

Thiamine Deficiency after Bariatric Surgery may lead to Wernicke Encephalopathy

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Bariatric surgery is gradually becoming the preferred solution for the treatment of morbid obesity. The volume of bariatric surgery suggests that any physician involved in clinical medicine will encounter patients who have been treated with obesity surgery. Practitioners will therefore need to familiarize themselves with the potential adverse effects associated with this therapy.

The complications of bariatric surgery are not insignificant and are, in part, related to the problems inherent in operating on obese patients. Reoperation may be necessary if bleeding, leak, abscess, stenosis or ventral hernia occurs. It is important to note that a variety of late complications are recognized as well, usually the consequence of nutritional deficiency. Nutrient deficiencies are common after bariatric surgery. Although these may be considered an inevitable consequence of bariatric procedures, in the majority of cases they are avoidable. Careful monitoring of certain nutrient parameters, most notably iron, vitamin B12, vitamin B1, folic acid and vitamin D, as well as calcium metabolism and bone density status, is necessary to prevent future problems.

NEUROLOGICAL COMPLICATIONS FOLLOWING BARIATRIC SURGERY

Of special note are neurological complications, which manifest at 3 to 20 months after bariatric surgery and all affect patients experiencing prolonged vomiting. The neurological symptoms include chronic and subacute peripheral neuropathy, acute peripheral neuropathy, burning feet, meralgia paresthetica, myotonic syndrome, posterolateral myelopathy, and Wernicke encephalopathy [1].

Since the number of bariatric surgeries is dramatically expanding and the attention to neurological symptoms increasing, more cases of WE are reported, though their percentage in the post-surgery population is decreasing. For example, in the United States 8 cases were reported out of 100,000 surgeries in 2002 and 9 cases were published in 2006–2007 among approximately 400,000 surgeries. In southern Europe a combined total of 6 WE cases among 3241 patients undergoing biliopancreatic diversion were reported.

A systematic review of the literature [2] was published in 2008. The report [2] describes 84 cases of WE between 1991 and 2008. Gastric bypass or a restrictive procedure had been performed in 80 cases (95%). Admission to hospital for WE occurred within 6 months of surgery in 79 cases (94%). Frequent vomiting was a risk factor in 76 cases (90%) and had lasted for a median of 21 days before admission. Intravenous glucose administration without thiamine was a risk factor in 15 cases (18%). Brain magnetic resonance imaging identified lesions characteristic of WE in 14 of 30 cases (47%).

WE = Wernicke encephalopathy

Incomplete recovery was observed in 41 cases (49%).

METABOLIC DEFICIENCY LEADING TO WE

Wernicke encephalopathy results from a deficiency of vitamin B1 [3] and is classically characterized by specific symptoms: confusional state, disorientation, ophthalmoplegia, nystagmus, diplopia, and ataxia [4,5]. Neuro-radiological findings (MRI T2-weighted and FLAIR imaging) usually show symmetric signal intensity alterations in the mamillary bodies, medial thalamic, tectal plate, and periaqueductal area [6].

WE was traditionally associated with chronic alcohol abuse but can be caused by many other pathological conditions such as tumors of the gastrointestinal tract, bariatric surgery for obesity [7], psychogenic refusal of food, hyperemesis gravidarum, anorexia nervosa, prolonged infectious-febrile disease, voluntary food starvation, chronic uremia, and parenteral therapy. In this issue of *IMAJ*, Sharabi and Bisharat [7] describe a case of a 34 year old bariatric patient who developed Wernicke-Korsakoff syndrome that led to her death. This case report emphasizes a well-known problem in bariatric surgery and encourages early diagnosis of the syndrome and immediate treatment that could prevent clinical deterioration in such patients. A “famous” occurrence in Israel was the Remedica case where infants’ formula was changed by the removal of vitamin B1, which caused severe neurological damage and even death in the babies fed on this defective, soy-based formula [8].

