Neurological manifestations of vitamin B12 deficiency, CNS manifestations

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Vitamin B12 (cobalamin) was discovered at Meck, structural actualization by Hodgkin, Minot and Murphy’s hallmark study on treatment of cobalamin deficiency. Castle’s discovery on the gastric component, intrinsic factor. And this is still the subject of intense research in its role in preventing the irreversible neurological lesions.

A water-soluble vitamin involved in the metabolism of the body. A cofactor of DNA synthesis, metabolism of amino acids and fatty acids, and vital in the normal functioning of CNS. According to current reports, cobalamin deficiency is being most seen in developing countries than developed countries.

The most frequent neurological manifestations of vitamin B12 Deficiency is sub-acute combined degeneration of spinal cord. Along with the symptoms of numbness, paresthesias, ataxia of gait, urinary incontinence and urgency. Other CNS manifestations include optic neuropathy, peripheral neuropathy and Cerebral symptoms such as apathy, depression, dementia, psychosis. An historical study by Reynolds et.al in 1992 founded that the low levels of cobalamin and be related with multiple sclerosis. Cobalamin deficiency can cause hyper homocysteinemia, which can be related to even stroke, MI, Premature atherosclerosis and venous thromboembolism. Laboratory diagnosis for this include serum cobalamin levels, schilling test and deoxyuridine suppression test etc.

Vitamin B12 deficiency and causes both axonal and demyelidine changes, is commonly occurring through the dietary deficiency. Blood smear test and bone marrow test are useful indication for the possible vitamin B12 deficiency.

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