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Neurological Complications of Bariatric Surgery

Jerry Clay Goodman [1]

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Abstract Obesity has attained pandemic proportions, and bariatric surgery is increasingly being employed resulting in turn to more neurological complications which must be recognized and managed. Neurological complications may result from mechanical or inflammatory mechanisms but primarily result from micro-nutritional deficiencies. Vitamin B_{12}, thiamine, and copper constitute the most frequent deficiencies. Neurological complications may occur at reasonably predictable times after bariatric surgery and are associated with the type of surgery used. During the early post-operative period, compressive or stretch peripheral nerve injury, rhabdomyolysis, Wernicke's encephalopathy, and inflammatory polyradiculoneuropathy may occur. Late complications ensue after months to years and include combined system degeneration (vitamin B_{12} deficiency) and hypocupric myelopathy. Bariatric surgery patients require careful nutritional follow-up with routine monitoring of micronutrients at 6 weeks and 3, 6, and 12 months post-operatively and then annually after surgery and multivitamin supplementation for life. Sustained vigilance for common and rare neurological complications is essential.

Keywords Bariatric surgery · Neurological complications · Thiamine deficiency · Copper deficiency · Cobalamin deficiency

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✉ Jerry Clay Goodman
jgoodman@bcm.edu

[1] Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030, USA

Introduction

For the first time in human history, there are as many human beings on earth are overfed as are underfed. This has resulted in rampant, indeed epidemic, obesity with the associated medical and social complications. When dietary restriction is insufficient, patient's resort to bariatric surgery which is basically designed to impair absorption macronutrients. This iatrogenic malabsorption may lead to unintended consequences including vitamin and metal deficiencies which in turn lead to neurological complications. In addition to late effects, operative and perioperative neurological complications have been reported. In this review, the surgical procedures, nutritional deficiencies, and neurological complications of bariatric surgery will be reviewed. Clinical recognition of these nutritional deficiencies is critical as these are amenable to correction, and if they are not corrected, permanent neurological deficits can result. There are also a number of rare neurological complications that can ensue following bariatric surgery including Guillain-Barré syndrome (GBS), unmasking of subclinical ornithine carbamylase deficiency, D-lactate encephalopathy, and possibly neurological disorders of the children of pregnant patients.

Obesity is defined by the body mass index consisting of an individual's weight in kilograms divided by the height in meters squared. A normal body mass index (BMI) is in the range of 18.5 to 24.9. The BMI between 25 and 30 is regarded as overweight, 30 or greater is considered obese, and the BMI exceeding 40 is morbidly obese. Some authors divide obesity into grades with grade 1 consisting of a BMI of 30 to 34, grade 2 being a BMI of 35 to 39, and grade 3 defined as a BMI greater than 40. Obesity increases the risk of diabetes mellitus, arterial hypertension, hypercholesterolemia, osteoarthritis, asthma, pulmonary disease, sleep apnea, and depression. Currently, it is estimated that more than two thirds of adults in the USA are

overweight or obese. Of these, 35 % are obese or extremely obese as measured in 2011 through 2012. Extreme obesity is somewhat more common in women than men, is common in middle age, and is most common in non-Hispanic black adults. Fortunately, there has been no significant increase in obesity prevalence in the USA in adults between 2003 and the present. However, obesity attained epidemic proportions during the 1980s and 1990s and will continue to contribute to health care spending, morbidity, and mortality [1].

Billions of dollars are spent annually on dietary measures, exercise, and other non-surgical approaches for weight reduction. Increasingly, however, patients were resorting to bariatric surgery. The number of surgeries performed in the USA has increased over the last three decades, and it is estimated by the American Society for Metabolic and Bariatric Surgery (ASMBS) that 179,000 bariatric surgery procedures were performed in 2013 and more recent estimates suggest that up to 220,000 procedures may be performed. Some bariatric surgery is commercially driven; however, national guidelines reserve bariatric surgery for individuals who have failed attempts at nonsurgical weight reduction and who have an elevated BMI with obesity-related comorbidity or a BMI greater than 40 with or without comorbidity. There is no question that bariatric surgery in appropriately selected patients results of the significant weight reduction. In some instances, weight reduction of 70 to 83 % occurs in the first 12 to 24 months following bariatric surgery, and often comorbidities are much better controlled. From a neurological therapeutics perspective, there has been case report of patients with pseudotumor cerebri as well as sleep apnea whose obesity was treated using bariatric surgery leading to resolution of symptoms [2–4]. Neurocognitive dysfunction is common in morbidly obese individuals, and improvement in cognitive function has been described following bariatric surgery, although the precise mechanism of this improvement is unclear [5].

