

The Neurological Complications of Bariatric Surgery

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Weight is a national obsession. In 1999, Americans spent more than \$300 million on prescription medications for obesity,¹ and 2.5% of the adult population reported using such preparations at the end of the 20th century.² By some estimates, the total cost of obesity in the United States is \$1000 billion annually.³ Obesity is now officially recognized by the surgeon general of the United States as a significant health risk factor. Obesity increases the risk for numerous medical illnesses, among them, diabetes mellitus, hypercholesterolemia, hypertension and other cardiovascular disorders, pulmonary disease, osteoarthritis, and some forms of cancer.^{4,5} Obesity also increases the risk of death from all causes; it is estimated that 300 000 adults in the United States die of obesity-related causes annually.⁶ Obesity currently ranks as the seventh leading cause of death in the United States.³ The problem of obesity is a global phenomenon consequent to the ready availability of food with high caloric content and the reduction of daily energy expenditure. While the percentage of overweight adults in most western European countries has not surpassed that of the United States, their overweight population is increasing rapidly. The percentage of obese children in many of these countries, such as England, is growing rapidly⁷ and has outstripped the percentage of obese children in the United States. In major population centers of developing countries, obesity is also seen with increasing frequency.

For medical purposes, obesity is defined by body mass index (BMI), which is derived by dividing an individual's weight in kilograms by the square of their height in meters. Normal weight is defined as a BMI of 18.5 to 24.9.⁸ A BMI exceeding this value is regarded as overweight. A BMI of 30 or greater is considered obese; a BMI of 40 or greater, morbidly obese.⁸ Prevalence data from the Centers for Disease Control and Prevention indicate that during 1999 to 2000, 64.5% of US adults were overweight, 30.5% were obese, and 4.7% were morbidly obese.⁹ Despite the billions of dollars spent on diets, dietary supplements, exercise programs, and other nonsurgical modalities for weight reduction, long-term success rates have been

quite variable. Increasing numbers of persons are, therefore, resorting to bariatric surgery for control of weight problems. The increasing popularity of these procedures may, in part, be attributable to media attention provided by television personalities who have resorted to surgery for their own weight reduction. In 1990, 2.7 bariatric surgeries were performed for every 100 000 persons; by 1997, that number had increased to 6.3 per 100 000 persons.¹⁰

BARIATRIC SURGERY

National guidelines reserve bariatric surgery for individuals who have failed attempts of nonsurgical weight loss and have a BMI greater than or equal to 35 with an obesity-related comorbidity or a BMI greater than 40 with or without a comor-

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bidity.¹¹ Several different surgical procedures have been employed for achieving weight reduction.¹² Gastroplasties rely on the mechanical restriction of food passage through the stomach, whereas gastric bypass surgery results in weight loss by a more physiological mechanism.¹³ Gastric bypass, typically the Roux-en-Y gastric bypass procedure, is now performed more commonly than gastroplasties.¹⁰ Many of these surgical procedures can be performed laparoscopically.¹⁴⁻¹⁶ Average excess weight loss following laparoscopic Roux-en-Y gastric bypass surgery has been reported to approach 70% at 12 months¹⁵ and 83% at 24 months.¹⁶ Although success rates have been variable, persistent, long-term weight loss at 10 or more years following these procedures has been documented.^{17,18} Studies of cost-effectiveness support the value of bariatric surgery.^{19,20}

The complications of bariatric surgery are not insignificant and are, in part, related to the problems inherent in operating on the obese person. In one series,¹⁶ major complications occurred in 3.3% of patients, and the in-hospital mortality was 0.4%. Reoperation may be necessitated by bleeding, abscess, and wound dehiscence. A variety of late complications are recognized, generally, the consequence of nutritional deficiency. Mineral deficiencies include iron, calcium, phosphate, and magnesium, and vitamin deficiencies include folate and vitamins B₁, B₁₂, D, and E. Iron, folate, and vitamin B₁₂ deficiencies have been reported to be the most common nutritional deficiencies observed following gastric bypass surgery.^{21,22}

NEUROLOGICAL COMPLICATIONS OF BARIATRIC SURGERY

A broad spectrum of neurological complications has been reported to occur in association with bariatric surgery, including the following.

