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Neurologic complications due to vitamin deficiencies after bariatric surgery

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Abstract. Bariatric surgery is the most effective method of treating morbid obesity, but after its performance the patient may develop vitamin deficiency. Against the background of insufficient food intake and absorption disorders after the surgery, the deficiency of vitamins B1, B6, B12 and vitamin E is observed first of all, which leads to complications in the form of central and peripheral nervous system dysfunctions. The most pronounced changes of the nervous system are observed with vitamin B1 deficiency in the form of polyneuropathy and severe Wernicke's encephalopathy. Vitamin B12 deficiency is clinically manifested by a picture of funicular myelosis and polyneuropathy. Insufficient intake of vitamin B6 and vitamin E leads to polyneuropathy. Only the early start of treatment guarantees a favorable outcome of the neurological deficit.
Keywords: *obesity, bariatric surgery, vitamin deficiency, Wernicke's encephalopathy, polyneuropathy.*

Materials and methods: analysis of the literature.

Results: as defined by the International Federation of Obesity and Metabolic Disorders Surgery, obesity is a chronic, lifelong, multifactorial, genetically determined, life-threatening disease caused by the accumulation of excess body fat, leading to serious medical, psychosocial, physical and economic consequences [1]. The problem of excessive body weight (ABW) is acute throughout the world. According to the WHO newsletter of January 2015, more than 1.9 billion people over the age of 18 have excessive body fat, of this number 600 million people are obese [2].

Treatment of obese patients includes therapeutic methods and surgical treatment. Non-surgical approaches to the treatment of patients with high BMI are limited to limiting the energy value of the food consumed, lifestyle changes, the use of moderate physical activity, and drug therapy. Currently, only orlistat, an intestinal lipase inhibitor, is

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approved for long-term (for a year or more) pharmacotherapy of obesity [3]. Surgical treatment can reduce 50-75% of excess body weight, corresponding to 20-40 kg of initial body weight, and reduce BMI by 10-15 kg/m2. After surgical treatment, body weight can be maintained for an average of 16 years [4].

The surgical treatment of obesity or bariatric surgery (from the Greek baros - heavy, obese, weighty) is aimed primarily at treating patients with obesity. All bariatric surgeries can be divided into 3 groups: malabsorptive, restrictive, and combined. Malabsorptive surgeries cause decreased absorption of nutrients by reducing the absorption area due to resection of the small intestine. Restrictive surgeries reduce gastric volume. Combined surgeries are a combination of the restrictive and malabsorptive component.

Restrictive operations on the stomach (gastric banding, vertical gastroplasty) are more physiological. After their performance sufficient effect of weight reduction, low incidence of short-term and long-term postoperative complications, absence of undesirable metabolic consequences with a minimal amount of substitution therapy are noted. Complex combined surgeries (gastros bypass surgery, biliopancreatic bypass surgery) provide a more significant and sustainable long-term result, more effectively affect carbohydrate and lipid metabolism. Combined surgeries enable patients to eat comfortably, but prevention of undesirable metabolic disorders necessitates lifelong substitution therapy [4].

Risk factors for postoperative complications: Preoperatively, overweight patients may already have micronutrient deficiencies. Between 35 and 80% of bariatric surgery candidates have nutritional deficiencies. The most common deficiencies are vitamin D (25-96%), folic acid (24%), selenium (14.5%), thiamine (15%), and iron (over 35%). Risk factors for nutritional deficiencies in the postoperative period include inadequate intake of vitamins and micronutrients, gastrointestinal disorders, and alcohol intake. Given the impact of bariatric surgery on vitamin and micronutrient intake, the studies conducted have mainly assessed the level of vitamin and micronutrient deficiencies. Deficiencies of thiamine, B12, folic acid, vitamin D, copper, and vitamin E are most commonly described [5].

Thiamine (vitamin B1). Thiamine deficiency leads to the most pronounced changes in the nervous system. Thiamine deficiency occurs in the postoperative period from 6 to 15

weeks. Manifestations of thiamine deficiency are observed after malabsorptive surgery, but can also develop after restrictive surgery due to reduced food intake. The main causes of thiamine deficiency are inadequate food intake (low intake, non-compliance with diet, patient refusal to take vitamin preparations, indomitable vomiting for several days) and insufficient absorption. Thiamine deficiency causes impaired function of the blood-brain barrier, resulting in vasogenic edema. Disruption of the blood-brain barrier can result from physical processes, such as mechanical opening of endothelial tight contacts with increased vesicular transport. Thiamine deficiency affects the cardiovascular, muscular, central and peripheral nervous system. The most pronounced changes are noted in Wernicke-Korsakov syndrome. In the neuropathic form, mainly the peripheral nervous system is affected, there are processes of demyelination of nerve endings. The earliest to develop is the "burning feet" syndrome, which is characterized by excruciating burning pain and paresthesias in the feet with increased general sweating. Symptoms are localized to the soles of the feet, but may extend to the shins. Hyporeflexia, decreased sensitivity and weakness in the extremities are also noted.

The cerebral form leads to the development of Wernicke's encephalopathy and Korsakian psychosis. Wernicke's encephalopathy manifests itself by a triad of symptoms: ophthalmoplegia, ataxia and cognitive disorders. Changes in mental state in the form of impaired spatial orientation, drowsiness, dizziness, and apathy are the most common. Ophthalmoplegia is rare, horizontal nystagmus is the most common, bilateral decrease in visual acuity, diplopia, paralysis of both lateral or other eye muscles are also noted. Equilibrium disorders may range from mild gait disturbance to a complete inability to stand. Without treatment, as the disease progresses, a picture of Korsakian syndrome develops, with apathy, fixation and retrograde amnesia, confabulation and pseudoreminisciences.

