Brief Reversible Psychosis and Altered Mental Status in a Patient With Folate Deficiency: A Case Report

To the Editor: Vitamin B_{12} and folate deficiencies can result from inadequate intake, impaired absorption, or increased requirement, or the cause can be multifactorial such as in the setting of chronic alcoholism.[1] Hematologic manifestations can include a megaloblastic, macrocytic anemia.[1] Many individuals with an underlying vitamin B_{12} deficiency are labeled with other psychiatric diagnoses including major depressive disorder, bipolar disorder, and panic disorder.[1] A deficiency of vitamin B_{12} or folate leads to an increased level of homocysteine, a highly toxic metabolite to neural and vascular development. Homocysteine has even been linked to a higher risk of cognitive decline and Alzheimer's disease and a 5-fold increase in the rate of brain atrophy.[2] Our case presents a patient with severe cognitive decline in the setting of a folate deficiency.

Case report. A 49-year-old white male patient of a psychiatric hospital was transferred to the emergency department for complaints of altered mental status. He was refusing to eat and did not sleep at night prior to his presentation to the emergency department. He demonstrated disorganized thoughts, disorganized behavior, and psychomotor retardation. His Montreal Cognitive Assessment (MoCA; English [Original] version, www.mocatest.org) score was 3/30 and he was giving 3- and 4-digit numbers as his phone number, which was a substantial change from his baseline. His past medical history was significant for hypertension and bipolar disorder, which were well maintained with metoprolol and risperidone, respectively. He has had 29 prior psychiatric hospitalizations, mainly related to his continuous substance abuse. At presentation to the emergency department, the patient's vital signs were within normal limits except for a blood pressure of 167/106 mm Hg. Initially, he was noted to have a mild tremor on bilateral upper extremities and to be looking toward the ceiling as if he were seeing things; however, he was nonverbal. He was disoriented to person and time, but aware and oriented to place. No neurologic deficits were documented; however, the examining physician was unable to assess muscle strength, gait, or cerebellar testing because the patient did not cooperate with the physical examination. The full physical examination was otherwise unremarkable. Differential diagnosis included metabolic derangement, encephalopathy, or delirium, which could be secondary to his long-standing history of illicit substance abuse. Noncontrast computed tomography scan of the head and subsequent magnetic resonance imaging were performed and showed no acute changes. Significant laboratory results included minor macrocytosis (mean corpuscular volume of 100.1 μm^3), thrombocytopenia (141×10^3/mL), and hypoalbuminemia (3.6 g/dL). Results of the thyroid panel and creatine kinase and ammonia levels were all within normal limits, and the urine drug screen was negative for substances of abuse. His B_{12} was found to be on the lower end of normal at 341 pg/mL, and he was folate-deficient at 6.8 ng/mL.

The patient was admitted to the hospital and given one B_{12} intramuscular injection and started on a daily regimen of folic acid (1 mg), thiamine, and a multivitamin. He was hospitalized for 2 days, during which he resumed oral food intake and increased his daily activities and communication. At that time, he was discharged back to the psychiatric hospital with instruction to continue folic acid supplementation and to have monthly B_{12} injections. He was alert and oriented to time, place, person, and situation. Mental status examination showed that he was more organized, with no disorganized behavior or hallucinations. His score on the MoCA returned to his baseline of 18/30 prior to admission, and at a later assessment was 19/30, which was appropriate to his baseline for age and education.

In our case, we were presented with a unique situation of severe cognitive decline in the setting of a folate deficiency. In our literature review, we were unable to identify any cases of acute dementia with an associated isolated folate deficiency. While the patient presented with both a vitamin B_{12} and a folate deficiency, the vitamin B_{12} deficiency appears to have been less severe than the folate deficiency. Supplementation with folic acid and B_{12} resolved our patient's symptoms in addition to augmentation with antipsychotics. Prior literature[3] has discussed the role of folate deficiency in psychiatric conditions by mentioning the masking effect of folate on a vitamin B_{12} deficiency and thereby delaying the presentation of macrocytic anemia. Other case reports[4] of cobalamin-responsive psychosis cited normal folate levels. While previous studies[5] have indicated the role of vitamin B_{12} deficiency in psychiatric manifestations, there have been no determinations of the time to irreversible myelin damage.

References

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