Bariatric Surgery Procedures

Several different surgical procedures have been used to assist people in weight reduction [6, 7] (http://asmbs.org/patients/bariatric-surgery-procedures) [8–12]. These interventions can be broadly as restrictive or absorptive, but there is significant mechanistic overlap. The overall objective of bariatric surgery is to reduce intake or absorption of macronutrients including fats, carbohydrates, and proteins which reduce caloric intake. Concurrently, however, there can be reduction in absorption of micronutrients including essential minerals and vitamins.

Laparoscopic adjustable gastric band and sleeve gastrectomy are considered restrictive procedures, deriving their efficacy from interference with the volume capacity of the proximal stomach. The gastric band approach involves mechanical constriction of the stomach, whereas the sleeve gastrectomy involves surgical removal of approximately 80 % of the stomach to produce a lower volume banana-shaped organ.

The biliopancreatic diversion and the Roux-en-Y gastric bypass are considered malabsorptive procedures, as they interfere with normal digestive and absorptive processing of food, but they both also have restrictive elements. The Roux-en-Y gastric bypass—often called gastric bypass—is considered the "gold standard" of weight loss surgery and is the most commonly performed bariatric procedure worldwide. A small stomach pouch, approximately 30 ml in volume, is created by dividing the upper portion of the stomach from the rest of the stomach. Next, the proximal portion of the small intestine is divided, and the distal end of the divided small intestine is brought up and connected to the newly created small stomach pouch. The procedure is completed by connecting the proximal portion of the divided small intestine to the small intestine further down so that the stomach acids and digestive enzymes from the bypassed stomach and first portion of small intestine will eventually mix with the food. The gastric bypass works by several mechanisms. First, similar to most bariatric procedures, the newly created stomach pouch is considerably smaller facilitating consumption of significantly smaller meals. Additionally, because there is less digestion of food by the smaller stomach pouch and there is a segment of small intestine that is bypassed, less absorption occurs. Importantly, the rerouting of the food stream produces changes in gut hormones that promote satiety, suppress hunger, and reverse the primary mechanisms by which obesity induces type 2 diabetes.

The biliopancreatic diversion with duodenal switch—abbreviated as BPD/DS—is a procedure with two components. First, a small, tubular stomach pouch is created by removing a portion of the stomach, very similar to the sleeve gastrectomy. Next, a large portion of the small intestine is bypassed. The duodenum is divided just past the outlet of the stomach. A segment of distal small intestine is brought up and connected to the outlet of the newly created stomach, so that when the patient eats, the food goes through a newly created tubular stomach pouch and empties directly into the last segment of the small intestine. Roughly three fourths of the small intestine is bypassed, but since this portion carries the bile and pancreatic enzymes that are necessary for the breakdown and absorption of protein and fat, the proximal small intestine is reconnected to the distal portion of the small intestine. The BPD/DS restricts the amount of food that is consumed; however, over time, this effect lessens and patients are able to eventually consume near "normal" amounts of food. The food does not mix with the bile and pancreatic enzymes until very far down the small intestine which results in a major decrease in the absorption of calories from protein and fat as well as fat-soluble vitamins. Like gastric bypass and sleeve gastrectomy, BPD/DS affects gut hormones in a manner that impacts

hunger and satiety as well as blood sugar control. The BPD/DS is considered to be the most effective bariatric surgery for the treatment of diabetes.

