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Blood Transfusion Therapy in Patients with Vitamin B12 Deficiency Anemia after Resection of 2/3 of the Stomach

1 Makhmonov Lutfullo Saydullayevich,
2 Babakalonov Shukhrat Ibragimovich,
3 Abdiev Kattabek Makhmatovich,
4 Abdullaev Dilshod Muradovich

[1]PhD, head of the department hematology Samarkand State Medical University
[2]Head of the surgical department Samarkand regional multi-network medical center
[3]Associate Professor of the department Hematology Samarkand State Medical University
[4]Head of the surgical department Samarkand State Medical University

Abstract: This article is devoted to blood transfusion therapy in patients with vitamin B12 deficiency anemia after 2/3 gastrectomy. The article describes the causes of vitamin B12 deficiency in patients, its therapy, methods of blood transfusion therapy in patients with anemia.

Keywords: anemia, blood, breath, palpitations occur, therapy, vitamin B12

Introduction

According to some reports, anemia occurs in 50% of patients undergoing gastrectomy. Symptoms depend on how severely the red blood cell and hemoglobin levels are reduced. Such patients experience constant weakness, loss of strength, do not tolerate physical activity, become pale, and their concentration decreases. In more serious cases, even with slight physical exertion, shortness of breath, palpitations occur, headaches, tinnitus disturb, sleep and appetite are disturbed. Severe anemia threatens the development of heart failure.

Literary review and methodology

After partial (resection) and complete (gastrectomy) removal of the stomach, many cancer patients develop anemia - a condition in which the content of red blood cells and hemoglobin decreases in the blood, organs and tissues no longer receive the right amount of oxygen. After surgery to remove the stomach, this is due to a violation of the absorption of three substances necessary for blood formation: iron, vitamin B12 (cyanocobalamin) and folic acid (sometimes called vitamin B9).

Iron-deficiency anemia: Iron is an important component of hemoglobin, a protein found in red blood cells that carries oxygen.

The main cause of iron deficiency anemia after removal of the stomach is indigestion:

- Iron in many foods (mainly of plant origin) is found in a hard-to-digest non-heme form (Fe3+). In the acidic environment of the stomach, it is converted to the easily digestible heme form

(Fe2+) and then absorbed in the duodenum and upper jejunum. After removal of the stomach, the conversion of non-heme iron to heme iron and its absorption are disrupted.

- After some surgeries, such as Roux-en-Y gastric reconstruction, the duodenum is shut down and there is no absorption of iron.

The risk depends on the volume and type of surgical intervention: after gastrectomy, it is higher than after subtotal resection (removal of most of the organ). Iron malabsorption can be exacerbated by factors such as decreased appetite, malnutrition, blood loss at the sutures in the stomach and intestines, and side effects of chemotherapy drugs.

Usually at first iron deficiency proceeds latently - without pronounced symptoms. After 1–3 years, pronounced manifestations of iron deficiency anemia appear.

How to detect iron deficiency anemia after removal of the stomach? You need to do a blood test. Determine indicators such as the level of hemoglobin, ferritin, transferrin, serum iron, the percentage of transferrin saturation, the latent iron-binding capacity of serum, the number of erythrocytes, hematocrit (the ratio of cell volumes and blood plasma), MCH (average hemoglobin content in one erythrocyte), MCV (average erythrocyte volume).

How to increase the content of hemoglobin and erythrocytes in the blood? In severe cases (hemoglobin level less than 70 g/l), red blood cell transfusion may be indicated. For the prevention and correction of iron deficiency anemia, the doctor may prescribe oral (to be taken by swallowing) iron preparations: sulfate, gluconate. If the patient is unable to take oral iron due to adverse reactions, or if it is not absorbed from the intestines, intravenous injections are given. Among food products, easily digestible heme iron is rich in meat, poultry, fish, and seafood. Vitamin C improves iron absorption.

Vitamin B12 deficiency: Vitamin B12 (cyanocobalamin) is essential for the maturation of red blood cells - erythrocytes. It is not synthesized in the human body and must be constantly supplied with food.

Vitamin B12 is absorbed in the lower small intestine. But in order to go into a digestible form, it must first bind to a protein called internal factor of Castle, which is produced by the cells of the mucous membrane of the bottom and body of the stomach. After a gastrectomy, the Castle factor is no longer produced, and the absorption of vitamin B12 is impaired. Pernicious anemia develops.

Possible manifestations of vitamin B12 deficiency: weight loss, loss of taste, nausea, diarrhea or constipation, burning sensation, enlarged tongue, bright red color of the tongue, "varnished tongue", slight yellowness of the skin, vitiligo (discolored spots on the skin), early graying of the hair. In addition, vitamin deficiency can lead to the death of nerve cells and neurological disorders: numbness, tingling in the arms and legs, disruption of the coordination of the work of different muscle groups. Symptoms of vitamin B12 deficiency may appear as early as a year after surgery. They do not occur immediately, because a fairly large supply of the vitamin is stored in the liver.

Discussion and results.

