

Wernicke encephalopathy after Sleeve Gastrectomy

Shalom Sharabi MD¹ and Naiel Bisharat MD PhD^{1,2}

¹Department of Medicine D, Emek Medical Center, Afula, Israel

²Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

KEY WORDS: bariatric surgery, Wernicke encephalopathy (WE), thiamine, morbid obesity

IMAJ 2012; 14: 708-709

For Editorial see page 692

Bariatric surgery has evolved significantly over the last decade and surgeons are showing increasing interest in this field, especially with the advent of newer and minimally invasive procedures. However, bariatric surgery is not without complications, some of which are life threatening, such as anastomotic leakage, pulmonary embolism, and Wernicke encephalopathy. The significant increase in the number of weight-loss procedures performed annually [1] suggests that physicians, both hospital-based and family physicians, need to familiarize themselves with the possible serious complications associated with these procedures. We describe here a young female patient who underwent bariatric surgery and suffered from two serious complications that eventually led to her death.

PATIENT DESCRIPTION

A 43 year old morbidly obese woman had undergone sleeve gastrectomy in another hospital. One week after hospital discharge she experienced nausea, vomiting, and poor oral intake. A week later she was hospitalized for these complaints. Upper gastrointestinal endoscopy showed mild esophagitis and gastritis. One month after hospital

discharge she continued to suffer from recurrent vomiting and poor oral intake and lost nearly 30 kg of body weight.

Two months after surgery she started to complain of generalized weakness and was unable to stand up from a seated position. In addition, her family members noticed an increased anxiety and restlessness.

On admission, 3 months after the surgery, the patient was afebrile with body temperature of 36.9°C and blood pressure 128/74; the rest of the physical examination was remarkable only for tachycardia of 105 beats/minute and a right inguinal abscess measuring 2 x 1 cm which ruptured spontaneously and drained foul-smelling pus. The white blood cell count was normal and the C-reactive protein level was mildly elevated. The patient was empirically treated with amoxicillin/clavulanate. A Gram stain of pus sample from the inguinal abscess showed numerous polymorphonuclear leukocytes and culture grew *Proteus mirabilis*. No growth was observed in blood cultures. Computed tomography of the abdomen and pelvis with contrast media was remarkable only for a small abscess in the right inguinal region without involvement of the adjacent blood vessels. On the second day of her admission the patient became confused with visual and auditory hallucinations. Neurological examination showed somnolence, nystagmus, and diplopia. A CT of the brain was normal. A presumed diagnosis of Wernicke encephalopathy was made and intravenous thiamine, 600 mg daily, was started. On the next day her condition deteriorated with respiratory failure and loss of consciousness; she was intu-

bated and mechanically ventilated, the thiamine dosage was increased to 1500 mg per day, and she was transferred to the intensive care unit. Within 48 hours a dramatic improvement was noted; the patient regained consciousness and started breathing normally. Pretreatment levels of vitamin B1 and 6 were low, 17 ng/ml (normal 28–85 ng/ml) and 7 ng/ml (normal 9–27) respectively, while vitamin B12 levels were within normal limits. After 1 week of thiamine treatment the patient was alert and coherent, and her neurological examination was remarkable for some memory loss and moderate to severe weakness in her lower limbs attributed to peripheral polyneuropathy.

After 3 weeks of hospitalization the patient was referred to a rehabilitation facility. After 2.5 months in rehabilitation the patient was still suffering from partial memory loss, gait ataxia, and severe peripheral neuropathy. In the 12th week of rehabilitation the patient was referred to our hospital due to abdominal pain, nausea, and vomiting. An abdominal CT scan showed distended and edematous loops of small and large bowels without evidence of mechanical obstruction, consistent with a presumptive diagnosis of paralytic ileus. The patient was treated with intravenous fluids, bowel rest, and total parenteral nutrition due to severe hypoalbuminemia. However, on the second day of admission her condition worsened with fever, hypotensive shock, and multi-organ failure. She was treated with broad-spectrum antibiotics and vasopressors but without any effect and she eventually died. Blood cultures grew *Escherichia coli* and *Providencia stuartii*.

COMMENT

We have described a patient who suffered from two major complications of bariatric surgery that eventually led to her death. This is the first reported case of WE after bariatric surgery in Israel. The potential complications of bariatric surgery and specifically the nutritional deficiencies were widely addressed in a recent review from Israel [2].

