Thiamine Deficiency

Gregory L. Brotzman, M.D.

With the increasing number of Southeast Asian as well as other refugees migrating to the United States, dietary-related thiamine deficiency (beriberi) could be more common than one would think.[1-4] Information pertaining to vitamin deficiency syndromes is often scattered throughout many reference sources and might not be readily available to the family physician. This report presents a case of presumed thiamine deficiency. The approach used to diagnose the illness was less than optimal. The discussion that follows describes briefly the pathophysiology of thiamine deficiency and the recommended approach for diagnosis and treatment.

Case Report

A 66-year-old female, non-English–speaking Laotian refugee came to the office for the first time with a 12-year history of paresthesia that began in the legs and gradually began to involve the arms as well. Her symptoms had worsened during the past 3 weeks. She also complained of mild, intermittent dizziness, fatigue, intermittent nausea and vomiting, occasional diarrhea, and generalized pains in her arms and legs. She denied any fever, weight changes, or syncope. Her medical history was important only for an asymptomatic goiter. The patient had moved from a Laotian refugee camp to the United States 12 years ago. She was under some stress at home with a son suffering from depression. Her usual diet consisted mainly of polished rice, meats, and some vegetables. On physical examination the patient was alert and quiet and had a rather flat affect. A neck examination revealed a large, smooth, nontender, asymmetric thyroid. Heart auscultation disclosed a grade 1/6 systolic murmur at the right upper and left lower sternal borders. There was no edema, atrophy, or tenderness in her extremities, and her muscle strength was 5+ and 5+ in arms and legs, respectively. Deep tendon reflexes were symmetric. Light touch sensation, vibration, and position sense were intact throughout. Findings of a cranial nerve examination were normal. Romberg station findings and gait were normal. Thyroid function studies, complete blood count, VDRL tests, and results of an automated blood chemistry analysis (Chem 20) were all within normal limits, except for the following components: potassium 3.4 mmol/L, normal 3.5–5.0 mmol/L (3.4 mEq/L [3.5–5.0 mEq/L]), lactate dehydrogenase (LDH) 3.03 μkat/L, normal 1.08–2.83 μkat/L (182 U/L [65–170 IU/L]), and chloride 111 mmol/L, normal 98–106 mmol/L (111 mEq/L [98–106 mEq/L]). Serum thiamine status was 0.71 IU/g hemoglobin (0.75–1.30 IU/g hemoglobin). No conversion to le Système International d'Unités (SI units) was available for this result. Electrocardiogram findings were normal. A chest radiograph showed borderline cardiomegaly. An echocardiogram was normal. Findings on a thyroid sonogram suggested a multinodular goiter.

The patient was given dietary counseling to improve the content of thiamine in her food as well as to assure adequate overall nutrition. Oral thiamine supplementation was prescribed, 12.5 mg three times a day. The patient was also told to take a multivitamin daily. There was some delay in starting the medications as a result of language difficulties, but within 2 weeks of therapy the patient's subjective complaints of paresthesia and pain had resolved. Two weeks after stopping the thiamine supplementation, a repeat thiamine level (using a different laboratory) was 30.6 nmol/L (3.8–24.1 nmol/L). Repeat electrolytes and LDH were normal. The chest radiograph was unchanged. The multivitamin was continued. The patient had gradual resolution of her fatigue over the ensuing weeks and her paresthesia did not recur.

Discussion

In the United States alcoholism is the major cause of thiamine deficiency. This deficiency is related to overall poor nutrition, a decreased ability for

Submitted, revised, 22 May 1991.

From the Department of Family Medicine, Medical College of Wisconsin, Milwaukee. Address reprint requests to Gregory L. Brotzman, M.D., Columbia Family Care Center, 210 West Capitol Drive, Milwaukee, WI 53212.

J Am Board Fam Pract: first published as 10.3122/jabfm.5.3.323 on 1 May 1992. Downloaded from http://www.jabfm.org/ on 16 November 2026 by guest. Protected by copyright.

the damaged liver to store thiamine, and a decrease in thiamine absorption resulting from chronic alcoholic gastritis.[5] Critically ill patients have increased requirements for thiamine, especially those requiring parenteral nutrition.[6] Thiamine deficiency can manifest as peripheral neuropathy (dry beriberi), Wernicke-Korsakoff syndrome, lactic acidosis, and high-output cardiac failure (wet beriberi). In developing countries thiamine deficiency is related mainly to the consumption of cooked, highly milled, thiamine-depleted rice. In general, it takes about 3 months of a diet that is grossly deficient in thiamine to develop a neuropathy.[5] Certain foods such as raw fish, fermented tea leaves, coffee, tea, and some vegetables contain thiaminases, which can inactivate whatever thiamine is present in an already compromised diet.[2,5,7,8]