DIETARY RECOMMENDATION OF VIT B1 PRE- AND POST-BARIATRIC SURGERY

Vitamin B1, also known as thiamine, plays a central role in cerebral metabolism. The thiamine-dependent enzymes essential for the cerebral metabolism of glucose use thiamine pyrophosphate as a cofactor, accounting for 80% of the total thiamine present in nervous tissues [3]. The dietary requirement for thiamine is proportional to the caloric intake of the diet and ranges from 1.0 to 1.5 mg/day for healthy adults. Since thiamine is a water-soluble vitamin, it is not stored in the body and the excess is excreted in the urine. A state of severe depletion in patients on a strict thiamine-deficient diet becomes evident within 18 days [9]. Moreover, the pharmacological half-life of orally or parenterally administered thiamine is in the order of minutes and should be taken into account when treating thiamine deficiency [10].

Morbidly obese patients have poorly controlled dietary habits. Their intake is high in calories and refined carbohydrates, and since they also often use diuretics for the treatment of comorbidities, they are subject to a superimposed thiamine deficiency. A study published in 2005 [11] indicated that of 303 consecutive morbidly obese patients who were scheduled for weight-reduction surgery, 47 patients (15.5%) presented with low preoperative thiamine levels. Although the deficiency is usually subclinical it can be assumed that with higher demands due to surgery, rapid weight loss and loss of absorptive area after weight-reduction surgery, this subclinical preoperative deficiency may become full-blown and potentially fatal. The authors suggested that patients who are diagnosed with low thiamine levels before surgery be given 100 mg of thiamine orally twice daily for 1 month or until their thiamine levels are satisfactory. An initial parenteral route should be considered to ensure maximal bioavailability of thiamine.

During follow-up, for patients with

marginal low serum thiamine levels and prolonged nausea and vomiting, intravenous thiamine treatment at a dose of 100 mg every 6–8 hours (depending on the neurological condition of the patient) for 48 hours, followed by 100 mg of thiamine orally twice daily for 1 month is advocated, in addition to oral multivitamin supplementation. In most cases, thiamine deficiency symptoms disappear within 24 hours, but it can take as long as 4 months [12]. Prolonged vomiting should be considered as an indication for empiric thiamine treatment, even without biochemical deficiency [11]. In addition, a thiamine-rich diet as found in various foods such as enriched bread and cereals (whole grain and enriched), peas, beans, nuts, brown rice, and meats (especially beef and pork) is recommended.

THE TYPE OF BARIATRIC SURGERY

The risk of WE evolution depends also on the type of bariatric surgery performed. Purely restrictive operations, such as laparoscopic adjustable gastric banding and vertical banded gastroplasty, tend to cause fewer deficiencies than do malabsorptive procedures such as laparoscopic Roux-en-Y gastric bypass and biliopancreatic diversion [13,14]. It is reported that laparoscopic sleeve gastrectomy leads to fewer nutrient deficiencies in the first 2 years compared to laparoscopic Roux-en-Y gastric bypass [15].

RECOVERY OF WE PATIENTS

A significant number of WE patients, if recognized early, fully recover after thiamine administration. A systematic literature review since 1991 [2] reports the clinical outcome of WE in 83 patients after 0–20 months follow-up (median 5 months). Outcomes show that recovery was complete in 42 cases (51%) and incomplete in 41 (49%). Frequent sequelae were cognitive impairments (n=16), gait difficulties (n=13) and nystagmus (n=7).

CONCLUSIONS

Clinicians should suspect the diagnosis of WE, especially if changes in consciousness occur in patients with malnutrition or malabsorption as a consequence of prolonged vomiting after bariatric surgery or due to malignant tumors of the gastrointestinal tract. Thiamine levels pre- and post-surgery should be carefully monitored and a thiamine supplement should be administered before surgery in patients with low blood levels of vitamin B1 and immediately post-surgery in all patients until adequate levels are attained. These and other recommendations can be found in a detailed guidelines manuscript published by the American Society for Metabolic and Bariatric Surgery in 2008 [16].

In our bariatric center all patients are evaluated thoroughly before surgery, by a multidisciplinary team including a bariatric surgeon, dietician, psychologist, and an endocrinologist when considered necessary. Follow-up is performed every 3 months in the first year post-surgery and once a year in the subsequent period. All patients are prescribed a multivitamin formula for the first year post-surgery or until nutritional values are satisfactory. In addition to physical examinations, blood analyses and assessments of co-morbidities, patients are carefully monitored by a dietician and a psychotherapist and participate in a cognitive behavioral therapy program.

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