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Original article

Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery

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Abstract

Background: Nutritional deficiencies are a recognized complication of bariatric surgery. Thiamine deficiency has been reported as a possible consequence of both restrictive and malabsorptive bariatric procedures. Most of the reported cases occurred after Roux-en-Y gastric bypass (RYGB) surgery; fewer were described after biliopancreatic diversion, vertical banded gastroplasty, or duodenal switch. Adults who have a high carbohydrate intake derived mainly from refined sugars and milled rice are at greater risk of developing thiamine deficiency, because thiamine is absent from fats, oils, and refined sugars. Currently, no reports have evaluated the preoperative thiamine status of bariatric patients. The aim of this study was to evaluate the degree of thiamine deficiency in obese patients before bariatric surgery at our institution.

Methods: The medical records of consecutive patients who underwent laparoscopic RYGB or laparoscopic adjustable gastric banding at our institution between March 2003 and February 2004 were retrospectively reviewed. Patients were selected for this study on the basis of predetermined criteria. Preoperative thiamine levels were retrospectively recorded. Excluded from this study were patients who had been taking multivitamins or other nutritional supplements before surgical intervention, had a history of frequent alcohol consumption, any malabsorptive diseases, or previous restrictive-malabsorptive surgical interventions, such as RYGB, biliopancreatic diversion, or adjustable gastric banding, according to the initial evaluation and questionnaire.

Results: Of 437 consecutive patients who underwent laparoscopic RYGB or laparoscopic adjustable gastric banding, 303 were included in the study. Forty-seven patients (15.5%) presented with low preoperative thiamine levels. The mean age and body mass index of these patients was 46 years and 60 kg/m^2, respectively. Male patients presented with greater mean preoperative thiamine levels (3.2 μg/dL) than female patients (2.4 μg/dL).

Conclusion: Obese patients undergoing bariatric surgery may have significant thiamine deficiency before surgery. © 2005 American Society for Bariatric Surgery. All rights reserved.

Keywords: Thiamine deficiency; Obesity; Bariatric surgery

Thiamine is one of eight water-soluble vitamins and is the first B vitamin to be discovered; thus, it is also named vitamin B_1. Thiamine is a coenzyme involved in a wide variety of intricate biochemical pathways necessary for proper tissue and organ function, including decarboxylation of pyruvate and oxidation of alpha-keto-glutamic acid, and functions as the link between the glycolytic and citric acid cycles. Deficiency of this essential vitamin leads to the accumulation of lactate and pyruvate and to decrease levels of alpha-keto-glutarate, acetate, citrate, and acetylcholine. If not detected in time, alterations in the levels of any of these compounds may lead to serious metabolic derangements, causing cardiovascular and neurologic manifestations [1].

Thiamine, along with iron, calcium, and vitamin A, is absorbed predominantly in the duodenum. The average total

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thiamine storage in a healthy adult is approximately 0.11 mmol (30 mg). The greatest concentrations are found in skeletal muscles and liver, heart, kidneys, and brain. It is important to emphasize that thiamine is not stored in large amounts in any tissue and may become depleted within a few weeks [1].

Thiamine is found in limited quantities in a variety of animal and vegetable products and is abundant only in a small number of foods. It is often destroyed in the process of food preparation. Food sources for thiamine are yeast, whole grain, beef, lean pork, and legumes. This vitamin is absent from fats, oils, and refined sugars, and it is rapidly destroyed in an alkaline environment with a pH >8, high temperature, and when ingested with ethanol. Tea, coffee (caffeinated and decaffeinated), raw fish, and shellfish contain thiaminase, which is able to destroy the vitamin during food storage or preparation or during passage in the gastrointestinal tract.

Adult populations considered vulnerable to the development of thiamine deficiency are those who have high carbohydrate intake derived mainly from milled rice and refined sugar and chronic ethanol abusers. An increase in the vitamin requirements caused by strenuous physical activity, fever, pregnancy, lactation, and adolescent growth may hasten clinical manifestations of deficiency. Hypermetabolic states, surgery, dialysis, and congestive heart failure treated with furosemide increase thiamine requirements and induce thiamine loss in the urine [1–3].