WE is an acute neuropsychiatric syndrome that is classically characterized by a triad of clinical symptoms: nystagmus and ophthalmoplegia, changes in mental status, and unsteadiness of stance and gait [3]. Many other symptoms and signs may also appear, especially if there is a delay in treatment; these include psychosis, incontinence, tachycardia, hearing loss, dysarthria, depression, papilledema, dysphagia, seizures, myoclonus, asterixis, and chorea. WE results from a deficiency of vitamin B1 (thiamine), which in its biologically active form, thiamine pyrophosphate, is an essential coenzyme in several biochemical pathways in the brain. The diagnosis of WE remains largely clinical, and the best aid for a correct diagnosis is clinical suspicion. Clinicians should consider the disorder in any given patient with unbalanced nutrition or in the clinical setting of sub-acute or chronic diseases that increase metabolism or alter the ingestion and absorption of food [3]. The presumptive diagnosis of WE can

be confirmed by determining blood thiamine concentrations (as in our case) or by measuring the red blood cell transketolase activity. However, these measurements are limited by a lack of specificity and technical difficulty [3].

The prevalence of WE after bariatric surgery is largely unknown. Nevertheless, a recent review addressing WE after bariatric surgery suggested that the number of WE cases after bariatric surgery is substantially higher than previously reported [4]. The author concluded that surgeons, allied health providers, and patients need to be aware of the predisposing factors and symptoms of WE. In the case presented, the patient's initial symptoms were related to the surgical procedure, and once that was ruled out her suffering was attributed to adaptational factors rather than to physical pathology, which ultimately caused a significant delay in her treatment.

The patient was initially treated with a high dosage (600 mg) of thiamine and due to her deterioration it was increased to 1500 mg/day. It is unclear what the most appropriate dose should be as there is insufficient evidence from controlled trials to guide clinicians regarding dosage, frequency, and duration of thiamine treatment in patients with WE [5]. Regretfully, and despite partial recovery from WE she succumbed to septic shock and died from multi-organ failure.

Currently, bariatric surgery has become the only modality that provides a considerable and sustained weight

loss for a morbidly obese individual with resultant improvement in obesity-related comorbidities. Nevertheless, there are potentially life-threatening, and preventable, complications. Patients should be warned of nutritional risk factors such as repeated vomiting. Clinicians need to keep in mind that behavioral disturbances or uncooperativeness could potentially be features of WE.

It is highly recommended that every patient who is referred to bariatric surgery be accompanied by professional health care providers during the first 3–6 months after surgery, preferably in a bariatric clinic.

Corresponding author:

Dr. S. Sharabi

Dept. of Medicine D, Emek Medical Center, Afula 18101, Israel

Phone: (972-4) 649-4521

Fax: (972-4) 649-4518

email: shalomsharabi05@gmail.com

References

1. Khalailah A, Matot I, Schweiger C, Appelbaum L, Elazary R, Keidar A. Laparoscopic Roux-en-Y gastric bypass for the treatment of morbid obesity: experience with 50 patients. *IMAJ Isr Med Assoc J* 2008; 10: 350-3.
2. Schweiger C, Keidar A. Nutritional deficiencies in bariatric surgery patients: prevention, diagnosis and treatment. *Harefuah* 2010; 149 (48): 715-20 (Hebrew).
3. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. *Lancet Neurol* 2007; 6: 442-55.
4. Aasheim ET. Wernicke encephalopathy after bariatric surgery: a systematic review. *Ann Surg* 2008; 248: 714-20.
5. Day E, Bentham P, Callaghan R, Kuruvilla T, George S. Thiamine for Wernicke-Korsakoff Syndrome in people at risk from alcohol abuse. *Cochrane Database Syst Rev* 2004: CD004033.

WE = Wernicke encephalopathy

A child of five would understand this. Send someone to fetch a child of five

Groucho Marx (1890-1977), American comedian. A master of quick wit, he made 13 feature films with his siblings the Marx Brothers. His distinctive appearance, carried over from his days in vaudeville, included quirks such as an exaggerated stooped posture, glasses, cigar, and a thick greasepaint mustache and eyebrows

Erratum

In the August issue, the article "Anemia associated with acute infection in children" by A. Ballin, Y. Senecky, U. Rubinstein, E. Schaefer, R. Peri, S. Amsel, M. Vol, Y. Amit and M. Boaz (IMAJ 2012; 14 (8): 484-7), a mistake occurred in the spelling of the fourth author's name. The correct spelling is E. Schaefer and not E. Schaeffert as printed.