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New Methods of Treatment of Funicular Myelosis in Anemia with Vitamin V12 Deficiency Have Been Developed

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Abstract: ***Funicular myelosis (Lichtheim syndrome) is a rare neurological disorder associated with vitamin B12 deficiency, often concurrent with pernicious anemia. This deficiency is typically due to malabsorption issues in the digestive tract, particularly prevalent among the elderly population. Vitamin B12 plays a crucial role in various metabolic processes, including DNA synthesis, hematopoiesis, and neurological function. Here, we present a case study of a 73-year-old patient with severe acquired B12 deficiency anemia, hemolytic jaundice, glossitis, and funicular myelosis. The patient exhibited pronounced motor and sensory deficits, which significantly improved following treatment with cyanocobalamin. We highlight the clinical features, diagnostic considerations, and therapeutic outcomes of this case, emphasizing the importance of early recognition and intervention in B12 deficiency-related neurological disorders.***

Keywords: ***Funicular myelosis, Lichtheim syndrome, vitamin B12 deficiency, pernicious anemia, malabsorption, neurological disorders, cyanocobalamin treatment, hematopoiesis, glossitis, hemolytic jaundice.***

I. INTRODUCTION

Funicular myelosis (Lichtheim syndrome) is a pathology of the posterior cord of the spinal cord, which is combined with pernicious anemia and is caused by vitamin B12 deficiency. Vitamin deficiency is based on the difficulty of adsorption of vitamin B12 in the digestive tract ("starvation in the midst of abundance"). The syndrome is combined with pernicious anemia, the frequency of which increases with age and is 0.1% in the young, 1% in the elderly, after 75 years it is registered in 4% of individuals, in general, there are from 1 to 50 cases per 100,000 population [1-3]. In many elderly patients, cyanocoblamine deficiency develops due to the inability to isolate it from food, for example, with atrophic gastritis, when there is a decrease in the secretion of hydrochloric acid and proteases [1, 2]. For normal vitamin B12 metabolism, such important factors as the presence of vitamin in food, adequate gastric and pancreatic secretion, preserved intestinal microflora, sufficient synthesis of Castle factor and transcobalamin [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 disrupted, inhibition of cell division develops, and nerve tissues are damaged [2-4].

Vitamin B12, which is 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, for example, 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 vitamin B12 deficiency, megaloblastic anemia develops, a DNA synthesis defect affecting all hematopoiesis cell lines is observed: erythrocyte division and maturation are disrupted, the number of erythrocytes in the blood decreases, the average volume of erythrocytes increases, neutrophils are also altered — hypersegmented, pancytopenia is often observed. Vitamin B12 is a cofactor of the enzyme homocysteine methyltransferase, involved in the conversion of homocysteine to methioin. Methionine is important for the synthesis of phospholipids and the myelin sheath of neurons, therefore, 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, manifested by neurological disorders.

Accumulation of homocysteine is a risk factor for the development of atherosclerotic changes. Cyancobalamin, 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., 73 years old, was admitted to the department on 16.01.2019 with complaints of pronounced weakness, rapid fatigue of the leg muscles, which limited mobility to 50-60 meters when walking and one flight of stairs when climbing stairs, pronounced shakiness when walking, decreased appetite, a constant feeling of dry mouth.

Until January 2019, he felt satisfactory, engaged in physical education, regularly participated in urban sports competitions in his age group, freely walked up to the fourth floor. On January 6, against the background of severe short-term stress, I noticed rapid fatigue when walking or doing ordinary homework, and I hardly climbed to the fourth floor. Three days later, he weakened sharply: he barely walked 50-60 meters, climbed the stairs with support.

Previously, he was observed for chronic gastritis, did not follow a diet. In 2007, fibrogastroduodenoscopy revealed hyperplasia of the subcardial part of the stomach. In addition, fatty hepatosis, microliths and kidney cysts were previously diagnosed.

Upon admission, the condition is of moderate severity. The skin is jaundiced in color, 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 wheezes. The heart rate is 76 per minute. The heart tones are clear, rhythmic. Blood pressure 125/80 mmHg. The tongue is pink, shiny. The abdomen is painless. The liver along the edge of the costal arch, the dimensions according to Kurlov are 9 × 8 × 7 cm. The spleen was not palpated. Defecation and urination are not impaired.