How to detect B12 deficiency anemia? In the blood test, an increased MCV (mean volume of erythrocytes), normal MCH (average hemoglobin content in one erythrocyte), anisocytosis (change in the size of erythrocytes), poikilocytosis (change in the shape of erythrocytes), a decrease in the number of reticulocytes (young forms of erythrocytes), detect megalocytes (large oval-shaped cells), leukopenia (a decrease in the number of leukocytes). In a biochemical blood test, a reduced content of vitamin B12 can be detected, but this is not a very reliable study.

How to provide the body with vitamin B12? Classically, after removal of the stomach, patients are advised to administer vitamin B12 in the form of intramuscular injections. However, studies show that oral (by swallowing) B12 supplementation also helps, because in this case, another absorption pathway, independent of the internal factor of Castle, begins to work. Subcutaneous injections may also be used. Vitamin B12 is rich in foods such as milk, red meat, fish, crustaceans, and beef liver.

The main reason for the development of vitamin B12 deficiency is a violation of its absorption in the intestine. The parietal cells of the body and fundus of the stomach secrete a protein, the so-called. "Castle's intrinsic factor" (discovered by W.Castle in 1930), necessary for the absorption of vitamin B12 (cobalamin, "external factor"). The formation of a stable complex "cobalamin - internal factor Castle" begins in the alkaline environment of the duodenum 12, then the absorption of vitamin B12 occurs in the small intestine, mainly in the ileum, where cubulin is localized - a specific protein receptor for "intrinsic factor". In the process of absorption, the “complex” breaks down, vitamin B12 penetrates the wall of the small intestine into the bloodstream, where it binds to transcobalamin, which delivers it to consumer cells, including cells in the bone marrow and liver.

The following pathological processes can lead to impaired absorption of vitamin B12:

- decreased production or absence of "intrinsic factor Castle" due to the presence of autoantibodies to it or to the parietal cells of the stomach, other atrophic gastritis, gastric resection;
- diseases of the small intestine (chronic enteritis with malabsorption syndrome, tumors, including lymphomas);
- competitive absorption (diverticulosis with a change in flora, diphyllobothriasis, "blind loop" syndrome with anastomosis of the small intestine);
- diseases of the pancreas that increase the acidity of the intestinal contents (tumor with the formation of gastrin, Zollinger-Ellison syndrome);
- long-term use of certain drugs (proton pump inhibitors [group A02BC according to the ATC classification], metformin **, etc.) [5,6]

Alimentary deficiency of vitamin B12 can develop in people who follow a vegetarian or vegan diet. All of the above risk factors for the development of cobalamin deficiency must be considered in patients with hereditary forms of hemolytic anemia. The main clinical manifestations of B12-deficiency anemia include gradually increasing weakness, apathy, exercise intolerance, palpitations, heart pain, dyspeptic disorders, as well as weakness in the legs and paresthesia, migrating pains, "numbness" of the limbs and a gradual loss of sensation in the fingers. Typical - puffiness of the face and amimism, pale icteric skin color, smoothness of the papillae of the tongue ("raspberry lacquer tongue"). In young people - often early graying of hair. Subfebrile temperature and an increase in the size of the spleen are often noted, which often lead to diagnostic errors. In the absence of timely diagnosis and treatment of B12 deficiency, a violation of superficial and deep muscle sensitivity, hearing loss, vision loss, areflexia develop, in advanced cases - dysfunction of the pelvic organs and severe cognitive impairment. In severe cases of B12 deficiency, damage to the peripheral nervous system (funicular myelosis) dominates: ataxia, hyporeflexia, the appearance of pathological signs - the Babinski reflex [1–4]

With B12-deficiency anemia, a general (clinical) blood test reveals macrocytic, hyperchromic anemia, with an increase in the average volume of erythrocytes (MCV), the average hemoglobin content (MCH) in erythrocytes and severe aniso-poikilocytosis. MCHC remains within the reference values. Characteristic changes in the general (clinical) blood test are basophilic puncture of erythrocytes, Jolly bodies and Cabot rings in erythrocytes, the presence of ovalocytes and single normoblasts. The absolute number of reticulocytes is reduced, while their relative number may be normal or increased. If technically possible, additional information can be provided by the determination of reticulocyte indices: there is an increase in the average volume of reticulocytes (macroreticulocytes), the average content of hemoglobin in reticulocytes and, often, an increase in the fraction of immature reticulocytes. An early sign of B12 deficiency is hypersegmentation of neutrophil nuclei, sometimes neutropenia. Moderate thrombocytopenia is often observed, rarely - deep, usually without hemorrhagic syndrome [8].

Conservative treatment: The daily requirement for vitamin B12 is 2.4 mcg. Most patients with vitamin B12 deficiency, manifesting megaloblastic anemia and/or neurological symptoms (funicular myelosis), have a malabsorption syndrome and require urgent parenteral administration of

cyancobolamine** (vitamin B12). Lack of timely replacement therapy can lead to the development of irreversible multiple organ failure [7].

