Case Report
Étude de cas

Alcohol and poor compliance as factors in Wernicke's encephalopathy diagnosed 13 years after gastric bypass

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A severely obese man achieved rapid and significant weight loss after Roux-en-Y gastric bypass. Thirteen years later Wernicke's encephalopathy developed as a result of the patient's alcoholism, poor compliance with his micronutrient intake, poor oral intake and the decreased absorptive ability of the small bowel. In selecting any operation to treat morbid obesity the possibility of metabolic problems must be considered as well as the potential for substantial weight loss.

Un homme très obèse a réussi à perdre beaucoup de poids rapidement après un pontage gastrique Roux-en-Y. Treize ans plus tard, une encéphalopathie de Wernicke a fait son apparition à la suite de l'alcoolisme du patient, parce qu'il a mal respecté l'absorption de micronutriments, mal mangé par la bouche et que la capacité d'absorption de l'intestin grêle avait diminué. Dans le choix de toute intervention qui vise à traiter l'obésité morbide, il faut tenir compte de problèmes métaboliques possibles, ainsi que de la possibilité d'une perte de poids importante.

Wernicke's encephalopathy is a tragic complication of alcoholism and severe starvation. It has been described as a rare but major complication of gastroplasty and gastric bypass that commonly occurs early after an operation and is associated with severe, prolonged vomiting and failure to take or retain vitamins. The manifestations of the condition include ophthalmoplegia, confusion, memory deficit, nystagmus, gait ataxia and death.[1,2] We describe a case of Wernicke's encephalopathy occurring 13 years after gastric bypass. We wish to emphasize the role that alcoholism compounded by poor follow-up and compliance may play in the development of this condition.

Literature review

The goal of obesity surgery is to achieve significant weight loss with improvement in the patient's comorbid conditions, which may include diabetes, hypertension and sleep apnea. Unfortunately, malabsorptive procedures can cause malnutrition.[3] Postoperatively, food intake is often restricted by emesis. If the patient does not drink sufficient milk or consume other high protein food or drink, protein malnutrition may result.[3] When the patient will not comply with the micronutrient regimen, deficiencies, including thiamin deficiency, will occur.[3]

Wernicke's encephalopathy can be a result of rapid weight loss.[1,4] Villar and Ranne reported that "the only prerequisite for the development of Wernicke's encephalopathy is a poor nutritional state."[1] Prolonged vomiting after gastroplasty or gastric bypass may be a contributing factor. Wernicke's encephalopathy may also result from starvation unrelated to alcohol in anorexia nervosa, refeeding syndrome or prolonged intravenous feeding.[1,5] This condition and severe peripheral neuropathy have been described after surgery for morbid obe-

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sity.[6–9] Abarbanel and associates[10] found a 4.6% incidence of Wernicke's encephalopathy in 457 patients who had undergone gastric bypass. MacLean, Rhode and Shizgal[3] reported a 50% incidence of low thiamin levels in 17 obese patients with excessive weight loss after gastroplasty. The thiamin deficiencies were observed clinically as well as biochemically. Thiamin stores are limited to 18 days.[3] Thus, depletion can occur rapidly with restricted dietary intake or prolonged vomiting. The Canadian recommendation for intake of thiamin is 1.1 mg/d, and the American value is 1.4 mg/d.[3,6,11]

Case report

The patient was first assessed for the surgical treatment of obesity in 1983 at 33 years of age when he weighed 153 kg and was 177 cm tall. His body mass index (BMI) was 49. He was on an antihypertensive medication and was a smoker. He was short of breath on exertion and had difficulty working because of leg and back pain. In a standardized preoperative questionnaire he claimed to drink only a few bottles of beer on weekends. On preoperative assessment by an internist he was thought to be a reasonable candidate for gastric bypass. Psychological assessment showed that he was anxious and immature. He admitted to a high intake of Pepsi and a beer intake of 8 to 14 bottles on weekends. He had good family support, an important factor following an operation.