The recommended dietary allowance of thiamine for healthy adults is between 1.1 and 1.5 mg daily, depending on the gender, weight, and height of the individual [2]. In addition, a balanced adult diet should contain approximately 0.27 mg of thiamine per 1000 kcal of ingested food. Slightly greater levels are required during pregnancy and lactation, illness or stress, and at surgery [3].

Thiamine has been extensively studied since the late 1940s [4–6]. Its well-known biologic role as a coenzyme in carbohydrate metabolism and nerve conduction has recently been complemented by new findings regarding its molecular, genetic, and transporter properties [7,8]. Thus, thiamine deficiency may lead to severely debilitating cardiovascular and neurologic derangements, such as wet or dry beriberi and central nervous system manifestations such as Wernicke's encephalopathy and/or Wernicke-Korsakoff syndrome.

Clinically, dry beriberi manifests predominantly by peripheral neuropathy; it may include calf muscle tenderness and difficulty in rising from a squatting position. In wet beriberi, in addition to peripheral neuropathy, some of the common features include edema, tachycardia, and congestive heart failure. Involvement of the central nerves system is termed Wernicke's encephalopathy and is characterized by confusion, nystagmus, ophthalmoplegia, and ataxia. The advanced stages of this disease include memory loss and confabulation, leading to Korsakoff's psychosis and permanent neurologic manifestations. The combination of these two entities is referred to as the Wernicke-Korsakoff syndrome. Although ethanol consumption is a major cause of thiamine deficiency in Wernicke-Korsakoff syndrome, iatrogenic causes, including parenteral glucose administration and chronic dialysis, can worsen the syndrome in patients with marginal thiamine status.

Malnutrition can be defined as a condition that develops when the human body does not receive adequate quantities of energy-providing nutrients (protein, fat, and carbohydrates), vitamins, minerals, and water, which are required for maintaining normal healthy tissue and organ function. This condition is, for most part, a gradual process that generally spans a period of years and tends to develop in stages. Malnutrition can result from various causes, including starvation, an unbalanced diet, stress from illness or surgery, and various metabolic disorders. Subclinical vitamin and mineral deficiencies are not uncommon among the normal population.

Morbid obesity is among the most intractable health problems in the United States. The combination of a sedentary lifestyle, a high-fat, high-caloric Western-style diet, increased portion sizes, and low socioeconomic status have been named as major culprits of this epidemic [9–12]. A recent report by the Centers for Disease Control and Prevention stated that obesity, defined as a body mass index $\geq$30 kg/m^2, had risen considerably in the adult population in the United States between 1991 (12%) and 1998 (17.9%) [13]. During the past decade, the overall prevalence of this disease has increased from 25% to 33%, doubled between the ages of 20 and 55, and found to be greater among women than men. In addition, the prevalence of morbid obesity and super-morbid obesity in the general population has escalated to 30.5% and 4.7%, respectively during this period [14].

According to the National Research Council, >80% of Americans consume a diet that is below the recommended daily allowance for vitamins and minerals [15]. In the morbidly obese population, deficiencies in thiamine and other essential vitamins, and the development of irreversible neurologic sequelae, have been reported after weight-reduction surgery such as Roux-en-Y gastric bypass (RYGB), adjustable gastric banding (AGB), biliopancreatic diversion, and duodenal switch [16–20]. These deficiencies develop more rapidly and become more pronounced when protracted vomiting is added to the clinical scenario. Nausea and vomiting are common among symptomatic patients after gastric bypass surgery and generally become evident 1 week to 4 months after the weight reduction procedure [21–25]. Regardless of the weight reduction surgery performed, the reduction in nutrients transported to the portal circulation is always significant, placing the patient at risk of malnutrition. In patients with occult preoperative thiamine deficiency, the operative stress, nature of the weight reduction procedure, and subsequent weight loss may aggravate the deficiency and lead to clinical manifestations [26].

Numerous publications have discussed the nutritional

complications caused by gastrointestinal surgical procedures, but reports discussing the nutritional state of vitamin and trace mineral deficiencies in the obese population before surgery are sparse. The amount of food consumed by obese patients is often overwhelming, but the nutritious value of that food is usually poor.

The obese population is often erroneously believed to be "well-nourished," but their dietary habits, consumption of large amounts of carbohydrates and refined sugars, ethanol consumption, eating disorders with purging, use of diuretics (to treat comorbidities or for weight loss) have led us to assume that vitamin and trace-element deficiencies exist in these patients before surgical intervention.