Wernicke's encephalopathy is treated by administering a thiamine solution of 100 mg intravenously or intramuscularly, with more severe deficiency 500 mg intravenously 3 times daily for 2-3 days, then 500 mg intravenously daily until symptoms improve, and then 50-100 mg orally. When thiamine is started rapidly, the eye symptoms regress more rapidly, the ataxia lasts longer, and in about 50% of patients it does not regress completely. Confusion also regresses slowly.

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Cyanocobalamin (vitamin B12). Vitamin B12 deficiency develops several years after bariatric surgery because long-term stores of it are retained in the liver. Vitamin B12 stores in the liver are sufficient to meet the physiological needs of the body for 3-5 years after the gastric internal factor disappears, but in the absence of the hepatic-intestinal circuit, this period is shortened to a year or a few months.

Vitamin B12 deficiency is observed after resection of the stomach and part of the small intestine (combined bariatric surgery). This surgery results in decreased or absent production of hydrochloric acid, decreased production of intrinsic factor by gastric cells, and decreased number of cells with receptors to the vitamin B12-internal factor complex. In addition, vitamin B12 deficiency develops in pancreatic insufficiency because there is an insufficient amount of the enzyme that releases B12 from the carrier protein and a calcium deficiency that is necessary to connect the vitamin B12-internal factor complex to the receptor. Vitamin B12 is essential for normoblastic hematopoiesis, epithelial cell synthesis, nervous system function by affecting myelin formation, growth, and tissue regeneration. Vitamin B12 deficiency leads to damage to the myelin sheath of cranial and peripheral nerves. Often in the early stages of the disease there are neurological disorders, and more often peripheral nerves are affected. Paresthesias, tingling and stiffness in the legs appear. Changes in the spinal cord (funicular myelosis) occur somewhat less frequently. At first, the posterior columns are affected, deep sensitivity in the legs is lost, and ataxia develops. Later the lateral columns are affected, spasticity, hyperreflexia, positive Babinski reflex appear. Drowsiness, general fatigue, irritability, moderate depression or paranoid syndrome often develop. Neurologic symptoms of vitamin B12 deficiency include peripheral polyneuropathy, optic atrophy, and mood disorders. Neurologic manifestations are the only marker of vitamin B12 deficiency in 16 to 25% of cases. Normal plasma levels of vitamin B12 range from 200 to 750 pg/ml. In neurological practice for treatment of pathology caused by vitamin B12 insufficiency high doses of vitamin B12 - 200 micrograms are used, since its pronounced analgesic effect and ability to activate resynthesis of myelin, including in damaged parts of the nerve, are noted when using high doses of this vitamin. Cyanocobalamin is usually administered

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parenterally - subcutaneously, intramuscularly, intravenously.

Vitamin B6 (pyridoxine). Vitamin B6 is absorbed in the small intestine, some of it is deposited in internal organs and primarily in the liver. Vitamin B6 is involved in the synthesis of neurotransmitters, so it is important for the normal function of the central and peripheral nervous systems. In adults, vitamin B6 deficiency is manifested by numbness, paresthesias, burning pains in the legs, which may then transfer to the arms. Neurological examination reveals signs of distal polyneuropathy with decreased deep reflexes, mild decrease in muscle strength in the distal limbs, decreased sensitivity, ataxia. Plasma level of vitamin B6 is normal at 8.7-27.2 mcg/L. In manifestations of vitamin B6 deficiency the drugs are given orally in a dose of 10-50 mg/day.

Vitamin E. Vitamin E deficiency occurs 6-12 months after surgery, but can develop even after several years. Vitamin E is absorbed in the upper small intestine and requires bile acids and fatty acids for absorption. Vitamin E deficiency develops in patients with impaired absorption (in malabsorptive and mixed bariatric surgery). Neurological manifestations of vitamin E deficiency include ataxia, hyporeflexia, loss of proprioceptive and vibratory sensitivity. Neurologic examination may also reveal dysarthria, nystagmus, and ophthalmoplegia. Peripheral nerve conduction studies reveal sensory axonal neuropathy. Diagnosis is made on the basis of serum α-tocopherol measurements (normal 5-18 mcg/L), but serum vitamin levels may be normal even if there are clinical symptoms of vitamin E deficiency. Treatment with vitamin E begins with a dose of 400 IU 2 times daily enterally, with severe disorders using intramuscular preparations. Neurological complications. The most common complications of the nervous system after bariatric surgery against a background of vitamin deficiency are polyneuropathies, neuropathies (tunnel, mononeuropathies), encephalopathy.

Conclusion: Due to the increase in the number of bariatric surgeries, the possibility of encountering these patients by physicians of various specialties, including neurologists, is increasing. As a consequence, it is necessary to increase doctors' awareness of the peculiarities of managing these patients, and the possible side effects of surgery in both the early and delayed postoperative periods. It is necessary to involve a neurologist in the management of the patient in

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the preoperative and postoperative period for the earliest detection of symptoms of neurological deficit and the earliest possible correction of neurological disorders to prevent serious complications such as Wernike's encephalopathy and severe forms of polyneuropathy. It should be remembered that neurological disorders after bariatric surgery against a background of vitamin deficiency may be of a sterile nature, with normal levels of vitamins in the blood plasma. This necessitates close examination by a neurologist, because even with full treatment, the regression of neurological symptoms is incomplete when a complete clinical picture of neurological deficit develops.

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