndrome is treatment aimed at the underlying disease, usually chronic liver disease. When chronic liver disease develops, additional therapeutic options may need to be taken. After assessing the severity of hepatocellular dysfunction in a patient with chronic liver disease, splenectomy may be considered if the splenic vein is thrombosed. An alternative approach is partial or complete embolization of the splenic artery, with good results in some recent studies, particularly with lower morbidity and mortality rates than those associated with surgery.

Patients with cirrhosis often develop folic acid and vitamin B12 deficiency. These deficiencies may be related to inadequate food intake or intestinal malabsorption. Anemia caused by folic acid deficiency can be caused not only by a lack of folic acid in the diet, but also by the weak antifolate effect of ethanol.

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Supplements of vitamins A, B and C can be administered empirically to patients who have developed alcoholism. Alcohol anemia can also occur as a result of the direct toxic effects of alcohol on bone marrow erythrocyte precursors. Management of suppression of alcoholic erythropoiesis includes abstinence from alcohol and a nutritious diet with appropriate supplements.

Conclusion.

Liver disease is often associated with hematologic abnormalities. Most of these patients have anemia of various etiologies. Bleeding is one of the most serious causes of anemia, with high mortality and impaired blood clotting contributing to anemia. Other mechanisms of anemia include secondary aplastic anemia to anterior hepatitis or side effects of treatment of hepatitis with interferon and ribavirin. In patients with alcoholic liver disease, various effects of alcohol can lead to anemia, such as malabsorption, malnutrition, or direct toxic effects. In each case, the pathogenesis of anemia is different and it is important to start proper therapy.

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