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Vitamin B_{12} deficiency as a potential cause of dementia

Vitamin B_{12} (cobalamin) is present in food of animal origin, mainly in meat and liver. It can be found in small quantities in dairy products and eggs (2). Daily vitamin B_{12} requirement is about 5μg. Cobalamin is an extrinsic factor and it connects with intrinsic factor (Castle's), produced in stomach through parietal cells. After the connection it is absorbed in the small intestine (1, 2). The hypovitaminosis B_{12} may be due to disorder of absorption, inadequate supply or excessive waste. The most frequent cause of absorption disorders is the Addison-Biermer's disease (megaloblastic anaemia). There are antibodies directed against parietal cells of the stomach or antibodies against intrinsic factor. Besides, the hypovitaminosis can be caused by stomach diseases (e.g., gastric mucosa inflammation, resection), small intestine diseases (e.g., resection, diverticulosis of intestine, sprue), inborn deficiency or usage of drugs competing in absorption in the small intestine. The inadequate supply of vitamin B_{12} can be due to malnutrition or strictly vegetarian diet. The excessive waste is typical of parasitic disease, e.g., carriers of diphyllobothrium latum (1, 2). It is worth mentioning that inadequate supply is an extremely rare cause of hypovitaminosis B_{12} because systemic store is sufficient for about 30 months (2).

CASE DESCRIPTION

The case of a 31 years old patient, in whom occurred slowness, dysarthria and dysphasia, dysmnesia and concentration failure, was discussed. According to the interview the patient did not apply special diet. He consumed balanced meals. He did not admit any medicines chronically. He denied any addictions. The neurological investigation showed minor degree disorders of alternating movements, disorders of equilibrum and brachybasic gait. The MRI investigation showed inadequate to age, massive cortico-subcortical atrophy of cerebral tissue located symmetrically both in hemispheres of cerebrum and cerebellum (there was not the atrophy progression with relation to the prior investigation done two years earlier). Blood analysis showed: ERY – 4.56 M/ul; HGB – 14.4 g%; MCV – 92.2 fl; MCH –31.5 pg; MCHC – 34.2 g/dl; RDW – 14.8%; HT – 42.1%; WBC – 8420/ul; LYM – 27.4%; NEU – 60.1%; MONO – 6.33%; EOS – 5.56%; BASO – 0.549%; PLT – 289 K/ul. Urine parameters were in normal rate. The level of cobalamin was 111 pg/ml (n. 220–960). There were no abnormalities in pulmonary and cardio-vascular system. Genetic examinations towards spinocerebellar ataxia type 1, 2, 3 (SCA1, SCA2, SCA3) were correct. The cerebrospinal fluid examination was correct. The neuropsychological examination showed bradypsychia, intelligence on average level. There was insignificant intensification of motor failure and dysarthria with relation to the prior examination before one year. HIV test was negative. The gastroscopy showed the pale gastric mucosa with distinct net of blood vessels (in the histopathologic examination without any morphological changes in the body stomach). The patient was treated with cyanocobalamin applied in intramuscular injections, 1000 μg a day. The patient was dismissed with dementia in the course of hypovitaminosis B_{12} and suspicion of megaloblastic anaemia.

DISCUSSION

The clinical picture of hipovitaminosis B_{12} consists of neurological, psychiatric and internal medicine symptoms. Neurological symptoms often predominate and they can be the only ones present in the patient. There can appear at the beginning painful paraesthesia of lower limbs or tottering. Next, there can be found symptoms of posterofunicular syndrome and disorders of position sense, kinaesthesia, vibratory and touch sensibility, finally hypalgesia and temperature hypanaesthesia. There develops polyneuropathy. Among psychiatric symptoms depressive and delusional syndrome, hallucinations, and confusion can occur. Dementia can appear after 3 to 5 years. Then may predominate bradyphrenia and slowness of cognitive processes, disorder of concentration and aprosexia. Internal medicine symptoms can express as the inflammation of tongue mucosa, dyspepsia and diarrhoea (1). The hypovitaminosis B_{12} is treated with cyanocobalamin injections initially 1 mg a day, then with reduced dose after clinical improvement. The therapy must often be continued throughout the life of the patient. The clinical symptoms can improve quickly in answer to the use of vitamin B_{12}, although the clinical state and MRI abnormalities can even improve forty-four months after the beginning of supplementation (4, 5). There is favourable prognosis when symptoms are subjective and treatment is applied early. In case of late supplementation changes can be irreversible (3)

CONCLUSIONS

Easy treatment is very effective when the hypovitaminosis is diagnosed early and changes can be completely reversible but the correct diagnosis can be difficult because there may be multitude of symptoms found in patients in the course of hypovitaminosis.

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SUMMARY

There is a patient case with dementia and brain MRI massive abnormalities, probably in the course of vitamin B_{12} deficiency.

Niedobór witaminy B_{12} jako potencjalna przyczyna zespołu otępiennego

Przedstawiamy pacjenta z procesem otępiennym i masywnymi zmianami w badaniu MRI, nieadekwatnymi do wieku, prawdopodobnie w przebiegu niedoboru witaminy B_{12}.