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EARLY DIAGNOSIS AND TREATMENT OF FUNICULAR MYELOSIS IN V12 DEFICIENCY ANEMIA

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ABSTRACT

A clinical observation of a 73-year-old patient with severe signs of funicular myelosis against the background of severe vitamin B12 deficiency is presented. A feature of this case was the long-term preservation of good health and general well-being of the patient, despite the development of a severe degree of hyperchromic anemia in him, the detection in the patient of signs of both normo- and megaloblastic types of hematopoiesis. Administration of vitamin B12 to the patient gave a quick and stable clinical effect.

KEYWORDS: *Funicular Myelosis, Vitamin B12, Deficit, Syndrome, Anemia, Acid Metabolism.*

INTRODUCTION

Funicular myelosis (Lichtheim's syndrome) is a pathology of the posterior cords of the spinal cord, which is combined with pernicious anemia and is caused by vitamin B12 deficiency. The basis of vitamin deficiency is the difficulty in adsorption of vitamin B12 in the digestive tract ("starvation among abundance"). The syndrome is combined with pernicious anemia, the frequency of which increases with age and is 0.1% in young people, 1% in the elderly, after 75 years it is registered in 4% of people, in general, it occurs from 1 to 50 cases per 100,000

population [1–3]. In many elderly patients, cyanocoblamin deficiency develops due to the inability to excrete it from food, for example, in atrophic gastritis, when there is a decrease in the secretion of hydrochloric acid and proteases [1, 2].

For the normal metabolism of vitamin B12, such important factors as the presence of the vitamin in food, adequate gastric and pancreatic secretion, intact intestinal microflora, sufficient synthesis of the Castle factor and transcobalamin are necessary [1, 2, 5]. A defect in one of these factors leads to the development of vitamin B12 deficiency, as a result of which DNA synthesis and nucleic acid metabolism are impaired, cell division is inhibited, and nerve tissues are damaged [2–4]. Vitamin B12, closely related to folic acid in its mechanisms of action, plays an important role in metabolic processes, participates in protein, fat and carbohydrate metabolism. With its deficiency, the most pronounced changes are observed in rapidly proliferating cells, such as cells of the bone marrow, oral cavity, tongue and gastrointestinal tract, which leads to impaired hematopoiesis, the appearance of glossitis, stomatitis and intestinal malabsorption. With a deficiency of vitamin B12, megaloblastic anemia develops, a defect in DNA synthesis is observed, affecting all cell lines of hematopoiesis: division and maturation of erythrocytes are disturbed, the number of erythrocytes in the blood decreases, the average volume of erythrocytes increases, neutrophils are also changed - hypersegmented, pancytopenia is often observed. Vitamin B12 is a cofactor for the enzyme homocysteine methyltransferase involved in the conversion of homocysteine to methionine. Methionine is important for the synthesis of phospholipids and the myelin sheath of neurons, so B12 deficiency is accompanied by neurological symptoms (mental disorders, polyneuritis, funicular myelosis - damage to the spinal cord). Zinc balamin deficiency often develops in the elderly, manifesting as neurological disorders. The accumulation of homocysteine is a risk factor for the development of atherosclerotic changes. Cyanocobalamin, participating in the synthesis of choline and methionine, has a beneficial effect on the liver, prevents the development of fatty hepatosis [4–7].

Patient B., aged 73, was admitted to the department on January 16, 2019 with complaints of severe weakness, rapid fatigue of the leg muscles, which limited mobility to 50–60 meters when walking and one flight of stairs when climbing stairs, severe unsteadiness when walking, loss of appetite, constant feeling of dry mouth. Until January 2019, he felt satisfactory, went in for physical education, regularly participated in city sports competitions in his age group, freely walked up to the fourth floor. On January 6, against the background of severe short-term stress, he noticed rapid fatigue when walking or doing ordinary housework, he had difficulty climbing to the fourth floor. Three days later, he sharply weakened: he could hardly walk 50-60 meters, climbed the stairs with support. Previously observed for chronic gastritis, did not follow the diet. In 2007, fibrogastroduodenoscopy revealed hyperplasia of the subcardial part of the stomach. In addition, fatty hepatosis, microliths and kidney cysts were previously diagnosed. On admission he was in a state of moderate severity. The skin is icteric, the sclera of the eyes are icteric. The patient has a normosthenic physique, satisfactory nutrition. In the lungs, breathing is vesicular with a frequency of 18 per minute, there are no wheezing. Heart rate 76 per minute. Heart sounds are clear, rhythmic. BP 125/80 mmHg Art. The tongue is pink, shiny. The abdomen is painless. Liver along the edge of the costal arch, dimensions according to Kurlov 9 × 8 × 7 cm. The spleen was not palpable. Defecation and urination are not disturbed.

Neurological status: the patient is conscious, oriented, communicative. The pupils are round, narrow, D=S. Photoreactions (direct and friendly) are sluggish, symmetrical. The pupillary response to convergence and accommodation is reduced. There is no movement of the eyeballs, diplopia and nystagmus. The face is symmetrical. Tongue in the midline. Swallowing and phonation are not disturbed. Symptoms of oral automatism are positive on both sides. The gait is ataxic. Muscle strength 5 points. Deep reflexes are low, equal. Muscle tone is diffusely reduced. On the left side, Babinski's sign is positive. Performs coordinating tests with a slight intention on both sides. Unsteady in the Romberg position. There are no meningeal signs. On the left, hypoesthesia from the level of the upper third of the lower leg with a hyperpathic component. Decreased articular-muscular feeling in III-V toes of both feet. Clinical blood test at admission: hemoglobin 72 g/l, erythrocytes 1.73 × 1012/l, leukocytes 4.45 × 109/l, po box 2%, po box 61.2%, eosinophils 2.16 %, monocytes 8.82%. basophils 0.6%, lymphocytes 25.2%, platelets 159 × 109/l, col. n. 1.25, cf. about. er. 113 μm3, avg. sod. gem. in er. 41.7 pg, hematocrit 19.5%, ESR 24 mm/h, erythrocyte anisocytosis +++ (microcytes, macrocytes, megalocytes), erythrocyte poikilocytosis ++ (schizocytes, teardrop-shaped erythrocytes), segmented neurophiles with hyperpigmented nuclei.

