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CLINICAL SIGNIFICANCE OF VITAMIN B12 DEFICIENCY IN PATIENTS WITH DIABETIC POLYNEUROPATHY

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Introduction. Vitamin B12 is a co-enzyme of two important reactions: 1) the formation of succinic acid from fatty acids and amino acids; 2) the formation of homocysteine and methionine, which are necessary for the synthesis of choline, phosphatidylcholine, as well as adrenaline, creatine and carnitine. In addition, during the second reaction, folic acid is retained in the cell and thus adequate nucleic acid synthesis is ensured.

Vitamin B12 deficiency is biochemically accompanied by an increase in the level of methylmalonic acid (MMK) and homocysteine, and a decrease in the level of methionine and folic acid. Clinically, vitamin B12 deficiency is manifested by megaloblastic anemia, funicular myelosis, distal paresthesias, increased tendon reflexes, the appearance of ataxia, disorientation, hallucinations and memory impairment. Vitamin B12 deficiency and its accompanying hyperhomocysteinemia and elevated levels of methylmalonic acid cause sensory polyneuropathy, very similar to diabetic neuropathy. Patients with concomitant vitamin B12 deficiency have also been noted to worsen the course of diabetic neuropathy. On the other hand, prolonged use of Metformin is associated with the development of vitamin B12 deficiency. The aim of our study was to investigate the relationship of clinical manifestations of diabetic polyneuropathy with vitamin B12 levels in patients with type 2 diabetes mellitus.

Materials and methods. We examined 35 patients with type 2 diabetes receiving inpatient treatment at the clinic of the Republican Specialized Scientific and Practical Medical Center of Endocrinology. Mean age 55.9 ± 11.5 years, mean experience of diabetes 9.2 ± 7.7 years. All patients received Metformin at an average dose of 1690 ± 531.9 mg. All patients were examined by a podiatrist for the presence of diabetic polyneuropathy (DPN). The level of vitamin B12 was determined by the chemiluminescence immunoassay method.

Results. 14% of patients were diagnosed with stage 1 diabetic polyneuropathy, 77% - stage 2, 9% - stage 3. Analysis of the data showed a trend towards a decrease in the level of vitamin B12 in persons with reduced sensitivity compared to persons with preserved sensitivity: 243.85 ± 117.8 versus 336.66 ± 98.3 pg/ml for pain sensitivity; 299.1 ± 84.3 versus 326.9 ± 121.2 pg/ml for tactile sensitivity; 297.0 ± 175.2 versus 357.0 ± 134.9 pg/ml for temperature sensitivity; 117.8 ± 73.3 pg/ml for persons with vibration sensitivity from 0 to 3 points versus 522.5 ± 188.2 pg/ml for persons with vibration sensitivity from 4 to 6 points (p<0.05). Also, among persons with a decrease in tendon reflexes, the level of vitamin B12 was lower (197.0 ± 78.5 pg/ml) compared with persons with preserved reflexes (316.2 ± 117.1 pg/ml).

Conclusion. The presence of clinical manifestations of polyneuropathy was associated with a trend towards a decrease in the level of vitamin B12 in the blood in patients with type 2 diabetes mellitus taking Metformin, a significant decrease in the level of vitamin B12 was shown with reduced vibration sensitivity. Patients with clinical manifestations of diabetic polyneuropathy should measure vitamin B12 levels.