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Omeprazole and vitamin B12 deficiency.

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Abstract

The mainstay for cobalamin deficiency is correction of the underlying disorder and replacement therapy. Because the defect is often one of absorption, parenteral or intranasal routes are recommended. In most cases, replacement therapy is all that is needed. The vitamin preparation most commonly used is cyanocobalamin (also called vitamin B12), which has no known physiologic role but instead is converted to a biologically active form before it can be used by tissues. The studies reviewed in this article clearly show that omeprazole therapy will decrease the absorption of vitamin B12 by preventing its cleavage from dietary proteins. However, these data are insufficient to infer that clinically significant deficiency will occur over time. In fact, some of the studies suggest that the simple addition of juices or other acidic drinks into the diet may dramatically increase cobalamin absorption. Clearly, well-designed clinical trials are needed to evaluate this theory over an extended follow-up period to determine the clinical significance of omeprazole-associated vitamin B12 deficiency and possibly identify patients at risk for deficiency. In conclusion, the possibility of dietary vitamin B12 malabsorption should be considered in patients receiving chronic omeprazole treatment and presenting with signs and symptoms of deficiency. All healthcare workers should be made aware of the potential clinical complications of omeprazole-associated vitamin B12 deficiency since it may go unrecognized and is easily corrected. This is particularly relevant for elderly patients with poor dietary intake of vitamin B12, impaired vitamin B12 stores, and certain gastrointestinal disorders.