Urinalysis: specific gravity 1025; acid reaction, urobilinogen 66.0 mmol/l, bilirubin 8.6 mmol/l, cylinders were not detected; protein, sugar, ketone bodies, leukocytes, erythrocytes, bacteria were not detected. Biochemical blood test: total bilirubin - 40.8 μmol/l, blood urea - 5.6 mmol/l, blood creatinine - 101 μmol/l, blood glucose - 4.9 mmol/l, cholesterol - 2.23 mmol /l, triglycerides — 0.54 mmol/l, HDL cholesterol — 0.70 mmol/l, LDL cholesterol — 1.28 mmol/l, uric acid — 234 μmol/l, aspartate aminotransferase — 31U/l, alanine aminotransferase — 29 U / l, total protein - 63 g / l, albumin - 42 g / l, calcium - 1.22 mmol / l, potassium - 4.35 mmol / l, sodium - 143 mmol / l, folic acid - 4, 4 ng/ml, vitamin B12 — 54 pg/ml, iron — 18.9 μmol/l, homocysteine — 17.0 μmol/l, thyroid hormones: T4 St. - 15.2 pmol / l, TSH - 0.72 mU / l, At-TPO < 3 U / l. Coagulogram: APTT — 27 s, fibrinogen — 2.33 g/l, INR — 1.31. No pathology was detected in blood tests for tumor markers. ECG: sinus rhythm, horizontal position of the electrical axis of the heart, intraventricular slowing of conduction, signs of left ventricular hypertrophy. Chest X-ray - no pathology. FGDS: distal non-erosive esophagitis grade 0, cardia insufficiency, atrophic focal gastritis of the antrum, subatrophic diffuse bulbitis.

Colonoscopy: acute splenic angle, chronic internal hemorrhoids, no pathological changes up to the distal part of the transverse colon were revealed. Sternal puncture was not performed in the conditions of the rehabilitation department. Clinical diagnosis: “acquired B12 deficiency anemia of severe degree; hemolytic jaundice; funicular myelosis; glossitis". Treatment was prescribed: cyanocobalamin 500 mcg intramuscularly 2 times a day daily. The patient's well-being began to improve almost “on the needle”. On the second day after the injection of cyanocobalamin, the severity of weakness decreased, the patient could walk more than 400 meters without stopping, on the third day he began to exercise. On the third day clinical blood test: hemoglobin 106 g/l, er. 2.68 × 1012/l, l. 6.8 × 109/l, s/b 1%, s / i 66.2%, eos. 1.6%, mon. 7.6% base 0.5%, lymph. 23.6%, tr. 300 × 109/l, col. 1.18. Wed about. er. 121 μm3, avg. sod. gem. in er. 39.7 pg, hematocrit 32.4%, ESR 8 mm/h, anisocytosis of erythrocytes ++ (microcytes, macrocytes), poikilocytosis of erythrocytes ++ (schistocytes, teardrop-shaped erythrocytes), segmented neurophiles with hyperpigmented nuclei, reticulocytes 12%. One week after the start of treatment: hemoglobin: 115 g/l, er. 2.9 × 1012/l, l. 6.3 × 109/l, s/b 1%, s/b 59.2%, eos. 2.1%

mon. 6.5% base 0.8%, lymph. 31.6%, tr. 292 × 109/l, col. n. 1.15, cf. about. er. 120 μm3, avg. sod. gem. in er. 38.6 pg, hematocrit 36.2%, ESR 4 mm/h, erythrocyte anisocytosis +, erythrocyte poikilocytosis +, segmented neurophiles with hyperpigmented nuclei, reticulocytes 14%. By this time, the patient had increased muscle strength in the legs, motor activity was restored, and sensory disorders regressed. After 1.5 months. after the start of treatment, a clinical blood test: hemoglobin: 159 g/l, er. 4.78 × 1012/l, l. 7.02 × 109/l, tr. 176 × 109/l, col. p. 0.99, hematocrit 45.9%, ESR 2 mm per hour, s/i 1%, s/i 57.8%, eos. 2.91% mon. 5.54% base 0.7%, reticulocytes 0.2%. Biochemical blood test: homocysteine — 13.6 μmol/l, vitamin B12 — 920 pg/ml.

Thus, the diagnosis of B12-deficiency anemia in the described patient was based on the features of the clinical manifestation of the disease, the detection of hyperchromic anemia in his blood, vitamin B12 deficiency, and signs of erythrocyte hemolysis. In the given example, the disease debuted with funicular myelosis, manifested by pronounced motor and sensory disorders: muscle weakness in the legs, impaired joint-muscular feeling, and sensitive ataxia. The good clinical response of the body in general and the red hematopoietic germ in particular to the administration of cyanocobalamin confirmed the correctness of the diagnosis.

A feature of this case was an acute, sudden onset in an elderly patient against the background of good health, general well-being, the detection of a fairly pronounced decrease in hemoglobin in the blood, which, apparently, developed gradually, without leading to decompensation, and only as a result of severe stress manifested clinically, the presence of both macro and microcytes in the blood, which indicates the development of hematopoiesis in two directions. The revealed high level of reticulocytes before the introduction of B12 is explained by the activation, possibly final, of compensatory mechanisms.

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