The aim of this report was to evaluate the degree of preoperative thiamine deficiency in obese patients who underwent either laparoscopic RYGB or AGB at our institution as treatment for morbid obesity.

Methods

After institutional review board approval, all medical records of consecutive patients who underwent laparoscopic RYGB or AGB at our institution between March 2003 and February 2004 were retrospectively reviewed.

In our department, 2 weeks before surgery, the patient undergoes an evaluation that includes measurement of the blood level of thiamine (in micrograms per deciliter). We retrospectively reviewed those results from the patients' medical records. Patients were excluded from this study if the information documented in the preoperative evaluation sheet indicated ≥ 1 of the following parameters: ingestion of fortified foods, such as protein supplements, energy/sports drinks, or sports bars; consumption of multivitamins or nutritional supplements before surgical intervention; frequent alcohol intake; or a positive history of malabsorptive diseases or previous restrictive-malabsorptive procedures.

Results

Of 437 patients, 303 (69%) were included in this study; 201 were women (66.3%) and 102 were men (33.7%), who underwent either laparoscopic RYGB or laparoscopic AGB. The patient age range was 18–74 years (mean 46), and the body mass index range was 31–89 kg/m^2 (mean 60; Table 1). The blood levels of thiamine 2 weeks before surgery indicated that 47 patients (15.5%) had low preoperative thiamine levels (normal range 3.8–12.2 μg/dL), with values of 1.2–3.6 μg/dL (Table 2). Of these, 34 (72.3%) were women and 13 (27.6%) were men (Table 3). Of the patients with low levels of thiamine, the male patients presented with relatively greater preoperative thiamine levels (mean 3.2 μg/dL) compared with the female patients (mean 2.4 μg/dL). Despite the very low thiamine levels detected in some of these patients, neurologic symptoms related to thiamine deficiency were not documented.

Table 1
Patient demographics

Patients	303
Age (yr)	
Mean	46
Range	18–74
Gender (%)	
Women	201 (66.3)
Men	102 (33.7)
BMI (kg/m^2)	
Mean	60
Range	31–89

BMI = body mass index.

Discussion

One common assumption within the general medical practice is that normal, healthy individuals meet their nutritional needs through diet alone. This assumption is even more misleading in obese patients. Reports have indicated that most people do not consume adequate amounts of vitamins by diet alone, and may, in fact, benefit from regular nutritional supplements [27,28].

Because of their long-standing poorly controlled dietary habits, associated comorbidities, and medical problems, such as hypothyroidism and liver disease, and the use of diuretics, weight-reduction diets, or starvation, morbidly obese individuals may present with superimposed nutritional deficiencies. In addition, the inadequate dietary habits of this group of patients often leads to a decreased intake of essential nutrients, including essential vitamins [29]. Nutritional status is an important issue in all patients undergoing surgery, but is particularly important in morbidly obese individuals who undergo restrictive and/or malabsorptive procedures in an effort to reduce their excess weight and improve the associated comorbid conditions. As demonstrated by several published reports, weight loss surgery may lead to nutritional deficiencies, even in purely restrictive procedures such as AGB, which does not entail rearrangement of the gastrointestinal tract anatomy [30–32]. Others have suggested that postoperative nutritional deficiencies are proportional to the length of absorptive area and to the percentage of weight loss [33].

In 1984, Villar and Ranne [34] presented a series of 250 patients who underwent gastric partitioning for the control of morbid obesity and found that 15 (6%) had low preoperative thiamine levels. In addition, they found a dramatic decline in thiamine levels 6 weeks after this type of weight-reduction surgery and warned of severe permanent neurologic sequelae possibly due to thiamine deficiency in this patient population. Two decades later, our current study has demonstrated greater rates of low thiamine levels among morbidly obese patients before weight-reduction surgery. In addition, our results have indicated that 7.9% (24 of 303) of patients presented with greater than normal preoperative thiamine levels, despite having not taken multivitamins. These findings could be a result of the peculiar dietary

Table 2
Demographics in low-thiamine level population

Gender	Age (years)		BMI (Kg/m^2)		Weight (Lb)		Thiamine levels (μg/dl)	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Women	41.5	24–59	36.5	36–67	340	210–470	2.4	1.2–3.6
Men	51.5	33–70	52.5	34–71	408	265–551	3.2	2.8–3.6

