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THE COURSE OF DIABETIC POLYNEUROPATHY IN THE COMORBID DEFICITIES OF THE BODY (CLINICAL CASE)

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ABSTRACT

Today, the incidence of diabetes mellitus (DM) in the population is increasing at very high rates worldwide. Diabetic polyneuropathy (DPN) is the most common complication of diabetes mellitus (DM) in the peripheral nervous system. A common clinical form of DPN is a distal symmetrical, mainly sensory, polyneuropathy. The basis of the pathogenesis of DPN is chronic hyperglycemia, which leads to the accumulation of excess intermediate products in various organs and tissues, including damage to the vasa nervorum, which is involved in the blood supply of peripheral nerves. This article examines the deficiency states of the body in the case of diabetic and comorbid polyneuropathy.

Key words: diabetic polyneuropathy, treatment, therapy, study.

INTRODUCTION

Distal polyneuropathy is one of the most frequent chronic complications of DM and one of the main causes of morbidity and mortality in DM in general and among elderly patients with DM in particular [2]. Distal symmetrical polyneuropathy is an example of diffuse PN and the most common form of diabetic PN, detected in more than 50% of patients with DM in general and in 20% of cases at the time of diagnosis of DM [3]. In patients with DM, chronic symmetrical PN is an example of a group of "typical" DNP: it develops against the background of prolonged chronic hyperglycemia, is associated with metabolic disorders (accumulation of glycation end products, oxidative stress, lipotoxicity),

cardiovascular risk factors, hyperhomocysteinemia, microangiopathies (nephropathy, retinopathy). As a result of metabolic damage to small blood vessels, nerve fibers do not receive enough oxygen and nutrients. In addition, the end products of excessive glucose metabolism have a negative effect on nerve fibers. In the case of diabetic polyneuropathy, the pathological process affects all nerves: sensitive, motor, vegetative - this means that almost all small nerves are affected [1-15]. If the process affects only certain nerves, we are talking about neuropathy - a condition that precedes polyneuropathy. Damage to sensitive nerves is manifested by symptoms such as a violation or loss of pain and temperature sensitivity. As a result, the patient begins to feel numbness, burning, tingling or crawling goosebumps in the absence of external stimuli. Diabetes mellitus is not the only cause of polyneuropathy, there are other causes, such as certain autoimmune diseases or vitamin B1, B12, folic acid deficiency, which can also affect the functioning of important nerves. Often, long-term use of hypoglycemic drugs leads to various disorders of the gastrointestinal tract accompanied with hypo or vitamin deficiency.

Metformin is one of the most widely used hypoglycemic agents for oral administration. An undesirable side effect of treatment with metformin and other biguanides is a violation of vitamin B12 metabolism associated with a decrease in serum levels of B12 vitamers, especially against the background of folic acid deficiency. The resulting hypovitaminosis B12 is accompanied by peripheral polyneuropathy, asthenia and, if taken for a sufficiently long time, can lead to megaloblastic anemia, an increased risk of depression, cerebrovascular and neurodegenerative diseases.

Clinical observation

Patient N., 62 years old, turned to the neurological department of the clinic of the Bukhara State Medical Institute. The patient has insomnia, dryness of the skin of the feet, brittle nails, pain when touching the skin of the feet, instability when walking, increases in the dark and causes periodic falls. It is known from the anamnesis that 10 years ago, during the annual medical examination, the patient was diagnosed with hyperglycemia (up to 10.6 mmol/l) and glucosuria (up to 2 mmol/l). Type 2 diabetes was established, and therefore the patient was recommended a diet in combination with glycemic control. The patient faithfully followed all the doctor's recommendations, but 5 years ago there was numbness, a tingling sensation in the feet, soreness when the skin of the feet touches the bed linen. During laboratory examination, decompensation of carbohydrate metabolism was noted, in connection with which Metformin was prescribed at a dose of 1500 mg / day, against the background of which the glycemic level decreased to 6.5-8.8

mmol / l, HbA1c indicators did not exceed 7.5%, the severity of sensory disorders decreased. After 4 years, instability when walking joined, which increases at night and often leads to falls. During the examination in the clinic, an increase in body weight was noted (BMI = 28.6, obesity), blood pressure was 130/90 mm Hg, pulse was 76 beats / min, rhythmic, dry skin of the shins and feet was present, brittle nails. Neurological examination revealed the following: cranial nerves without pathology, weakness in the extensors of the feet (4 points), inability to walk on the heels, tendon reflexes from the hands are reduced, knee and Achilles reflexes are absent, reduced pain and temperature sensitivity from the level of the middle thirds of the shins, lack of vibration sensitivity in the feet, its decrease from the level of the rib arches, violation of musculoskeletal feeling in the toes. In the Romberg trial, the patient is unstable when closing her eyes. Thus, the patient had the following neurological syndromes: polyneuropathic syndrome in the form of distal symmetrical, mainly sensory polyneuropathy, as well as sensitive ataxia syndrome.

