An increasing number of patients who have had massive weight loss are seeking body contouring surgery. We present the case of a patient who became severely hypotensive and hypothermic and required intensive care after undergoing a panniculectomy. We also discuss the lessons that can be learned from this case.

**CASE PRESENTATION**

A 72-year-old woman presented to the Plastic Surgery Department at Faith Regional Health Services (Norfolk, Nebraska) in October 2011 with a large pannus after claiming to have lost 322 lb of body weight by diet alone over a 5-year period (weight decreased from 550 lb to 228 lb; body mass index decreased from 78 to 32). Her weight had been stable for the past 2 years.

She had a medical history of diabetes mellitus, atrial fibrillation, tubal ligation surgery, depression, and oxycodone allergy. Her medications at the time of presentation included warfarin (suspended preoperatively), atenolol, sitagliptin, metformin, and sertraline. Results of blood tests performed 2 weeks preoperatively were unremarkable (hemoglobin, 14.6 g/dL; sodium, 141 mmol/L; potassium, 4.2 mmol/L; albumin, 3.7 g/dL; prealbumin, 17 mg/dL), and her electrocardiogram showed no evidence of ischemia.

An uneventful panniculectomy was performed by the senior author (T.H.) under a general anesthetic. Operating time was 3.5 hours, with an estimated blood loss of 250 mL. Intravenous fluids (3 L) were administered intraoperatively. The resected pannus weighed 16 lb.

Ms Gardiner is a Surgical Trainee Year 2 in the Plastic Surgery Department, Royal Free Hospital, London, United Kingdom. Dr Hartzell is a plastic surgeon at Faith Regional Health Services, Norfolk, Nebraska.

**Corresponding Author:**
Ms Sonya Gardiner, Royal Free Hospital, Pond Street, London, UK NW3 2QG.
E-mail: sg410@cam.ac.uk
At 6 hours postoperatively, the patient’s cardiovascular and respiratory signs were stable with a drain output of 10 to 20 mL/h. However, at 8 hours postoperatively, she began to show signs of clinical deterioration with a glucose level of 324 mg/dL, and insulin therapy was started. Soon after, she became cardiovascularly unstable with a blood pressure of 60/42 mm Hg and heart rate of 70 bpm. Her temperature was below 90°F and could not be registered on a thermometer. She was drowsy, but results from abdominal examination, blood tests, electrocardiography, echocardiography, and chest radiography were unremarkable. The patient showed no improvement after aggressive fluid resuscitation and was transferred to the intensive care unit with an Acute Physiology and Chronic Health Evaluation score of 27 and predicted mortality rate of 35%. Inotropic support and invasive monitoring were then started.

In the absence of a clear diagnosis, the following empirical therapies were administered: electrolyte repletion of calcium and magnesium, as well as ongoing fluid resuscitation, including blood products, broad-spectrum antibiotics, and steroids. Specialists in cardiology, hematology, renal, pulmonology, and infectious diseases were consulted. The patient continued to remain profoundly hypothermic, registering a maximum rectal temperature of 92°F despite aggressive rewarming. Her symptoms were nonresponsive to maximum inotropic support, including maximum doses of dopamine, phenylephrine, epinephrine, norepinephrine, and vasopressin. She eventually became anuric and acidic, with a pH of 7.0 and elevated blood lactate level of 7 mmol/L, and developed disseminated intravascular coagulopathy with a partial thromboplastin time of 200 seconds and fibrinogen level of 80 mg/dL. Abdominal and chest computed tomography revealed a small hematoma. Exploratory laparotomy was performed to investigate for ischemic bowel: the bowel was edematous but not ischemic. Despite massive resuscitation with blood products and clotting factors, the patient continued to deteriorate until she showed no response to verbal stimuli. On neurologic examination, painful stimuli were applied to assess for cerebral function. At that time, it was noted that she had lateral gaze palsy in her right eye, and a diagnosis of Wernicke encephalopathy was considered.

A dose of 500 mg intravenous thiamine was administered. Within 2 hours, the patient’s blood pressure returned to within the normal range, and vasopressors were withdrawn without any further intervention. She became normothermic at the same time. Within 6 hours after the administration of thiamine, the patient’s pH and blood lactate levels had decreased to within normal range (Figure 1).

The patient admitted to 15 to 20 episodes of forced emesis per day within the 2 weeks immediately before panniculectomy in an attempt to maximize weight loss. She made a full recovery with no residual neurologic deficit and was discharged from the hospital on postoperative day 7.

**DISCUSSION**

Thiamine (vitamin B₁) is a cofactor important in enzymatic carbohydrate metabolism, and thiamine deficiency can impair cardiac and neurologic function. Wernicke encephalopathy, an acute neurologic syndrome associated with thiamine deficiency, was first described by Carl Wernicke in 1881. It is characterized by a triad of mental status changes, ophthalmoplegia, and gait ataxia. However, this classic triad is apparent in as few as 10% of patients. Other associated signs include hypothermia, hypotension, lactic acidosis, high output cardiac failure, vestibular...
dysfunction, nystagmus, and polyneuropathy. Symptoms and signs resolve rapidly after parenteral thiamine is administered, but unfortunately, residual neurologic dysfunction is common. Recognition of Wernicke-Korsakoff syndrome relies heavily on clinical diagnosis. Neuroradiologic imaging provides low diagnostic sensitivity, despite radiologic advances and an improved knowledge of associated magnetic resonance imaging findings such as symmetrical alterations in the thalami and mamillary bodies.

Historically, thiamine deficiency and the related Wernicke-Korsakoff syndrome have been associated with severe alcohol misuse. However, there are many reported cases of thiamine deficiency in the nonalcoholic, nutritionally deficient population, including patients with hyperemesis gravidarum, malignancy, acquired immunodeficiency syndrome, postbariatric surgery, prolonged total parenteral nutrition use, and long-term dialysis.

Our case report is an important contribution to the scarce literature on thiamine deficiency in the context of plastic and reconstructive surgery. Sebastian et al reported a case of postoperative acute thiamine deficiency in a patient who underwent multiple aesthetic procedures, including a panniculectomy, brachioplasty, and face/neck lift. The patient presented postoperatively with hyperglycemia and confusion. In contrast with our case, that patient had a history of gastric bypass, which is known to be associated with thiamine deficiency. Thiamine deficiency in our patient was likely caused by repetitive emesis, and subsequent Wernicke encephalopathy was precipitated by surgery and hyperglycemia. We would therefore encourage the routine administration of thiamine in patients undergoing plastic surgery after weight loss to avoid the potentially fatal sequelae described in our case.

During preoperative evaluation, the patient appeared to be a good candidate for surgery. She had a history of massive weight loss but a stable weight for 2 years, a large pannus with rash and wounds, and normal blood test results, along with a satisfactory medical assessment 2 weeks preoperatively. She claimed to have lost the weight by diet alone, corroborated by her primary physician and family. No preoperative psychologic assessment was performed. In retrospect, a more thorough assessment may have revealed a history of bulimia and highlighted concerns about her nutritional state and, therefore, her fitness for surgery.

CONCLUSIONS

The importance of a high index of suspicion for thiamine deficiency is underscored by the fact that it does not present classically in most cases. To identify at-risk patients, surgeons should perform a thorough preoperative assessment of all patients seeking plastic and reconstructive surgery after massive weight loss. In addition, administration of intravenous thiamine should be included in the routine postoperative care of all patients who undergo plastic surgery after weight loss.

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