Five months after initial assessment a Roux-en-Y gastric bypass was performed with creation of a small horizontal gastric pouch and a 45-cm Roux-en-Y limb of jejunum. Regular follow-up was carried out for 2 years after the operation but was sporadic thereafter. Early weight loss was above average. One month after operation the patient weighed 137.5 kg, a 10% weight loss (15.5 kg). At 2 months his weight was 125 kg, an 18% weight loss (28 kg). He complained of some vomiting, slight dizziness and arm weakness, but there were no clinical findings of neuropathy. He claimed to be taking his multiple vitamins. Three months after operation, an upper gastrointestinal series with barium revealed a normal appearance after gastric bypass with no evidence of obstruction. However, he required admission for investigation of vomiting and had a low serum potassium level of 2.6 mmol/L and mildly elevated liver enzyme levels. His weight at 3 months was 112.5 kg, a 26% weight loss (40.5 kg). Under dietary supervision he was able to tolerate pureed foods. No gallstones were identified and screening for hepatitis gave a negative result. His condition improved after this, and although he had some vague leg pain, he continued to work. His weight at 7 months was 85 kg (44% weight loss, 68 kg). At 1 year postoperatively he weighed 72.5 kg (53 % weight loss, 80.5 kg). He was seen every 3 months over the second postoperative year when his weight ranged from 73 to 77 kg.

In 1985, 18 months after the gastric bypass he reported slight tingling in his fingers. He had stopped taking vitamins and was told to resume taking them. Serum iron and vitamin B_{12} levels were in the low normal range. Monthly parenteral B_{12} was started by his family doctor.

In 1992, he was noted once again to be at high risk for micronutrient deficiencies given his very poor diet, high alcohol intake and lack of micronutrient supplementation. He was given counselling for appropriate micronutrient supplements and for improved diet. His alcohol intake dated back 20 to 25 years. During his drinking years he would consume between 8 and 29 bottles of beer daily with an average of 12. He did not return to the bariatric clinic until 1996.

In December 1995, he had his top and bottom molars extracted. His dentures did not fit properly and he was unable to eat because of his poor dentition. His alcohol intake continued unabated during this period of poor nutrition.

In April 1996 he was admitted to a community hospital with the diagnosis of possible Wernicke's syndrome. Significant cognitive changes had been noticed by his wife 2 months earlier. He was confused and had gait ataxia. His short- and long-term memory was poor. Protein malnutrition was also recognized (serum albumin level 27 g/L, total serum protein level 45 g/L). He was referred to a hepatologist at the London Health Sciences Centre who confirmed the diagnosis of Wernicke's encephalopathy. Neuropsychology testing revealed severe anterograde memory deficits, consistent with Korsakoff's syndrome.

Table I

Levels of Fat-Soluble Vitamins in a Patient With Wernicke's Encephalopathy Before and After Vitamin Supplementation

Date	Vitamin A (normal range 1.2–2.8 μmol/L)	Vitamin D (normal range (25–120 pmol/L)	Vitamin E (normal range 18–29 μmol/L)
Oct. 3, 1996	1.4	NA	8
Jan. 8, 1997	1.3	45	18

NA = not available

The patient was referred back to the original bariatric surgeon in October 1996 for review of his micronutrient regimen and diagnosis. He complained of significant diarrhea (more than 10 bowel movements daily). Stools were unformed, pale and foul smelling. He had a number of slow healing lesions on his arms and face. His tongue was magenta in colour and had extensive fissuring. He did not note any change in taste sensitivity and his tongue was not painful.

Blood work done in October 1996 revealed striking abnormalities for someone taking both fat and water-soluble vitamins (Tables I and II). Vitamins C and E levels were low, and the vitamin A level was low normal (normal range 1.2 to 2.8 µmol/L). Vitamin B_{12} and folic acid levels were normal. The thiamin level was markedly low as were the riboflavin and vitamin B_6 levels. The magnesium value was low as were the trace elements selenium, zinc and copper (Table III). Serum chromium, at 3.8 nmol/L, was within the normal reference range (2.3 to 3.8 nmol/L). Both the hemoglobin and hematocrit were also low (Table IV).