Neurological status: the patient is conscious, oriented, and in contact. Pupils are round, narrow, D=S. Photoreactions (direct and friendly) are sluggish, symmetrical. Pupil response to convergence and accommodation is reduced. There is no movement of the eyeballs in full, no diplopia or nystagmus. The face is symmetrical. The tongue is in the middle line. Swallowing and phonation are not impaired. The symptoms of oral automatism are positive on both sides. The gait is ataxic. Muscle strength is 5 points. Deep reflexes are low, equal. Muscle tone is diffusely reduced. On the left is a positive symptom of Babinsky. He performs coordination tests with light intention on both sides. Romberg's pose is unstable. There are no meningeal signs. On the left, hypesthesia from the level of the upper third of the lower leg with a hyperpathic component. The articular-muscular feeling in the III–V fingers of both feet is reduced.

Clinical blood test at admission: hemoglobin 72 g/ l, erythrocytes 1.73 ×1012/ l, leukocytes 4.45 × 109/ L, n/a 2%, s/I 61.2%, eosinophils 2.16%, monocytes 8.82%. basophils 0.6%, lymphocytes 25.2%, platelets 159 × 109/l, c. p. 1.25, cf. volume. er. 113 mm3, cf. sod. hem. in er. 41.7 pg, hematocrit 19.5%, ESR 24 mm/h, erythrocyte anisocytosis +++ (microcytes, macrocytes, megalocytes), erythrocyte poikilocytosis ++ (schizocytes, teardrop-shaped erythrocytes), segmented neurophils with hyperpigmented nuclei.

Urine analysis: specific gravity 1025; acid reaction, urobilinogen 66.0 mmol/l, bilirubin 8.6 mmol/L, cylinders were not detected; protein, sugar, ketone tala, leukocytes, erythrocytes, bacteria were not detected.

Biochemical blood analysis: total bilirubin — 40.8 mmol/L, blood urea — 5.6 mmol/L, blood creatinine — 101 mmol/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 mmol/L, aspartate aminotransferase—31Ed/L,alanine aminotransferase — 29 Units/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 mmol/l, homocysteine — 17.0 mmol/l, thyroid hormones: T4 sv. — 15.2 pmol/l, TSH — 0.72 mEd / l, At-TPO < 3 Units /l. Coagulogram: ACTV — 27 s, fibrinogen — 2.33 g/l, INR — 1.31. No pathology was detected in blood tests for on-comarkers.

ECG: sinus rhythm, horizontal position of the electrical axis of the heart, intraventricular conduction retardation, signs of left ventricular hypertrophy.

Chest X—ray - without pathology.

FGDS: distal non-erosive esophagitis of the 0th degree of severity, cardia insufficiency, atrophic focal gastritis of the antrum of the stomach, subatrophic diffuse bulbitis.

Colonoscopy: acute splenic angle, chronic internal hemorrhoids, pathological changes up to the distal part of the transverse colon were not detected.

Sternal puncture was not performed in the conditions of the rehabilitation department.

Clinical diagnosis: "severe acquired B12 deficiency anemia; hemolytic jaundice; funicular myelosis; glossitis."

Prescribed treatment: 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 cyanocobalamin injection, the severity of weakness decreased, the patient could walk more than 400 meters without stopping, and on the third day he began to exercise.

On the third day, a clinical blood test: hemoglobin 106 g / l, er. 2.68 × 1012 / l, l. 6.8 × 109 / l, n/a 1%, s/a 66.2%, eos. 1.6%, mon. 7.6%, base. 0.5%, lymph. 23.6%, tr. 300 × 109/l, c. p. 1.18. sr. volume er. 121 mm3, cf. sod. hem. in er. 39.7 pg, hematocrit 32.4%, ESR 8 mm/h, erythrocyte anisocytosis ++ (microcytes, macrocytes), poy- kilocytosis of erythrocytes ++ (schizocytes, teardrop-shaped erythrocytes), segmented neurophils with hyperpigmented nuclei, reticulocytes 12%.

A week after the start of treatment: hemoglobin: 115 g/l, er. 2.9 × 1012 /l, l. 6.3 × 109/l, n/I 1%, s/I 59.2%, eoz. 2.1%, mon. 6.5%, base. 0.8%, lymph. 31.6%, tr. 292 × 109/l, c. p. 1.15, cf. volume er. 120 mm3, cf. sod. hem. in er. 38.6 pg, hematocrit 36.2 %, ESR 4 mm/h, erythrocyte anisocytosis +, erythrocyte poikilocytosis +, segmented neurophils with hyperpigmented nuclei, retinoculocytes are 14%. By this time, the patient's muscular strength in the legs had increased, motor activity had recovered, and sensitive disorders had regressed.

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, c. p. 0.99, hemato crit 45.9%, ESR 2 mm per hour, n/a 1%, s/I 57.8%, eos. 2.91%, mon. 5.54%, base. 0.7%, reticulocytes 0.2%. Biochemical blood test: homocysteine — 13.6 mmol/l, vitamin B12 — 920 pg/ml.