According to the results of a clinical blood test, moderate hyperglycemia was detected - 7.6-9.2 mmol/l, HbA1c was 7.0%, glucose was not determined in the urine analysis. In the laboratory study, there was also a decrease in the content of B1 (thiamine) in blood plasma to 1.6 ng/ml (norm - 2.1-4.3 ng/ml), B12 - to 127 ng/l (norm - 214-864 ng/l), folic acid - 3.9 ng/ml (norm - 7-45 ng/lml), as well as an increase in homocysteine to 16 mmol / l (the norm is 3.7-11 mmol / l).

The results of ENMG indicated the presence of axonal damage to the sensory and motor fibers of the nerves of the lower extremities. MRI of the thoracic spine (T2-weighted image) revealed two-sided symmetrical areas of hyperintensive signal in the posterior cord of the spinal cord.

Based on the data of anamnesis, examination and additional examination, the following clinical diagnosis was made: Type 2 diabetes mellitus. Diabetic distal symmetrical sensory-motor polyneuropathy. Polydeficiency condition. Subacute combined degeneration of the spinal cord.

The prescribed treatment included hypoglycemic therapy; a diet aimed at reducing body weight; vitamin B12 (as part of the drug Milgamma) intramuscularly at a dose of 1 mg / day for 15 days under the control of neurological status and B12 content in blood plasma; After prolonged use in the form of a tablet of the combined drug Milgamma compositum 2 tablets per day in for 8 weeks; preparation of alpha-lipoic acid (Thiogamma) at a dose of 600 mg / day; folic acid at a dose of 5 mcg / day. Against the background of the therapy, the manifestations of sensitive ataxia, allodynia regressed, the severity and the zone of violations of surface sensitivity decreased.

Discussion

This clinical observation presents a combination of two clinical syndromes: polyneuropathic, due to diffuse lesions of distal segments of fibers and peripheral nerves, and the syndrome of sensitive ataxia, which is based on a violation of deep sensitivity. Neurological examination revealed a conductive type of deep sensitivity disorders, which indicated a spinal lesion level not characteristic of distal symmetrical diabetic polyneuropathy. This assumption was confirmed by the results of MRI of the thoracic spine and spinal cord, which revealed the presence of a focal lesion of the spinal cord characteristic of funicular myelosis (subacute combined degeneration of the spinal cord). A decrease in the level of B12 in blood plasma in combination with hyperhomocysteinemia confirmed the fact of a deficiency condition, probably drug-induced. Currently, it is known that prolonged use of the hypoglycemic drug Metformin in therapeutic doses can lead to B12 deficiency and to funicular myelosis caused by it with possible hematological disorders in the form of megaloblastic anemia [14]. Hematological changes in B12 deficiency are less common, develop later and their sensitivity is less than neurological, which, in turn, are polymorphic and include clinical manifestations of encephalitis, myelo, polyneuropathy. In the treatment of funicular myelosis, it is necessary to focus on the neurological manifestations of the deficiency condition, and in cases of prolonged use of Metformin, regular monitoring of the level of B12 in blood plasma is recommended [8]. Thus, a patient with type 2 diabetes developed neurological disorders of various nature: vascular and metabolic disorders in the form of distal symmetrical DPN and deficient, manifested by symptoms of subacute combined degeneration of the spinal cord.

In the described clinical case, the effectiveness of complex therapy of a patient with type 2 diabetes with DPN and a polydeficiency condition was demonstrated. In addition to optimal hypoglycemic therapy and correction of risk factors for the progression of DPN, it is necessary to influence various links in the pathogenesis of this nosology. Timely detection and correction of vitamin B deficiency, as well as the appointment of anti-oxidant therapy can slow the progression of DPN, reduce the severity of clinical manifestations and delay, and in some cases prevent, the development of disability.

Conclusion

DPN is the most common neurological complication of DM, which significantly reduces the quality of life. Subacute combined degeneration of the spinal cord refers to the undesirable effects of Metformin, the drug of choice for the correction of carbohydrate metabolism in cases with type 2 diabetes. A thorough clinical analysis of neurological symptoms followed by the use of

informative laboratory and instrumental diagnostic methods ensures timely detection of combined pathology and the choice of pathogenetic treatment methods.

Thus, given the rather extensive formation of clinical manifestations of DPN in a patient with a long history of type 2 diabetes, the presence of classical diabetic DPN is beyond doubt. Nevertheless, in this observation, the presence of pathogenetically significant pathology was revealed — vitamin B1, B12 deficiency and folic acid deficiency. A significant subjective positive dynamics in the manifestations of DPN, general well-being, quality of life was noted against the background of achieving the target levels of vitamins B12, which indirectly indicates the dominance of clinical manifestations of vitamin-deficient conditions. This observation underlines the importance of the well—known thesis that the diagnosis of DPN is a diagnosis of exclusion. Indeed, the "diabetic" genesis of neuropathic symptoms and signs in a patient with a long history of DM is a priority diagnostic assumption. However, it should be remembered about the significant prevalence in some groups of patients of other diseases, in the clinical manifestations of which DPN is included, which can eventually form the phenomenon of comorbid pathology. Of course, the lack of clear recommendations for the diagnosis and therapy of some of the conditions discussed above makes it difficult to form a unified approach to the examination of a patient with manifestations of DPN. The more important are the skills of general clinical examination of the patient and a comprehensive therapeutic approach.

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