Gastric banding and sleeve gastrectomy mechanically restrict the passage of food through the stomach and is less likely to be associated with clinically significant nutritional deficiency, whereas when surgical bypass of the stomach is performed as in a Roux-en-Y bypass or BPD/DS, micronutrient malabsorption can ensue.

It is been estimated that up to 5 % of patients who undergo bariatric surgery may experience neurological complications largely due to micronutrient malabsorption. The risk of developing nutritional disorders following bariatric surgery increases as the intervention becomes increasingly malabsorptive. Therefore, the risk stratifies from least to greatest as follows: laparoscopic adjustable gastric banding < vertical sleeve gastrectomy < divided Roux-en-Y gastric bypass < BPD/DS.

There are well-developed and articulated guidelines for nutritional management following bariatric surgery, but issues of compliance, hyperemesis, preexisting micronutrient inadequacy, and incomplete follow-up may lead to micronutrient deficiency states leading to neurological complications [13–16]. Prompt recognition of these neurological complications is essential as they may become irreversible without appropriate intervention which consists simply of replacing micronutrients. In addition to micronutrient deprivation, there are other mechanisms of neurological complications of bariatric surgery including those occurring in the immediate postoperative period including rhabdomyolysis and of pressure neuropathies as well as less fully understood long-term metabolic effects. The precise frequency of neurological complications is not precisely known; however, not surprisingly, the more closely and carefully the patients are followed, the more complications are detected [17–19]. Early retrospective suggested that neurological complications occurred in about 1–5 % of patients with peripheral neuropathy accounting for 60–80 % of neurological complications and central nervous system (CNS) complications being less common. More recent reports suggest that neurological complications occur in up to 9 % of patients with peripheral nervous system (PNS) complications accounting for 75 %. It should be noted that the more recent studies are occurring at a time when neurological complications of bariatric surgery are better understood and mitigating strategies are stronger, so it is likely that early studies underestimated the frequency of neurological complications of bariatric surgery.

In considering the differential diagnosis of neurological complications of bariatric surgery, it is fruitful to classify them as those that occur in the immediate perioperative period, those that occur days to weeks following the bariatric surgery, and those which may occur months to years or even decades following the surgery. The timeline of the more common neurological complications of bariatric surgery are shown in Table 1.

Perioperative Complications

In the immediate perioperative period, neurological injury is largely confined to the PNS. Individuals undergoing bariatric surgery require attention to positioning, movement, and special operative equipment in order to avoid rhabdomyolysis from direct muscle pressure. Rhabdomyolysis can be severe and even lethal in this setting [18]. Compression mononeuropathies may also occur during anesthesia as the patient may be exposed to retractors, traction, poor positioning, or placement of arterial monitoring catheters which may precipitate immediate perioperative neural injury. Ulnar, median, femoral, and peroneal nerve palsies have all been described as complications of bariatric surgery. Lumbosacral and brachial plexopathies can also occur. Following surgery, as weight loss ensues, peripheral nerve compression can occur due to loss of tissue that previously padded the nerve.

Micronutrient Deficiencies Associated with Bariatric Surgery

Micronutrient deficiencies may manifest days, weeks, months, years, or even decades following bariatric surgery. Not surprisingly, individuals who are obese often come to

Table 1 Common neurological complications by time after bariatric surgery with most common complications indicated in italicized bold

Neurological complications in the immediate perioperative period
Compression neuropathy
Stretch injuries to brachial plexus
Rhabdomyolysis
Neurological complications occurring weeks to months after bariatric surgery
Thiamine deficiency
Wernicke's encephalopathy
Peripheral neuropathy
GBS
Episodic encephalopathy
Neurological complications occurring months to years after bariatric surgery
Vitamin A deficiency
Vitamin B_{12} (cobalamin) deficiency
Vitamin D deficiency
Vitamin E deficiency
Copper deficiency

Bold indicates the time frame after bariatric surgery that complications occur

bariatric surgery with preexisting micronutrient deficiency [20]. Some series suggest that 20 to 30 % of such patients are already micronutrient deprived at the time of surgery, and following surgery, these vitamin and mineral deficiencies may worsen due to prolonged vomiting, reduced absorption, altered diet, altered gut microbiome, and loss of intrinsic factor and gastric acid. The rate at which a patient develops micronutrient neurological complications may depend on their nutritional state prior to surgery. In the post-bariatric surgery period, deficiency of vitamin B_{12} has been described in up to 25 % of patients, folate in 20 %, and thiamine in 1 %. Other micronutrient deficiencies that can lead to neurological manifestations include copper, vitamin E, pyridoxine, niacin, and vitamin D.

