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CASUISTIC PAPER

Patrycja Mucha [1(ABD)], Natalia Leksa [1,2(ABDFG)], David Aebisher [2(DFG)], Sabina Galiniak [2(DFG)], Seweryn Bartosz [1(FG)], Dawid Leksa [3(FG)], Dorota Bartusik-Aebisher [2(DFG)]

Generation of retractive spine circuits in the process of vitamin B12 deficiency

[1] Department of Neurology, MSWiA Hospital, Rzeszów, Poland
[2] Medical College of Rzeszów University, Rzeszów, Poland
[3] Rzeszów Center for Vascular and Endovascular Surgery, Rzeszów, Poland

ABSTRACT

Introduction. Subacute degeneration of the posterior and lateral spinal cord is a rare neurological complication of B12 avitaminosis.

Aim. In this paper, we present the case of a 65-year-old man who, in the course of long-term vitamin B12 deficiency associated with atrophic gastritis, developed a severe set of neurological symptoms that are part of the retinal spinal cord degeneration with characteristic features in MR imaging of the cervical spine in the form of an inverted "V mark" in axial images and typical localization in the spinal cord.

Description of the case. After careful analysis of the syndrome and making an early diagnosis, parenteral vitamin B12 supplementation began, resulting in improved neurological status, laboratory parameters as well as regression of changes in magnetic resonance imaging.

Conclusion Significant symptoms of this syndrome are paresthesia in the distal parts of the limbs, ataxia, spastic paresis.

Keywords. ataxia, B12 avitaminosis, retinal spinal cord degeneratio

Introduction

Vitamin B12 belongs to the water-soluble B group vitamins, this compound is an important cofactor in the metabolism of carbohydrates, fats, amino acids and fatty acids in the human body. The daily requirement for this vitamin is 2 μg/day and 2.6-2.8 μg in pregnant women, but the guidelines for the recommended doses vary from country to country. Cyanocobalamin is stored in the liver, and its deficiencies may appear 3-6 years after taking the last dose with a loss of more than 90% of this vitamin. Absorption of vitamin B12 takes place in the final section of the small intestine, the ileum, after binding to the internal factor produced by the stomach's parietal cells in the presence of calcium ions.[1-6] Vitamin B12 deficiency causes a wide range of hematological, gastrointestinal and neuropsychiatric disorders.

Corresponding author: Natalia Leksa, e-mail: leksanatalia@gmail.com

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Symptoms of damage to these systems may occur in varying degrees and form different syndromes. A serious complication of vitamin B12 deficiency is polyneuropathy and spinal cord degeneration.[7-20]

Aim

The aim of this paper is to present a case of retractive spine circuits in the process of vitamin B12 deficiency.

Description of the case

A 68-year-old patient was admitted to the Department because of the progressive weakening of the muscular strength of the lower limbs with accompanying imbalances from 3 months. In addition, from two weeks before the party he complained about the feeling of numbness, awkwardness of his hands, he had difficulty fastening his shirt buttons, writing. He negated the occurrence of sphincter disorders and severe sensory disorders. He has not undergone any infection recently. In history, he was chronically treated for type 2 diabetes (metformin), hypothyroidism, and prostatic hyperplasia. In addition, he underwent L5-S1 discopathy surgery about 20 years ago. Neurological examination at admission showed weak precise hand movements on both sides, medium-degree lower limb pyramidal paresis more severe in the right lower limb with Babinski's identical symptom, impaired sensation of vibration in the upper and lower limbs both proximal and distal, with normal superficial sensation, paraparetic gait with the help of an elbow crutch.[8-12] MRI of the cervical spine was performed, in which, apart from multilevel degenerative-discopathic changes C3-C7, the longitudinal band of the increased signal in the spinal cord was imaged from the C2-C6 stem in T2 and STIR images, showing no contrast enhancement located in the area of the posterior cords myelopathy with a characteristic inverted "V" image in axial images. Basic laboratory tests showed macrocyte anemia - HB levels 10.7 [g/dl] [min: 13.7 max: 17.5], MCV 109.7 [fl] [min: 79 max: 92.2 F], deep vitamin B12 deficiency <83 [pg / ml] [min: 189 max: 883 F:] with normal levels of folic acid and iron. After completing the patient's medical history, the B12 deficiency was found 4 years ago, the patient stopped supplementation after a few months. Differential diagnostics also included primary demyelination and infectious causes. PMR was obtained in which no oligoclonal bands and anti-Lyme disease anti-Lyme Bodies were found in IgM and IgG. In the PMR general study, no significant irregularities were found. HIV and VDRL tests were negative. Glycated hemoglobin was normal. No anti-aquoporin 4 bodies were found in serum. The ENG study of nerve conduction revealed chronic axonal sensory-motor polyneuropathy. Gastroscopy showed atrophic gastritis, gastroesophageal reflux disease, histopathological grade II metaplasia. Diagnosis of spinal cord degeneration against vitamin B12 deficiency was made. Parenteral vitamin B12 supplementation was implemented for treatment. He was discharged to the Rehabilitation Department for further improvement. The patient was re-admitted to the Department of Neurology after 5 months to re-evaluate and perform follow-up tests: a significant improvement in the neurological condition was clinically observed, no weakening of the muscular strength of the lower limbs or pyramidal symptoms was observed, the patient was able to walk alone, on a broad basis, the study returned attention to ataxia from the lower limbs, as well as persistent disturbances of deep sensation, however, to a lesser extent than previously noted. Laboratory tests showed an improvement in blood count parameters. The MR examination of the cervical spine compared to the previous examination showed a clear regression of the previously described changes in the spinal cord. The patient is currently undergoing maintenance treatment with vitamin B12 1000ug /month. In addition, it awaits the control of the Gastroenterology Clinic.