Neither the patient or his wife were able to complete food records for him because he was suffering from an unusual eating disorder. His wife could complete food records for the daytime hours but was unable to account for his nocturnal eating. He was seen to have very large midnight meals. His wife noted that he often hid food, expressing the fear of starvation. His bizarre eating behaviour and food obsession resembled the Prader–Willi syndrome.

Immediately after the initial diagnosis of Wernicke's encephalopathy the patient received 3 100-mg injections of thiamin. He was discharged on a dosage of 50 mg of thiamin daily. His wife added a "natural source" multivitamin, a B-complex multivitamin and doubled the thiamin dosage to 100 mg/d orally.

He was also placed on an enhanced micronutrient regimen (Centrum Select, 100 mg thiamin, 500 mg vitamin C, 400 IU vitamin E, 50 mg/d zinc, 50 µg/d selenium, 100 mg/d elemental iron and 650 mg/d calcium). Most importantly his wife agreed to adhere to this regimen and not to alter it. She also wished to continue with 10 mg folic acid against nutritional advice. Persistent diarrhea (up to 20 movements daily) may have been a factor in his deficiencies. His diarrhea decreased to approximately 10 movements per day by January 1997.

At that time micronutrient and trace metal levels had improved. His serum vitamin C and selenium levels and his hemoglobin and hematocrit were still below normal. He was advised to increase his intake of ferrous gluconate as he was only taking 300 mg/d. His wife now agreed to decrease the folic acid to 2.5 mg/d, given his very high serum folate level. His serum albumin level was 32 g/L in December 1997, still below normal. When assessed on Feb. 4, 1998, his weight was 76 kg and his mental state unchanged.

Table II

Levels of Water-Soluble Vitamins in a Patient With Wernicke's Encephalopathy Before and After Vitamin Supplementation

Date	Vitamin B_{12} (normal range 110–660 pmol/L)	Vitamin C (normal range > 25 µmol/L)	Red blood cell folate (normal range 372–1970 nmol/L)	Thiamin (normal range 18–75 nmol/L)	Pantothenic acid (normal range 4.6–8.3 µmol/L)	Niacin (normal range 9.7–23.5 µmol/L)	Riboflavin (normal range 8–40 nmol/L)	Vitamin B_6 (normal range 14.6–72.8 nmol/L)
Oct. 3, 1996	250	< 5	> 45.3	7.5	4.6	14.6	3.2	4.0
Jan. 8, 1997	267	12	> 2605	74.0	8.5	23.0	16.5	28.3

Table III

Levels of Trace Minerals in a Patient With Wernicke's Encephalopathy Before and After Supplementation Regimen

Date	Selenium (normal range 1.26–1.82 µmol/L)	Zinc (normal range 9.8–20.2 µmol/L)	Copper (normal range 11.0–22.0 µmol/L)	Magnesium (normal range 0.72–0.94 mmol/L)
Oct. 3, 1996	0.86	7.6	10.7	0.64
Jan. 8, 1997	1.08	10.3	12.8	0.77

Table IV

Hemoglobin Level and Hematocrit in a Patient With Wernicke's Encephalopathy

Date	Hemoglobin (normal range 135–180 g/L)	Hematocrit (normal range 40.0%–54.0%)
Oct. 6, 1996	133	39.6
Jan. 8, 1997	133	37.9

Discussion

Severe obesity carries many complications and is difficult to treat by nonsurgical means. But surgery is controversial because of prejudice against the obese, complications of early operations such as jejunoileal bypass and disappointing late results. Gastric procedures for obesity have been carried out for many years, but there are multiple modifications in technique. The restrictive gastroplasty procedures are easier and safer but less effective than the malabsorptive gastric bypass procedures. The most common operations have been the vertical banded gastroplasty devised by Mason[12] and the Roux-en-Y gastric bypass. Our decision to use a gastric bypass in this case was supported by a later randomized trial comparing gastroplasty and gastric bypass.[13] Gastric bypass gives much better long-term weight loss but also more vitamin deficiencies. Both operations can cause stoma obstruction and vomiting and produce acute thiamin deficiency.