Nutritional Neurological Complications Occurring Weeks to Months After Surgery

One of the most common and serious complications of bariatric surgery is the development of Wernicke's encephalopathy due to thiamine deficiency [21–27]. The classical features that include ataxia, nystagmus, and memory disturbance can be seen within days to weeks following bariatric surgery, but the complete triad of findings is seen only in the minority of patients. These patients may experience hallucinations, confusion, and may become combative. A common theme among patients who develop this complication is uncontrollable emesis. To some extent, this resembles the thiamine deficiency that can be seen in emesis gravidarum. The diagnosis can be confirmed by measuring red blood cell transketolase activity, a thiamine-dependent enzyme. Neuroimaging may show aqueductal gray and mammillary body hemorrhage as well as T2 prolongation in the dorsal medial thalamus, fornix, and aqueductal region. Hemorrhage has been seen in the fornix and anterior thalamus. Individuals with Wernicke's encephalopathy respond to IV thiamine administration. Some advocate emergency treatment with a dose of 500 mg IV three times a day for 2 to 3 days followed by 250 mg daily [28••]. Thiamine should particularly be given prophylactically to individuals who are experiencing emesis following bariatric surgery. Recovery will generally occur in the weeks to months following administration of thiamine; however, if the condition is insufficiently treated, permanent deficits including ocular motility disturbances, ataxia, and mental status changes may occur. The CNS manifestations of thiamine deficiency are frequently accompanied by peripheral neuropathy taking the form of a symmetrical axonal lower extremity predominant sensorimotor neuropathy [13, 14, 18, 28••, 29••, 30, 31]. This is a rapidly progressive polyradiculoneuropathy which is often painful and ascending. In contrast to GBS, the patient has normal cerebrospinal fluid (CSF) protein levels.

Nutritional Neurological Complications Occurring Months to Years After Surgery

Neurological complications that occur weeks to months after bariatric surgery most commonly include vitamin B_{12} (cobalamine), folate, vitamin D, vitamin E, and copper deficiency.

Vitamin B_{12} deficiency results from absence or reduction of stomach acidity as well as intrinsic factor impairing absorption in the distal ileum. Hepatic stores are usually sufficient to maintain the patient for several months and possibly years following initial inadequate dietary supplementation although, as noted earlier, a significant number of patients coming to bariatric surgery are already micronutrient depleted [17, 28••, 29••, 32–35]. The patient will present with paresthesias, decreased muscle stretch reflexes, and weakness associated with spasticity and loss of position and vibratory sense. The patients may develop loss of vision, dementia, psychosis, and altered mood. Rarely, autonomic symptoms may develop. All patients who underwent bariatric surgery should receive vitamin 12 supplementation, but this complication takes months to years to develop, and both patient and physician may become lax about supplementation.

Folate deficiency is a very rare complication of gastric bypass and is associated with peripheral neuropathy and myelopathy [28••, 32, 36]. Prophylactic supplementation is effective but, as in the case of conventional combined system degeneration, the folate may mask the hematological manifestations of B_{12} deficiency.

Gastric bypass has also been associated with vitamin D deficiency with secondary hypocalcemia [28••, 29••, 32, 35]. These patients may have nonspecific musculoskeletal pain and weakness, but some will develop facial twitching and ophthalmoplegia due to hypocalcemia. Proximal muscle weakness ascribable to a myopathy has also been seen in vitamin D deficiency.

