Vitamin B12 Deficiency Manifested As Dementia Without Anemia

S Penupolu, F Aziz, S Kallu, S Doddi

Citation

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Abstract

Vitamin B_{12} deficiency has long been associated with a wide variety of hematological, neurological, and psychiatric disorders. The role of vitamin B_{12} deficiency as one of the few treatable causes of dementia, however, is still controversial. We, here report one patient suffering from cognitive impairment and psychotic symptoms probably related to cobalamin deficiency, who showed clinical improvement after parenteral vitamin B_{12} substitution.

INTRODUCTION

Dementia is a frequently occurring syndrome, especially in the elderly population. The differential diagnosis of the dementia syndrome includes a large number of disorders, from Alzheimer's disease and alcoholic dementia to posttraumatic and vascular dementia. Hypovitaminosis is one of the few disorders causing dementia that are potentially curable today.

The following case report illustrates the importance of vitamin B_{12} deficiency as a possible cause for the development of dementia. Whereas common routine parameters like the serum level of vitamin B_{12} or the Schilling test may not be sufficient to diagnose borderline cobalamin deficiency, newer functional assays may help to exclude vitamin B_{12} deficiency as a cofactor for dementia in the future.

CASE

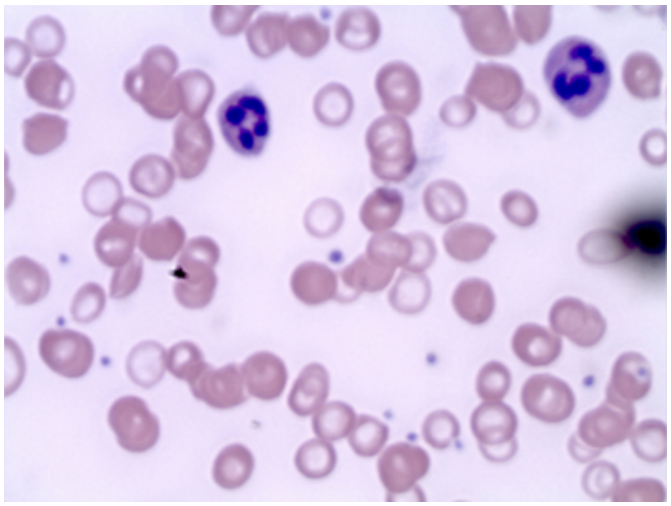
A 58 year old Caucasian male with past medical history of Hypertension and chronic alcohol abuse is brought to the hospital by his girl friend because of his confusion over the last 2 weeks. The patient was in usual state of health, taking care of his daily activities and leading a fairly functional life, when he started showing these symptoms. He became increasingly forgetful and confused. He started to wake up in the middle of the night, dress himself and walk out of his house, and was usually found on the roads by the family. On presentation, patient was found to have a slow mentation, responding very slowly to even simple questions. He could recall his date of birth and date of graduation correctly but could not recall the current year, the president of United States and what he had in breakfast. The only complaint he had was pain in his heels for many years and denied another problem. He scored 10 out of 30 in Mini Mental Status Examination done at the admission. Examination revealed presence of marfanoid features, diminished tendon reflexes and reduced sense of posture and vibration in both lower extremities.

CT scan of head revealed age appropriate cerebral atrophy. Blood work revealed low vitamin B12 levels but no anemia, macrocytosis or hypersegmented neutrophils (Fig 1). Thyroid profile was normal and rapid HIV and RPR were negative. Serum Methyl malonic acid and homocysteine levels were above normal range confirming B12 deficiency.

Patient was started on IM supplementation of Vitamin B12 along with Lorazepam for possible alcohol withdrawal and monitored. Patient started showing gradual improvement in mentation that included improvement in the response to the questions, and attention span. He managed to score 16 out of 30 in a repeat Mini Mental status Examination at end of 10 days.

Figure 1

Fig 1: Normal Peripheral Smear



DISCUSSION

In this case report we presented, cobalamin deficiency is a likely contributor to the neuropsychiatric malfunctioning. In our patient, there was no hematological effect of vitamin B12 deficiency. Cobalamin deficiency was confirmed by a low vitamin B_{12} serum titers. Substitution of vitamin B_{12} together with additional treatment resulted in improved cognitive function, which was documented by psychological re-testing and clinical restoration of orientation of our patient.

Although in our patient, as well as in previously reported cases, the effects of vitamin B_{12} substitution cannot be positively distinguished from the effects of co- medication, supporting therapeutic measures, and retest improvement, there is substantial evidence supporting the crucial involvement of vitamin B_{12} in several pathophysiological conditions affecting the CNS, starting from myelination to transmitter function. Even though the causal relationship between cobalamin deficiency and dementia in individual patients is hard to prove and may often remain circumstantial, subclinical vitamin B_{12} deficiency, which today can be unambiguously identified, is a common condition in the elderly population. Considering the devastating impact of dementia on the quality of life of the individual and also the vast costs, this often incurable condition causes, the proper diagnosis and inexpensive treatment of cobalamin deficiency should not be missed, especially in the early phases of cognitive decline.

Modern diagnostic tools like the measurement of HY and MMA, as well as longitudinal testing of cognitive function and neurophysiological parameters, will help to further define the role of vitamin B_{12} deficiency as a cofactor in the development of dementia and to elucidate why all cobalamin-deficient patients do not develop psychological symptoms.

References

Author Information

Sudheer Penupolu, MD
Jersey Cirt Medical Center, NJ, USA

Fahad Aziz, MD
Jersey Cirt Medical Center, NJ, USA

Swapna Kallu, MD
Jersey Cirt Medical Center, NJ, USA

Sujatha Doddi, MD
Jersey Cirt Medical Center, NJ, USA