Discussion

Cord degeneration of the spinal cord refers to degenerative disease of the central and peripheral nervous system damage, pathological changes in the spinal cord more often involve white matter than gray matter. More often than the loss of axons, a symmetrical absence of myelin sheaths is observed, the changes are most noticeable in the posterior and lateral cords.[12-21] SCD is a rare neurological complication of vitamin B12 deficiency in the body, it occurs most often around 50 years of age, this disease is rarer seen in younger people, very rarely in children. Symptoms of severe damage to the nervous system are now rare. Early diagnosis and the possibility of successful treatment reduced the number of patients with cord degeneration.[14] The most common causes of B12 avitaminosis are malabsorption (e.g. pernicious anemia, gastrectomy, bariatric surgery, ileum resection, congenital Castle internal deficiency, gastritis caused by *H. pylori*, Lesniowski and Crohn's disease, non-immunological) chronic atrophic gastritis, chronic pancreatitis, congenital selective absorption disorders, Zollinger-Ellison syndrome, bacterial hyperplasia syndrome, congenital metabolic disorders, including transcobalamin deficiency), interactions between vitamin B12 and chronic medications (e.g. metformin, medications hydrochloric acid secretion, nitric oxide), insufficient food intake (veganism or vegetarianism, malnutrition, alcoholism).[5,6,11,19] Clinical symptoms of cobalamin deficiency usually appear in the chronic period of 3-5 years after the occurrence of absorption disorders or lack of it in food. The body has a large supply of vitamin B12 estimated at about 1-10mg (mainly liver) and a mechanism for saving this vitamin, which consists