Vitamin E deficiency may also develop within months following back bariatric surgery, particularly procedures such as the BPD/DS that impair absorption of fat-soluble vitamins, but some instances have been described of delayed onset deficiency as long as a decade after the surgery [28••]. Neurological features include loss of muscle stretch reflexes, ataxia, impaired proprioception, ophthalmoplegia, eyelid ptosis, and dysarthria.

Vitamin A, another fat-soluble vitamin, deficiency may develop late (1 to 4 years) after bariatric surgery as the liver usually has significant stores of this vitamin. Nocturnal vision failure ("night blindness") is the major neurological manifestation, and this may be accompanied by cutaneous features including pruritus, dry skin, and dry hair. Xerophthalmia with corneal and conjunctival xerosis can threaten vision as well.

The major mineral micronutrient deficiency seen following bariatric surgery is copper deficiency [36–43]. This deficiency

leads to a syndrome closely resembling B_{12} deficiency which may involve common defects of single carbon transfer (methyl group) metabolism of myelin via methionine synthase and *S*-adenosylhomocysteine. Copper is an essential component of a number of other metalloenzymes including superoxide mutase, cytochrome oxidase, tyrosinase, and dopamine monooxygenase. Patients present with myelopathy with spasticity coupled with loss of proprioception and fine touch sense. There is usually an associated polyneuropathy which will reduce muscle stretch reflexes and result in length-dependent paresthesias in a stocking glove distribution. The likely cause of the copper deficiency is reduced effective absorption especially when the food bypasses most of stomach and the duodenum. Copper deficiency can also result from interference with copper absorption by excessive zinc which paradoxically can be quite high in multivitamin tablets.

Copper deficiency myelopathy (hypocupric myelopathy) may be accompanied by anemia and neutropenia but not invariably so. Laboratory studies show low serum copper and ceruloplasmin levels accompanied by low urinary copper levels. Imaging shows increased T2 signal of the dorsal columns usually in the cervical and thoracic, and occasionally lateral column signal abnormality may be seen. Most cases of hypocupric myelopathy follow bariatric surgery or zinc overloading, but up to 20 % are of unknown cause.

Treatment consists of copper repletion parenterally followed by long-term supplementation. As with thiamine deficiency, prompt intervention is essential to prevent irreversible neurological damage. Complete resolution of neurological manifestations is very uncommon even with treatment which usually results in simple stabilization and partial improvement.

Selenium deficiency has also been described following bariatric surgery, but the nervous system is largely spared. Cardiomyopathy, which may be lethal, is the primary clinical manifestation [44].

Uncommon Neurological Complications of Bariatric Surgery

Polyneuropathy due to thiamine deficiency is well described following bariatric surgery, but demyelinating as well as axonal polyradiculoneuropathy has also been described [18, 30, 45–48]. The presentation is that of classical GBS with ascending paralysis, loss of muscle stretch reflexes, and dissociation of protein and cell count (albumino-cytologic dissociation) in the CSF. The etiological relationship to the surgery is unclear.

The metabolic stress of high protein diet may unmask previously asymptomatic ornithine transcarbamylase (OTC) deficiency [49]. There is a single case report of a patient in her late 20s who had bariatric surgery for morbid obesity, and within weeks of the surgery, she experienced thiamine deficiency with concurrent peripheral neuropathy as well as Wernicke's encephalopathy. Appropriate vitamin supplementation therapy was administered, but after several months, she developed episodic encephalopathy associated with hyperammonemia. Measurement of OTC showed markedly reduced levels. In retrospect, it was noted that the patient had previously avoided high protein loads and thereby avoided encephalopathy.

