CASE REPORT

Low Cobalamin Levels—An Underestimated Cause of Dementia: A Case Report

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Abstract

Background: Vitamin B12 deficiency is widely known for its hematological and neurological manifestations. Neuropsychiatric manifestations of vitamin B12 deficiency include dementia, delirium, cerebellar ataxia, psychosis, neuropathy, and mood disorders. Dementia caused by vitamin B12 deficiency may not be accompanied by anemia and it may be the sole manifestation of cobalamin deficiency.

Case description: A 70-year-old woman, premorbid normal, presented with rapidly progressive cognitive impairment over a period of 7 months in the form of short-term and episodic memory disturbances, decreased interaction, withdrawn behavior, apathy, wayfinding difficulties, and sleep disturbances. Neurologic examination revealed a mini-mental state examination (MMSE) score of 9/30. Tests for attention and sustained attention were impaired. Speech and language examination revealed reduced word output and impaired animal naming. Both immediate and recent memory was impaired. Lobar examination revealed frontal and temporal involvement. The patient's initial laboratory investigations indicated mildly decreased hemoglobin (10.2 gm/dL) and normal MCV - 98 fL. There were very low levels of vitamin B12 (116 pg/mL). Magnetic resonance imaging (MRI) of the brain showed age-related cortical atrophy. The patient was treated with parenteral methylcobalamin initially then switched to oral cobalamin. After a period of 3 months of treatment, MMSE was reassessed and was 16/30. She was fully oriented and had significant improvement in her memory and behavior.

Conclusion: The role of vitamin B12 deficiency as one of the few treatable causes of dementia, however, is still controversial. The purpose of this case report is to show that if vitamin B12 deficiency remains undiagnosed, serious sequelae may occur. Assessment of B12 levels should be included as a standard evaluation in patients presenting with dementia. Therefore, prevention, early detection, and management of this reversible state are of profound importance.

Keywords: Case report, Cobalamin, Deficiency, Dementia, Elderly, India, Rapidly progressive, Vitamin B12.

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Introduction

Vitamin B12 deficiency is common in developing countries. It can cause neurological disorders like myeloneuropathy, peripheral neuropathy, cerebellar ataxia, optic atrophy, and dementia.[1–4] Dementia caused by a deficiency of vitamin B12 may not be accompanied by megaloblastic anemia and neurological manifestations may be the only presentation in vitamin B12 deficiency. We hereby report a case of cobalamin deficiency presenting with memory and behavioral disturbances for seven months. The patient's neurological status improved with parenteral cobalamin supplementation.

Case Description

A 70-year-old female, homemaker with no formal education, premorbid normal, presented with a progressive cognitive decline for 7 months. Initial signs of her condition included lack of concentration followed by short-term and episodic memory impairment. She had behavioral disturbances in the form of decreased interaction, withdrawn behavior, apathy, and social disinhibition in the form of repeated dressing and undressing in front of others and unconcerned micturition. As symptoms progressed, she required prompting for activities of daily living.

She had a history of getting lost in familiar places and sleep disturbances like increased sleep latency and frequent awakening. She was unaware of her own illness. She did not have a history of hallucinations, hemineglect, delusions, apraxia, or bowel and bladder incontinence. There was no history suggestive of cranial nerve involvement, weakness or sensory disturbances.

A general examination revealed mild pallor. A higher mental function examination revealed mini-mental state examination (MMSE) score of 9/30. Tests for attention and sustained attention were impaired. Speech and language examination revealed reduced word output and impaired animal naming. Comprehension and repetition were intact. Both immediate and recent memory were impaired and remote memory was intact. Lobar examination revealed frontal and temporal involvement. Occipital and parietal lobes were relatively spared. Cranial nerves, motor, sensory,

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cerebellar and extra pyramid system examination were normal. The patient was diagnosed with dementia due involvement of more than one cognitive domain according to DSM-5 criteria.

The investigations revealed reduced hemoglobin of 10.2 gm/dL and MCV of 98 fL while WBC and platelet counts were normal. The cobalamin levels were found to be decreased i.e., 116 pg/mL. Her Liver and renal parameters were normal. The thyroid function test (TFT) and vitamin D3 values were normal. The patient is HIV seronegative. Magnetic resonance imaging (MRI) of the brain revealed age-related cortical atrophy and small vessel ischemic changes (Fazekas grade 2). Her X-ray chest and USG abdomen and pelvis were normal.

The patient was treated with 1500 µg intramuscular methylcobalamin daily for the first week followed by 1500 µg once a week by intramuscularly for the next 6 weeks. She was then started on 500 µg of vitamin B12 orally thrice a day for life.

She was treated with oral supplementations for three months. The MMSE at the end of three months of treatment was found to be 16/30. She had profound improvement in her memory and behavior and was fully oriented.

Discussion

Cobalamin is used as a cofactor by multiple enzymes in deoxyribonucleic acid (DNA) synthesis, fatty acid and amino acid synthesis. In the nervous system, it is required for the production and maintenance of myelin. Hence, its deficiency leads to hematological and neurological disorders. However, in those cases with neurological symptoms, anemia is usually milder and in cases with severe megaloblastic anemia, neurological symptoms or signs are less common.[1,4] The cause of such a relationship has not yet been hypothesized.[2]

Cobalamin deficiency may present only as a neurological manifestation in some cases, which many physicians are unaware of, as doctors usually rely on mean corpuscular volume and mean corpuscular hemoglobin values for the diagnosis, but those changes occur late in the disease. There are postulates which state that even before the serum vitamin B12 levels become deficient there is a deficit of cobalamin in neuronal tissue.[4,5] In these cases as such, homocysteine and methylmalonic acid assays increase the specificity of determining cobalamin insufficiency.[6]

Conclusion

The intent of the case report is to show that cobalamin deficiency is one of the most underdiagnosed diseases. If not treated adequately, it can lead to serious sequelae. The assessment of cobalamin levels should therefore be used in regular evaluation of patients presenting with new onset dementia. Therefore, prevention, early detection, and management of this reversible state are of profound importance.

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