in the reabsorption of bile secreted cobalamin from the gastrointestinal tract.[5] Pearce and other colleagues also emphasize the important role of nitrous oxide in their work, which due to the possibility of rapid inactivation of vitamin B12 may cause the development of SCD symptoms in a few weeks.[4] To quote Merrit, a single dose of anesthetic in a susceptible person or chronic exposure usually associated with intoxication with gas available in dental offices or other treatment rooms or commercially available may lead to vitamin B12 deficiency.[17] Haematological disorders are not normally associated with nitrous oxide abuse. Vitamin B12 deficiency in most patients is asymptomatic, it is thought that 40% of all patients with vitamin B12 deficiency have neurological problems or symptoms and are often the first symptoms of the disease.[17] The clinical picture of SCD consists of signs of damage to both posterior and lateral scars. Significant symptoms of this syndrome are paresthesia in the distal parts of the limbs, ataxia, spastic paresis. Therefore, there is a combination of pyramidal symptoms with sensory symptoms of varying severity, namely in some cases pyramidal over sensory symptoms predominate and in other sensory symptoms over pyramidal symptoms. Sphincter disorders are rare, usually occur late in untreated patients. Middle darkness without changes at the fundus and optic atrophy are very rare symptoms.[13,14] In the presented clinical case, the patient due to malabsorption due to atrophic gastritis confirmed by gastroscopy and long-term vitamin B12 deficiency developed a severe mixed neurological disorder syndrome, in which spastic paresis of the lower limbs dominated with pyramidal symptoms with less pronounced impairment of deep sensation and ataxia . The above syndrome with symptoms of severe damage to the nervous system is currently rare.[12-15] Diagnosis of SCD is primarily based on demonstrating a reduced level of vitamin B12, some patients with low levels of vitamin B12 do not have a deficiency of it, then additional tests may be useful. Methylmalonic acid and homocysteine accumulate as a result of impaired cobalt-dependent biochemical reactions. Abnormally high levels of both of these substances can be found in serum in more than 99% of patients. These studies have limited utility in some populations, as increased homocysteine occurs in hereditary hyperhomocysteinemia, and increased concentration of methylmalonic acid in patients with renal failure. Of the patients with neurological symptoms, only 20% have severe anemia. Both hematocrit and mean corpuscular volume may be normal, although macrocytic anemia is a classic abnormality found in this deficiency.[17] Other ancillary tests used in the diagnosis of cobalamin deficiency may be electoneurographic tests or magnetic resonance imaging (MR).Regarding magnetic resonance imaging, only 11.1% to 36.7% of patients show characteristic abnormalities.[3] The results of MRI in SCD are extremely diagnostic and even pathognomonic. MR shows a very typical pattern with hyperintensive changes in sequences. T2 dependent usually limited to the posterior and/or lateral columns of the spinal cord, most often located in the range of the lower cervical and thoracic sections. On the axial cross-sections characteristically adopting the characteristic "inverted V" sign, "pair of binoculars", "dot sign".[3-10] In the presented clinical case of a patient of our Ward in a radiological examination, the pathological band of the increased signal was depicted in the T explain sensory-motor disorders occurring in a neurological examination.[11-17] The characteristic location of lesions in the spinal cord is typical for B12 avitaminosis. As mentioned in the work of Ravina et al. MR also seems to be a good diagnostic tool also for assessing the control effectiveness of treatment.[10] It was found that younger age <50 years with shorter disease duration, changes in MR involving <7 core segments are associated with a higher probability of a positive treatment outcome. However, diagnostic delay or late start of treatment can cause permanent spinal cord injury with little or no improvement. Another auxiliary study in B12 avitaminosis is an electroneurographic study, in which features of sensory-motor polyneuropathy with a predominance of sensory symptoms can be found, and the ENG picture corresponds to axonal neuropathy.[17-18] Also, the patient presented above revealed characteristic features in the ENG study. Peripheral neuropathy may be mistakenly diagnosed as diabetic neuropathy, and further failure to recognize the cause may further damage central nervous system function.[5] Differential diagnosis of SCD can be broad and can include nutritional deficiencies (copper deficiency, vitamin E), demyelinating processes (MS), infectious causes (HIV vaculopathy, herpes), inflammatory processes (sarcoidosis), ischemic and cancer (astrocytoma and ependymomas), syndromes hereditary (Fredreich's ataxia, Leukoencephalopathy with brainstem and spinal cord involvement).[9] In the presented patient in differential diagnosis, mainly demyelinating and infectious processes were taken into account, however, macrocytical anemia with deep vitamin B12 deficiency and characteristic changes in MR imaging present in the basic studies with significant improvement after the treatment was associated with confirmation of the diagnosed diagnosis of cord degeneration of the spinal cord and did not require further differentiation. Treatment of vitamin B12 deficiency should be started as early as possible. It should include causal and substitution treatment. For the degeneration of the spinal cord, we use cyanocobalamin at a dose of 1000 μg administered intramuscularly every other day until the symptoms have disappeared, then a monthly maintenance dose of 1mg for life, in addition to vitamin B12 deficiency therapy, appropriate dietary recommen-

dations should also be implemented, i.e. a rich diet in animal protein. [6,19,20] After parenteral administration of vitamin B12, hematological improvement may be evident after 48 hours, there is also a subjective improvement in well-being. Paresthesia is the first neurological ailment that regresses (within the first 2 weeks), cortical and cognitive disorders respond more slowly usually over 3 months, further improvement in clinical condition may take up to a year. If there is no response to treatment after 3 months, the disorders are probably not due to vitamin B12 deficiency. About half of the patients have some abnormalities found in physical examination.[17] In the presented patient, after 5 months of treatment, a clear but incomplete improvement of the neurological condition was found, first of all an improvement in motor functions was achieved, thanks to which the patient is able to move independently, moreover, an improvement in blood morphotic parameters, as well as a marked regression changes in resonance imaging.[18-20]

Conclusion

Subacute degeneration of the posterior and lateral spinal cord is a rare neurological complication of B12 avitaminosis. In this paper, we present the case of a 65-year-old man who, in the course of long-term vitamin B12 deficiency associated with atrophic gastritis, developed a severe set of neurological symptoms that are part of the retinal spinal cord degeneration with characteristic features in MR imaging of the cervical spine in the form of an inverted "V mark" 'in axial images and typical localization in the spinal cord. After careful analysis of the syndrome and making an early diagnosis, parenteral vitamin B12 supplementation began, resulting in improved neurological status, laboratory parameters as well as regression of changes in magnetic resonance imaging.

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