Similarly, episodic encephalopathy characterized by lethargy, confusion, behavioral alterations, ataxia, and weakness has been seen with D-lactic acidosis following short bowel syndrome and jejunoileostomy [50–54, 55•, 56–58]. The D-lactic acid stereoisomer as opposed to physiological L-lactic acid is produced by gut flora following a high carbohydrate load in the diet, and this condition has also been described in short bowel syndrome. Elevated D-lactate can be detected in urine, blood, and stool, and it is essential to recognize that conventional clinical laboratory methods detect only the L-lactate isomer. The clinical presentation of D-lactic acidosis is characterized by episodes of encephalopathy and metabolic acidosis. The diagnosis should be considered in a patient who presents with metabolic acidosis and high serum anion gap, normal lactate level, negative Acetest, short bowel syndrome or other forms of malabsorption, and characteristic neurologic findings. Development of the syndrome requires the following: (1) carbohydrate malabsorption with increased delivery of nutrients to the colon, (2) colonic bacterial flora of a type that produces D-lactic acid, (3) ingestion of large amounts of carbohydrate, (4) diminished colonic motility, allowing time for nutrients in the colon to undergo bacterial fermentation, and (5) impaired physiological D-lactate metabolism. The most effective immediate treatment consists of elimination of all oral intake of food while providing intravenous nutrition. Long-term management consists of carbohydrate restriction and suppression of the intestinal flora with non-absorbable antibiotics.

While not a neurological complication of bariatric surgery, per se, Javorsky and colleagues noted that individuals with unrecognized Cushing's syndrome may undergo surgery because of obesity and comorbidities such as diabetes and hypertension [59••]. They assembled a cohort of 16 patients with Cushing's syndrome diagnosed after bariatric surgery, and 15 of these had a pituitary adenoma and one had an adrenal tumor. Although their numbers are small, only 17 % of their patients had resolution of hypertension or diabetes mellitus after bariatric surgery, despite a median of 2.5 years follow-up before definitive surgery for Cushing's syndrome. This contrasts with reported resolution of 65.6 % for hypertension and 76.8 % for diabetes mellitus after bariatric surgery indicating that the hypercortisolism associated with Cushing's syndrome attenuates the beneficial metabolic effects usually experienced by bariatric surgery patients. This suggests that more rigorous consideration of Cushing's syndrome should be done prior to bariatric surgery and that patients who do

not accrue the usual benefits should be also be considered candidates for an endocrinological evaluation.

Pregnant and postpartum women with a history of bariatric surgery are potentially at risk of micronutrient deficiencies resulting from the combination of physiologic alterations of pregnancy and iatrogenic postoperative alterations in the absorption and metabolism micronutrients [60, 61]. There have been a very small number of studies and case reports raising the possibility that these patients' offspring may be at risk for neural tube defects, developmental impairment, germinal matrix hemorrhage, or retinal damage. A very carefully performed systemic analysis of these papers concludes that the evidence for neonatal harm is weak and that more definitive studies are required [62].

Conclusions

Bariatric surgery is an effective means of treating obesity and has even been proposed as "the cure for the obesity epidemic." It is essential that the uncommon but serious and potentially reversible neurological complications of bariatric surgery be promptly considered, recognized, and managed. In particular, the rapidly progressive neurological emergencies rhabdomyolysis, Wernicke's encephalopathy, and GBS must be entertained in the perioperative and early operative period.

Consideration starts with a careful medical history including inquiry about previous surgery. Some patients may not recognize bariatric surgery as "real" surgery but rather a cosmetic procedure, so it may be helpful to ask patients and their families if they have ever been treated for obesity or had anesthesia. As always, reliance on the history taken by others is fraught with the perils of incompleteness or inaccuracy. Consideration and recognition require the realization that the neurological complications of bariatric surgery can occur immediately, be delayed weeks to months, or even occur years to decades after the surgery. Temporal proximity is not a requirement, and the neurological differential diagnosis is driven in part by temporal distance from the procedure. Finally, recognition leads to therapeutic intervention which commonly involves rapid repletion of micronutrients. It is possible that the patient may have multiple micronutrient deficiencies, so while definitive restoration of the symptomatic deficiency is essential, repletion of other potential deficiencies should be done.

The focus of this review has been neurological complications of bariatric surgery, but it must be noted that micronutrient deficiency can result from other conditions that impair gastrointestinal function. Of note, one of the first three patients described by Wernicke had thiamine deficiency due to caustic esophageal damage; therefore, the lessons learned from bariatric surgery are generalizable to other conditions leading to micronutrient malabsorption [29••].

Compliance with Ethical Standards

Conflict of Interest Jerry Clay Goodman declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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