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Thiamine and Hashimoto's thyroiditis: a report of three cases.

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Abstract

OBJECTIVES: In a previous study on fatigue and related disorders in inflammatory bowel disease (IBD), we observed that IBD patients improved after treatment with high-dose thiamine. We hypothesized that the chronic fatigue accompanying inflammatory and autoimmune diseases is the clinical manifestation of a mild thiamine deficiency that is probably due to a dysfunction of the intracellular transport or to enzymatic abnormalities. Hashimoto's thyroiditis is both a common automimmune disease and cause of hypothyroidism. Although levothyroxine, a thyroid hormone, is the treatment of choice for hypothyroidism, a significant number of patients on thyroid hormone replacement therapy report not feeling well despite having thyroid function tests within the healthy range. Based on our hypothesis, we started treating the fatigue in patients affected by Hashimoto's thyroiditis and taking a thyroid hormone with thiamine. This is a report of the outcomes of three cases in which the fatigue component reported by patients with Hashimoto's thyroiditis was treated with thiamine.

DESIGN: Three patients on thyroid hormone replacement because of Hashimoto's thyroiditis were treated for the fatigue component of the disease from May to July 2011. Fatigue was measured using the Fatigue Severity Scale. Free thiamine in the serum and thiamine pyrophosphate in red cells were tested before and after the therapy. All three patients received oral (600 mg/day) or parenteral (100 mg/ml every four days) doses of thiamine.

RESULTS: Treatment with thiamine led to partial or complete regression of the fatigue within a few hours or days.

CONCLUSION: As the administration of thiamine led to a partial or complete regression of the fatigue and related disorders, it is reasonable to infer that the administration of large quantities of thiamine restores thiamine-dependent processes. The mild thiamine deficiency suggested by fatigue and related disorders may be due a dysfunction of the intracellular transport of thiamine or to enzymatic abnormalities most likely related to the autoimmune process of the disease.

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