Thus, the diagnosis of B12-deficient 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 articular-muscular feeling, and sensitive ataxia. A good clinical response of the body as a whole and the red germ of hematopoiesis in particular to the administration of cyanocobalamin confirmed the correctness of the established diagnosis.

The peculiarity of this case was an acute, sudden onset in an elderly patient against the background of well-being, general well-being, the detection of a sufficiently pronounced decrease in hemoglobin in the blood, which, apparently, developed gradually, without leading to decompensation, and only as a result of severe stress was clinically manifested, the presence of both macrocytes 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 inclusion, possibly final, of compensatory mechanisms.

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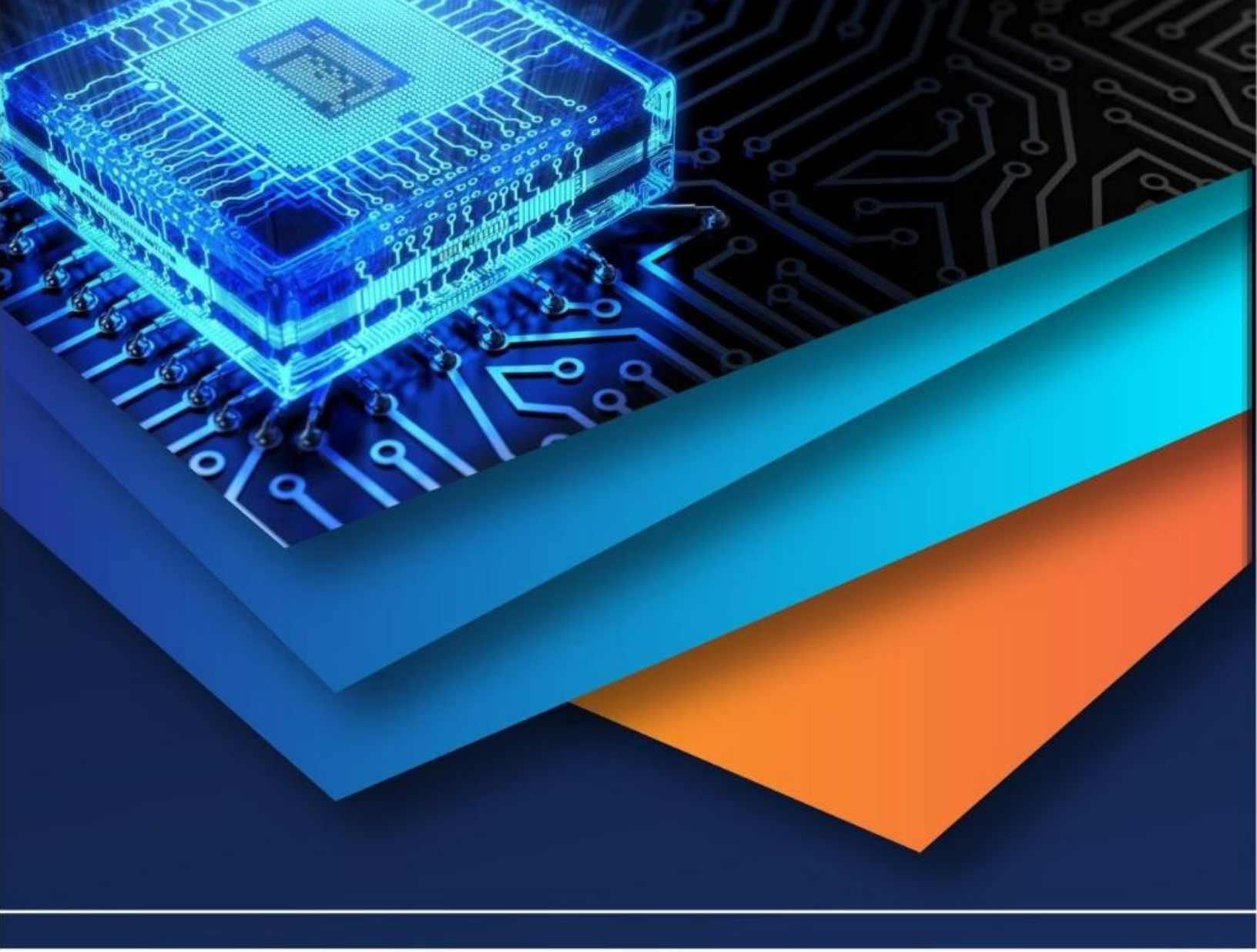

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