BMI = body mass index.

habits among this subset of patients, a greater number of thiamine transporters in their circulation, or to larger thiamine stores despite low circulating levels. Although their study was based on a small group of subjects, Patrini et al. [35] suggested that obese women, in particular, tend to maintain greater levels of thiamine and stated that this may be a result of a preferential intracellular recycling process that leads to greater thiamine storage. This may offer some explanation of the greater-than-normal preoperative thiamine levels observed in 7.9% of our reported patient population. Of this subgroup, 17 (71%) of the 24 patients were women.

Because of the intestinal anatomic divergence, absorptive alterations, starvation, anorexia, recurrent vomiting, and/or prolonged parenteral feeding resulting from bariatric procedures, morbidly obese patients are normally administered additional daily nutritional supplements, including vitamin B complex [36]. MacLean et al. [23] collected data on 96 patients after weight reduction operations; 17% of the patients who had rapid weight loss had low levels of vitamin B_{12}, thiamine, and serum and red blood cell folate levels. Only 1 patient with markedly decreased serum thiamine had obvious neuropathy. Low thiamine and serum folate levels were also seen in patients who postoperatively were administered a liquid diet of 750 kcal with multivitamin supplements. The authors defined malnutrition as a total exchangeable sodium (Na^+e) to total exchangeable potassium (K^+e) ratio of >1.22. By this definition of malnutrition, none of the patients mentioned above would have been classified as malnourished. Thus, despite the additional nutritional guidance and the supply of multivitamins given to morbidly obese patients after RYGB or AGB placement, a small number will develop clinical manifestations of vitamin deficiency, most notably of thiamine [37–40].

We believe that the postoperative course is influenced by the nutritional status of the patient before the surgical intervention. To avoid postoperative complications as much as possible, we monitor blood levels of thiamine, folic acid, calcium, iron, vitamin B_{12}, and lipids at 2 weeks preoperatively, 2, 6, and 12 months postoperatively, and yearly thereafter. We determined these intervals because the body stores of vitamins and minerals vary tremendously. Some vitamin stores, such as for vitamin B_{12}, are large, and others, such as for thiamine and folate, may become depleted within weeks of an insufficient diet. When combined with the postoperative stress, diet regimen, and patient compliance to supplement therapy, thiamine might be depleted within <2 weeks [1]. In our opinion, all bariatric patients should have thiamine levels monitored before bariatric surgery as a part of a comprehensive nutritional assessment to prevent possible complications due to deficiency.

Table 3
Patient distribution of thiamine level

Thiamine level	Gender	
	Women (%)	Men (%)
Low (<3.8 μg/dl)	34 (72.3)	13 (27.6)
Normal (3.8–12.2 μg/dl)	150 (64.6)	82 (35.3)
High (>12.2 μg/dl)	17 (71)	7 (29)

In our practice, patients who present with low thiamine levels before surgery are given 100 mg of thiamine orally twice daily for 1 month or until their thiamine levels achieve adequate levels. During the postoperative follow-up, in patients with marginal low serum thiamine levels and prolonged nausea and vomiting, we advocate intravenous thiamine treatment at a dose of 100 mg every 6 hours for 2 days, followed by 100 mg of thiamine orally twice daily for 1 month, in addition to oral multivitamin supplements containing other B vitamins.

The rationale behind the administration of large doses of thiamine is to replenish thiamine stores, which correlate with the concentration of thiamine in the serum and the cerebrospinal fluid, stimulate enzymatic-dependent reactions, and improve cardiovascular disorders. The latter is especially significant in obese patients, who have a predisposition to cardiovascular comorbidities. The parental route is desirable initially to ensure maximal bioavailability of thiamine [1].