Folic acid deficiency: Folic acid is also important for normal blood formation, and it works in conjunction with vitamin B12, as it is activated by it. In order to detect a deficiency of this compound, it is necessary to determine the level of folic acid in erythrocytes (RBC).

To prevent and combat folic acid deficiency, appropriate drugs are used. It is also found in foods such as citrus fruits, beets, broccoli, leafy vegetables, legumes, asparagus, and eggs.

REFERENCES:

1. A.I. Vorobyov, editor. Guide to Hematology: in 3 vols. M.: "Niyudiamed"; 2005.
2. Rumyantsev AG, Maschan AA, Zhukovskaya EV. Children's hematology. 2015.
3. Green R, Allen LH, Bjørke-Monsen A-L, Brito A, Guéant J-L, Miller JW, et al. Vitamin B12 deficiency. Nat Rev Dis Prim 2017;3:17040.
4. Green R., Folate, cobalamin, and megaloblastic anemias, Williams Hematology, 9th ed., Chapter 41, p. 583-615, n.d.
5. Lam JR, Schneider JL, Zhao W, Corley DA. Proton Pump Inhibitor and Histamine 2 Receptor Antagonist Use and Vitamin B 12 Deficiency. JAMA 2013;310:2435. https://doi.org/10.1001/jama.2013.280490.
6. Ahmed MA, Muntingh G, Rheeder P. Vitamin B12 deficiency in metformin-treated type-2 diabetes patients, prevalence and association with peripheral neuropathy. BMC Pharmacol Toxicol 2016;17:44. https://doi.org/10.1186/s40360-016-0088-3.
7. Specker BL, Black A, Allen L, Morrow F. Vitamin B-12: low milk concentrations are related to low serum concentrations in vegetarian women and to methylmalonic aciduria in their infants. Am J Clin Nutr 1990;52:1073–6. https://doi.org/10.1093/ajcn/52.6.1073.
8. Michael Whitehead V. Acquired and inherited disorders of cobalamin and folate in children. Br J Haematol 2006;134:125–36. https://doi.org/10.1111/j.1365-2141.2006.06133.x.
9. Бобоев, С. С., & Аллаяров, А. Т. (2020). ЭФФЕКТИВНОСТЬ ХИРУРГИЧЕСКОГО ЛЕЧЕНИЯ ПТЕРИГИУМА ПО МЕТОДУ АУТОПЛАСТИКИ КОНЪЮНКТИВАЛЬНЫМ ЛОСКУТОМ. In *Молодежь и медицинская наука в XXI веке* (pp. 526-527).
10. Shavkatjonovna, K. M., Tolibovich, A. A., & Abduazizovich, Y. A. (2023). Optimization of the Ophthalmic Service in Diabetic Retinopathy. *Central Asian Journal of Medical and Natural Science*, *4*(1), 308-312.
11. Khakimova, M. S., & Allayarov, A. T. (2022). OPTIMIZATION OF METHODS OF TREATMENT OF ACCOMMODATION SPASM IN CHILDREN. In *НАУЧНЫЕ ИССЛЕДОВАНИЯ 2022* (pp. 309-311).
12. Мадашева, А. Г., & Жураева, М. З. (2019). Биохимические показатели и комплексное лечение больных псориазом с лечебным плазмаферезом. *Достижения науки и образования*, (10 (51)), 78-82.
13. Дадажанов, У., & Мадашева, А. (2019). Эритропоэтинлар. *Журнал вестник врача*, *1*(4), 153-155.
14. Мадашева, А. Г., Дадажанов, У. Д., Абдиев, К. М., Маматкулова, Ф. Х., & Махмудова, А. Д. (2019). Динамика электронейромиографических показателей и эффективность электрической стимуляции мышц у больных гемофилией с мышечными атрофиями. *Достижения науки и образования*, (10 (51)), 26-30.
15. Madasheva, A. G., Yusupova, D. M., & Abdullaeva, A. A. EARLY DIAGNOSIS OF HEMOPHILIA A IN A FAMILY POLYCLINIC AND THE ORGANIZATION OF MEDICAL CARE. *УЧЕНЫЙ XXI ВЕКА*, *37*.

16. Жураева, М. З., Нурматова, И. Б., & Мадашева, А. Г. Псориаз билан касалланган беморлар биокимёвий кгрсаткичлари ва уларни комплекс даволашда плазмаферез усулини іґллашнинг самарадорлиги. *МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ УЗБЕКИСТАН РЕСПУБЛИКАНСКИЙ СПЕЦИАЛИЗИРОВАННЫЙ НАУЧНО-ПРАКТИЧЕСКИЙ МЕДИЦИНСКИЙ ЦЕНТР АКУШЕРСТВА И ГИНЕКОЛОГИИ АССОЦИАЦИЯ ВРАЧЕЙ ЧАСТНОЙ ПРАКТИКИ УЗБЕКИСТАНА КЛИНИКА «MAHLIYO-SHIFO» & V «MAHLIYO-SHIFO» & V*, 13.