Vitamin B12 Deficiency and Depression in the Elderly: Review and Case Report

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To the Editor: Vitamin B12 is a water-soluble essential vitamin. A member of vitamin B complex, vitamin B12 is also called "cobalamin" because it contains the metal cobalt. Vitamin B12 is synthesized by bacteria and is found mainly in meat, egg, and dairy products but lacks a reliable plant source. It is essential for the formation of red blood cells and maintenance of a healthy nervous system as well as for the rapid synthesis of DNA during cell division. It is believed that B12 along with folic acid may help prevent disorders of central nervous system development, mood disorders, and dementias. Megaloblastic anemia is the common and serious illness associated with B12 deficiency, but it is believed that a mild decrease in the B12 level is associated with neurologic and psychiatric problems such as ataxia or mood disturbances.

A common cause of vitamin B12 deficiency is poor intake or absorption. The protein-bound vitamin B12 is released by hydrochloric acid in the stomach during digestion. Once released, B12 combines with the gastric intrinsic factor, and this complex is absorbed in the intestinal tract. Although vitamin deficiencies are relatively uncommon in the Western world, it is estimated that 10% to 15% of individuals over the age of 60 years may suffer from B12 deficiency and it is believed that atrophic gastritis type B, which may afflict 20% to 50% of the elderly, may lead to poor absorption of B12 and folate. As the population ages, nutrition and its impact on the health of the elderly become increasingly important in health maintenance of the elderly. In this report, we describe an elderly woman who suffered from severe psychotic depression who did not improve with conventional treatment but recovered significantly following administration of vitamin B12, illustrating the possibility that vitamin B12 deficiency may play a role in the development of mood disorder.

Case report. Ms A, a 66-year-old married, African American retired teacher, was living with her daughter and granddaughter. She was seen in 2004 by one of the authors (S.H.) in the outpatient clinic following a psychiatric hospitalization for DSM-IV major depressive disorder with psychosis. The patient and the family reported a 6-month history of sad mood and lack of energy, interest, and motivation along with sleep disturbances following the loss of Ms A's foster children. She had no family history or past history of mental illness or substance abuse.

During those 6 months, she stayed in her bed most of the time, was sad and withdrawn, and cried often. She neglected self-care and hygiene and lost about 30 pounds of body weight. In August 2004, she was hospitalized (as noted in the previous paragraph) following worsening of her condition that included agitation, sleeplessness, and a fear that something bad was going to happen to her. She reported no hallucinations or suicidal ideas. She was treated with sertraline 150 mg/d and risperidone 2 mg/d and stayed in the hospital for 10 days.

At the time she was seen in the clinic for follow-up (less than a month after discharge from the hospital), she had a noticeable stiffness in her gait but otherwise was healthy physically. She was well dressed and groomed but exhibited slow psychomotor activity. She appeared withdrawn and was slow in her speech. She denied feeling sad but was tearful and depressed during the interview. She did not exhibit any overt psychotic symptoms but continued to have a vague fear that something bad might happen to her. She had no suicidal thoughts but was generally hopeless about herself and her future. She did not exhibit any evidence for intellectual decline (score of 25 out of 30 on the Mini-Mental State Examination) or neurologic deficits.

The patient was maintained on treatment with sertraline and risperidone and had a working diagnosis of major depressive disorder with psychotic features, in partial remission. As part of the initial workup, blood tests were ordered, including thyroid-stimulating hormone (TSH), thyroid antibody (anti-TPO), vitamin B12, and folate. The serum vitamin B12 concentration was 294 pg/mL (reference range, 200–900 pg/mL). The serum folate concentration was 6.9 ng/mL (reference range, 3–20 ng/mL). Subsequent treatment included vitamin B12 injections and the patient's condition improved significantly with a decrease in her depression. The patient's TSH was within the reference range, and thyroid antibody levels were normal.

It is concluded that vitamin B12 deficiency may play a role in the development of mood disorder. Further studies are needed to determine the prevalence of vitamin B12 deficiency in the elderly and the impact of vitamin B12 therapy on mood disorder.
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Deficiency is beneficial therapeutically. Large-scale controlled studies and surveys of vitamin status among the elderly and as well as patients who are above age 50. It is, however, encouraging to know that replacement of vitamin B deficiency, especially among the elderly. It may also be prudent to consider this possibility in all patients with organic mental disorders, atypical psychiatric symptoms, poor response to conventional treatment, and fluctuating symptomatology. Assessment of B12 levels should be included as a standard evaluation with treatment-resistant depressive disorders, dementia, psychosis, or risk factors for malnutrition or among individuals with history of poor nourishment as well as patients who are above age 50. It is, however, encouraging to know that replacement of vitamin B12 in the presence of a deficiency is beneficial therapeutically. Large-scale controlled studies and surveys of vitamin status among the elderly and mentally ill may shed more light on this topic and may help improve the care of the mentally ill.
REFERENCES


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