

B12, Thiamine and the Nutritional Neurology of Psychiatric Symptoms

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Abstract

Vitamin B12 and thiamine deficiencies can produce cognitive, neurological and psychiatric manifestations. These disorders show that a nutritional deficiency can sometimes resemble or complicate primary psychiatric illness. They also demonstrate why nutritional medicine must remain diagnosis-specific: a broad catalogue of symptoms cannot establish a deficiency, and giving supplements without recognizing a serious neurological syndrome may be inadequate.

Two nutrients, distinct disease processes

Vitamin B12 is required for one-carbon metabolism and normal nervous-system function. Deficiency can cause macrocytic anemia, peripheral neuropathy, cognitive symptoms and neuropsychiatric presentations, sometimes without prominent anemia. Thiamine is essential to energy metabolism; severe deficiency can produce Wernicke encephalopathy, a potentially devastating disorder associated with confusion, ataxia and ocular motor abnormalities. These are not interchangeable “brain nutrition” problems, and each has a different clinical context and urgency.

Risk is about absorption as well as intake

Dietary restriction, pernicious anemia, intestinal disease and certain medications can contribute to B12 deficiency. Thiamine risk may arise with alcohol use disorder, malnutrition, prolonged vomiting, gastrointestinal surgery or serious systemic illness. A person may consume a nutrient yet fail to absorb or use it appropriately. This supports the Institute's recurring emphasis on transport and biological context, but it should not be stretched into the claim that every mood disorder is nutrient malabsorption.

Diagnostic nuance

Serum B12, complete blood counts, methylmalonic acid and related findings can be informative when interpreted appropriately; no single test is perfect. Wernicke encephalopathy is primarily a clinical emergency, and treatment should not be delayed in a highly suspicious presentation awaiting laboratory confirmation. Neurological examination and risk history often matter more than an indiscriminate micronutrient panel. Excessive doses of some supplements can also cause adverse effects, as illustrated by pyridoxine-related neuropathy.

Psychiatric outcomes

Correcting a true deficiency may improve mental status or cognitive function, yet neurological damage can persist after prolonged untreated disease. Patients can also have a genuine psychiatric disorder concurrent with nutrient deficiency. That dual possibility is more clinically useful than a false choice between psychology and biology.

Conclusion

Nutritional neuropsychiatry is strongest when it connects a defined deficiency to a compatible syndrome and an appropriate treatment. It provides a template for examining speculative dietary metabolites such as oxalate with greater rigor.

Selected readings

- [Neuropsychiatric manifestations in vitamin B12 deficiency](#)
- [Wernicke encephalopathy diagnostic challenges](#)