Loh et al. [41] described a patient who presented with acute Wernicke's encephalopathy 2 months after laparoscopic bariatric surgery. The patient was initially misdiagnosed with acute stroke, and, because of intractable vomiting, he received treatment with parenteral antiemetics along with intravenous fluid-dextrose resuscitation, which caused an exacerbation of the clinical symptoms. The patient was then diagnosed with Wernicke's encephalopathy and was treated with intravenous thiamine. In this case, postoperative oral vitamin supplementation did not prevent this complication. The patient had permanent neurologic sequelae. The authors concluded that even with early recognition and aggressive therapy, acute Wernicke's encephalopathy com-

monly results in permanent disability. They recommended prevention by administration of parenteral thiamine 50–100 mg beginning at 6 weeks postoperatively in patients with protracted vomiting as a preventative measure. In patients presenting with neurologic symptoms, although the differential diagnosis of stroke, hypoglycemia, ethanol intoxication, and/or a past medical history may indicate possible thiamine deficiency, it is of great importance to begin the intravenous thiamine therapy before the intravenous infusion of glucose solutions. As cited above, glucose infusion may precipitate this disease in previously asymptomatic patients or cause rapid deterioration of the disease.

The prevalence of thiamine deficiency among morbidly obese patients undergoing bariatric procedures has not yet been clearly reported. Our results have indicated that the overall prevalence of thiamine deficiency in the morbidly obese patient population is great (15.5%). Despite years of research and numerous published reports, few have placed emphasis on the preoperative thiamine levels as a key predisposing factor to postoperative neurologic sequelae. In our reported series, none of the patients who had low thiamine levels before undergoing bariatric surgery developed symptomatic neurologic manifestations. We attribute that to our preoperative protocol of oral thiamine and/or to favorable changes in dietary habits initiated by patients after surgery. These dietary changes may have consequently replenished any postoperative thiamine deficiencies. Other reasons may include the absolute cessation of ethanol consumption and improved folate and protein nutritional status with the addition of regular vitamin supplements [1,42–45].

However, the reason the prevalence of thiamine deficiency in our series was as great as 15.5% is not clear. Possible contributing factors for thiamine deficiency, such as the patients' socioeconomic status, chronic medication ingestion [46], or detailed dietary and nutritional histories, were not recorded in this study, and their absence is one of its methodologic flaws.

Although we did not use a more specific and sophisticated thiamine detection method, such as red blood cell transketolase activity, we were still able to determine the degree of preoperative thiamine levels in our study population. These promising early findings in this pilot study warrant additional investigation. As a result, we are currently in the process of retrospectively analyzing the postoperative thiamine levels in a group of >1400 morbidly obese patients who have undergone bariatric surgery in our institution and correlating these with the preoperative thiamine levels.

Conclusion

Obese patients undergoing bariatric surgery may have significant thiamine deficiency before surgery. All morbidly obese patients who are scheduled to undergo bariatric surgery undergo a nutritional status evaluation and are administered adequate nutritional supplementation before surgery to prevent the possible development of postoperative deficiencies. This is especially true for thiamine. It is imperative that treating physicians understand that any bariatric procedure carries the risk of nutritional deficiency; therefore, these patients should be closely monitored to detect cardiovascular and neurologic manifestations, and adequate therapy should be administered to prevent irreversible outcomes.

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Editorial Comment

I would like to commend Carrodeguas et al. [1] for publishing the first article dealing with the *preoperative* quantification of thiamine deficiency in the bariatric surgery population. Postoperative thiamine deficiency has been well described, initially after restrictive procedures by MacLean et al. [2] and later after gastric bypass operations by the late John Halverson [3]. In addition, the need for bariatric surgeons to realize its significance, as illustrated by Mason [4], must not go unnoticed. This complex nutritional derangement, with its radiographic [5] and clinicopathologic sequellae [6], has received a tremendous amount of press in the last 24 months. However, to date, little instruction has been given to bariatric surgeons on its preoperative and postoperative management.

In this highly litigious arena, Carrodeguas et al. provide us with the armamentarium to effectively treat postoperative thiamine deficiency and offer an approach for further monitoring. With a documented frequency of 15.5% before bariatric surgery, the surgeon's exposure to this complication is inevitable. Clearly, individuals with persistent postoperative emesis after purely restrictive procedures and combined restrictive/malabsorptive procedures are at a heightened risk for thiamine deficiency. Perhaps now, even those individuals with low preoperative thiamine levels can be spared the disastrous, yet preventable, complications of Wernicke's encephalopathy. Ongoing study of this phenomenon is required, and great credit is given to Carrodeguas et al. for yet again reinforcing the implications of our procedures.

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