Thiamine deficiency is often referred to as beriberi, which means "I cannot."[3] Beriberi can vary widely in its presentation. The dry form of beriberi usually has symmetric sensory, motor, or reflex abnormalities of the lower limbs initially, followed by the arms and, at times, even the cranial nerves. One of the senses can be more affected than another. Symptoms begin distally and progress in an ascending fashion. Peripheral nerves show axonal as well as myelin sheath damage, especially distally. There can be slight edema of the inner tibial areas. Patients complain of burning or pain in the glove-stocking distribution and weakness, particularly of the feet and ankles; they can have calf tenderness, diminished or absent deep tendon reflexes, vomiting, constipation, irritability, depression, and palpitations.[1,4,5,9]

Wernicke-Korsakoff syndrome consists of acute onset of ataxia, ophthalmoplegia, and confusion with anterograde amnesia. This syndrome can be brought on in a patient with a subclinical deficiency state if he or she is given intravenous glucose, because thiamine is required for glucose metabolism.[7,8]

Thiamine-deficient alcoholics can have an acute onset of severe lactic acidosis, which is readily reversed with thiamine therapy.[10] Cardiac, or wet, beriberi presents as congestive heart failure, and in its fulminant form is known as Shoshin disease.[9,11] Because of the abrupt onset of Shoshin beriberi, edema might not be present.

Infantile beriberi is the result of thiamine deficiency in mothers of breast-fed infants. It usually occurs in infants under 6 months of age and presents as an abrupt onset of listlessness, aphonia, vomiting, anorexia, constipation, and at times congestive heart failure.[7]

Diagnosis

Thiamine deficiency can be difficult to diagnose, because the symptomatology can be vague, especially with mild deficiency. Measurement of serum thiamine alone is difficult to interpret, because levels can fluctuate.[12] Diagnosis is usually based upon thiamine activity as a coenzyme in its phosphorylated form, thiamine diphosphate (TDP).[13] TDP is a coenzyme for oxidative decarboxylation of α-keto acids as well as for the transketolase reaction in the pentose phosphate pathway.[1,5,8] Transketolase activity is decreased before the onset of symptoms by as much as 30 to 50 percent of normal levels.[5] If transketolase activity is measured after the addition of TDP, which is the physiologically active form of thiamine, the effective increase in activity is a useful measure of the degree of thiamine deficiency.[12,13] A TDP effect of 0 to 15 percent is normal, 15 to 24 percent indicates marginal deficiency, and 25 percent or more indicates severe deficiency.[1,7] In chronic deficiency states, transketolase activity can be decreased as a result of decreased apoenzyme synthesis. This decrease might not normalize even after adequate supplementation.[7,13]

A more accurate means of evaluating thiamine status is by direct measurement of erythrocyte thiamine diphosphate (ETDP). This is the primarily active form of thiamine and is not affected by factors that interfere with transketolase activity levels.[13]

Treatment

Treatment of thiamine deficiency is aggressive parenteral therapy with 50 to 200 mg daily (or more) if cardiac failure or Wernicke-Korsakoff syndrome is present.[1,5,6,10,14] Adverse effects such as allergic reactions can occur, and respiratory depression has been reported with large parenteral doses.[7] Oral therapy with 5 to 25 mg of thiamine three times a day should be prescribed after the acutely ill patient has recovered (which is usually very rapidly) and for the neuropathic form of beriberi. Greater quantities are generally excreted

J Am Board Fam Pract: first published as 10.3122/jabfm.5.3.323 on 1 May 1992. Downloaded from http://www.jabfm.org/ on 16 November 2026 by guest. Protected by copyright.

by the kidneys.[1,5] If patients are not able to tolerate oral therapy, thiamine can be given intramuscularly 10 to 25 mg daily.[5,8] A rapid clinical response to thiamine supplementation, particularly in patients with cardiac or central nervous system involvement, is highly suggestive of a diagnosis of thiamine deficiency.[14] Carbamazepine has been reported to be helpful in patients with chronic neuropathic pain.[15]

With all forms of deficiency, therapy includes proper nutritional counseling. Body stores of thiamine are minimal, and it is therefore essential to ensure proper dietary intake. Recommended daily allowances of thiamine are 0.5 mg/4200 J (0.5 mg/1000 cal) of intake, or usually 1.3 to 1.5 mg/d for adults.[8] This daily intake should be increased by 0.3 to 0.5 mg for pregnant or breast-feeding women.[1,2,8] Foods rich in thiamine consist of whole or enriched grains and cereals, organ meats, pork, beef, yeast, nuts, and vegetables (especially green beans and peas).[3,5] Many patients are deficient in vitamins other than thiamine, so it is prudent to supplement them with a multivitamin as well.