Selection criteria for gastroplasty candidates are well defined by age, weight and health.[14] Prediction of compliance and weight loss is more difficult. A history of emotional problems or psychiatric admission may indicate a risk for postoperative complications.[15] Screening for alcohol intake is also very important in light of our experience with this patient. He was known to go on weekend drinking binges but the extent of his drinking problem was underestimated. Of course, drinking may increase after gastric bypass when the ability to eat large meals is controlled. In this patient there were other signs warning of trouble. He had lost 53% of initial body weight 1 year after the operation compared with an average of 33%. He was not reliable in taking vitamins or attending appointments. He was lost to follow-up for a number of years.

All candidates for obesity surgery must receive counselling on the importance of a lifelong regimen of micronutrients. Specific brands of multivitamins and minerals should be prescribed to ensure adequate intake of all vitamins, especially thiamin. Patient selection should include screening for alcohol intake and warning about the potential danger of alcohol, which inhibits the absorption of thiamin in a gut already surgically altered to be malabsorptive. This patient population needs long-term monitoring and nutritional counselling. In selecting any operation to treat morbid obesity the possibility of metabolic problems must be considered as well as the potential for significant weight loss.

References

1. Villar HV, Ranne RD. Neurologic deficit following gastric partitioning: possible role of thiamine. *JPEN J Parenter Enteral Nutr* 1984;8:575-7.
2. Reuler JB, Girard DE, Cooney TG. Current concepts. Wernicke's encephalopathy [review]. *N Engl J Med* 1985;312(16):1035-9.
3. MacLean LD, Rhode BM, Shizgal HM. Nutrition following gastric operations for morbid obesity. *Ann Surg* 1983;198(3):347-55.
4. Drenick EJ, Joven CB, Swendseid ME. Occurrence of acute Wenicke's encephalopathy during prolonged starvation for the treatment of obesity. *N Engl J Med* 1966;274(17):937-9.
5. Nadel AM, Burger PC. Wernicke encephalopathy following prolonged intravenous therapy. *JAMA* 1976;235 (22):2403-5.
6. Ockowski WJ, Kertesz A. Wernicke's encephalopathy after gastroplasty for morbid obesity. *Neurology* 1985;35: 99-101.
7. Fawcett S, Young GB, Holiday RL. Wernicke's encephalopathy after gastric partitioning for morbid obesity. *Can J Surg* 1984;27(2):169-70.
8. Marynaik O. Severe peripheral neuropathy following gastric bypass surgery for morbid obesity. *CMAJ* 1984;131(2):119-20.
9. Feit H, Glasberg M, Ireton C, Rosenberg RN, Thal E. Peripheral neuropathy and starvation after gastric partitioning for morbid obesity. *Ann Intern Med* 1982;96(4):453-5.
10. Abarbanel JM, Berginer VM, Osimani MD, Solomon H, Charuzi I. Neurologic complications after gastric restriction surgery for morbid obesity. *Neurology* 1987;37(2):196-200.
11. Health and Welfare Canada. Scientific Review Committee. *Nutrition recommendations: the report of the Scientific Review Committee*. Ottawa: The Committee, 1990.
12. Mason EE. Vertical banded gastroplasty. *Arch Surg* 1982;117:701-6.
13. Sugerman HJ, Starkey JV, Birkenhauer R. A randomized prospective trial of gastric bypass versus vertical banded gastroplasty for morbid obesity and their effects on sweets versus nonsweets eaters. *Ann Surg* 1987;205: 613-24.
14. Grace DM. Patient selection for obesity surgery. *Gastroenterol Clin North Am* 1987;16:399-413.
15. Valley V, Grace DM. Psychosocial risk factors in gastric surgery for obesity: identifying guidelines for screening. *Int J Obes* 1986;11:105-13.