Prognosis

Cardiac changes usually respond dramatically, whereas the neuropathies are more resistant to therapy.[11] With treatment, deep tendon reflexes should return, muscle weakness and soreness should abate, and paresthesia should resolve. These changes can be seen within a few weeks of therapy or can take months to resolve.[5] Approximately 5 percent of patients will have irreversible neuropathy.[7,16]

Conclusion

Thiamine deficiency can present in several forms. It can be more prevalent than expected, especially in refugee immigrants, critically ill patients, and alcoholics. Thiamine deficiency can manifest itself as cardiac failure, neuropathy, or Wernicke-Korsakoff syndrome. Transketolase activity, TDP effect, and ETDP levels are useful in diagnosing a deficiency state. Treatment consists of parenteral or oral supplementation and proper nutrition.

References

1. Skelton WP 3d, Skelton NK. Thiamine deficiency neuropathy. It's still common today. Postgrad Med 1989; 85:301-6.
2. McCormick DB. Thiamine. In: Shils ME, Young VR, editors. Modern nutrition in health and disease. 7th ed. Philadelphia: Lea and Febiger, 1988: 355-61.
3. Burtis G, Davis J, Martin S, editors. Applied nutrition and diet therapy. Philadelphia: W.B. Saunders, 1988:177-81.
4. Victor M. Nutritional disorders of the nervous system. In: Spittell, JA Jr, editor-in-chief. Clinical medicine. Vol II. Neurology. Philadelphia: Harper & Row, 1986; 11:12-43.
5. Farmer TW. Neurologic complications of vitamin and mineral disorders. In: Joynt RJ, editor. Clinical neurology. Philadelphia: JB Lippincott, 1988:1, 4-7,83.
6. Cruickshank AM, Telfer ABM, Shenkin A. Thiamine deficiency in the critically ill. Intensive Care Med 1988; 14:384-7.
7. Haas RH. Thiamine and the brain. Annu Rev Nutr 1988; 8:483-515.
8. Recommended dietary allowances. Subcommittee on the Tenth Edition of the RDAs, Food and Nutrition Board, Commission on Life Sciences, National Research Council. Washington, DC: National Academy Press, 1989:125-32.
9. Platt BS. Beriberi II: clinical features of endemic beriberi. Federation Proc 1958; 17(Suppl):8-20.
10. Campbell CH. The severe lacticacidosis of thiamine deficiency: acute pernicious or fulminating beriberi. Lancet 1984; 2:446-9.
11. Meulders Q, Laterre PF, Sergant M, Corbeel L. Shoshin beriberi: a fulminant beriberi heart disease. Acta Clin Belg 1988; 43:115-9.
12. Kuriyama M, Yokomine R, Arima H, Hamada R, Igata A. Blood vitamin B_1, transketolase and thiamine pyrophosphate (TPP) effect in beriberi patients, with studies employing discriminant analysis. Clin Chim Acta 1980; 108:159-68.
13. Baines M, Davies G. The evaluation of erythrocyte thiamine diphosphate as an indicator of thiamine status in man, and its comparison with erythrocyte transketolase activity measurements. Ann Clin Biochem 1988; 25:698-705.
14. Baron RB. Nutrition. In: Krupp MA, Schroeder SA, Tierney LM Jr, editors. Current medical diagnosis and treatment. Norwalk, CT: Appleton & Lange, 1987:804.
15. Skelton WP 3d. Neuropathic beriberi and carbamazepine. Ann Intern Med 1988; 109:598-9.
16. Gill GV, Bell DR. Persisting nutritional neuropathy amongst former war prisoners. J Neurol Neurosurg Psychiatry 1982; 45:861-5.

J Am Board Fam Pract: first published as 10.3122/jabfm.5.3.323 on 1 May 1992. Downloaded from http://www.jabfm.org/ on 16 November 2026 by guest. Protected by copyright.