Neurological Complications of Bariatric Surgery

- Encephalopathy
- Behavioral abnormalities
- Seizures
- Cranial nerve palsies
- Ataxia
- Myelopathy
- Plexopathies
- Peripheral neuropathy
- Mononeuropathies
 - Carpal tunnel syndrome
 - Meralgia paresthetica
- Compartment syndromes
- Myopathy
- Myotonia

No part of the neuraxis is exempt from these complications. To date, all studies addressing the neurological complications occurring in the setting of bariatric surgery have been retrospective in nature. In 1987, Abarbanel and colleagues²³ reported that 23 (4.6%) of 500 patients undergoing bariatric surgery experienced neurological complications. Of the 500 patients, 457 underwent a Roux-en-Y procedure, and 43 underwent gastroplasty.²³ The neurological complications became manifest 3 to 20 months after surgery, and all affected patients experienced protracted vomiting, a symptom that may

occur in up to one third of all patients undergoing gastric bypass procedures.²² The constellation of neurological complications included chronic and subacute peripheral neuropathy (52%), acute peripheral neuropathy (4%), burning feet (9%), meralgia paresthetica (9%), myotonic syndrome (4%), posterolateral myelopathy (9%), and Wernicke encephalopathy (9%).²³ In this series, individuals with burning feet and Wernicke encephalopathy responded to thiamine administration.²³

In a retrospective review of 556 patients undergoing bariatric surgery at the Mayo Clinic (Rochester, Minn) from 1980 through 2003, Thaisetthawatkul²⁴ observed 48 patients (8.6%) with complications affecting the peripheral nervous system. Of these 48 patients, 23 (48%) developed mononeuropathies with carpal tunnel syndrome, the most common adverse effect accounting for 74% of the total.²⁴ Peripheral neuropathies were observed in 20 (42%), plexopathies in 4 (17%), and myopathy in 1 (4%).²⁴ Perhaps the most debilitating of the neurological consequences of bariatric surgery are those occurring from vitamin deficiency, particularly vitamins B₁ and B₁₂, as these may result in permanent neurological disability.

VITAMIN B₁ DEFICIENCY

Vitamin B₁ deficiency alters mitochondrial function, impairs oxidative metabolism, and causes selective neuronal death by diminishing vitamin B₁-dependent enzymes.²⁵ A deficiency of vitamin B₁ results in peripheral neuropathy, ophthalmoplegia and nystagmus, ataxia, and encephalopathy, and may lead to permanent impairment of recent memory. This constellation is referred to as Wernicke encephalopathy. The diagnostic criteria for Wernicke encephalopathy require 2 of the following 4 features: (1) dietary deficiency, (2) oculomotor abnormality, (3) cerebellar dysfunction, and (4) confusion or mild memory impairment. These criteria have a very high interrater reliability for the diagnosis.²⁶ This condition is most commonly described in the setting of alcohol abuse when the individual has had insufficient dietary intake of vitamin B₁ over a long period. However, it is important to recall that 1 of the 3 cases in the initial description in 1881 by Carl Wernicke was the consequence of esophageal damage from sulfuric acid ingestion with associated refractory emesis.²⁷ The mechanism by which bariatric surgery leads to vitamin B₁ deficiency, whether resulting in an encephalopathy or peripheral neuropathy, is almost certainly inadequate vitamin repletion together with persistent, intractable vomiting.

In 1977, Printen and Mason²⁸ described 4 patients who developed peripheral neuropathy and protracted emesis following gastric operations for obesity. In this and other early reports of the neurological complications occurring with gastric bypass procedures, the specific cause of the neurological disorder remained uncertain. In 1981, Ayub and colleagues²⁹ described confusion, slurred speech, and unsteadiness in 7 of 110 patients undergoing bariatric surgery. The authors listed several potential causes, including nutritional disturbances, metabolic abnormalities, medication adverse effects, lactic acidosis, or potential toxic bowel product attributable to

a microorganism.²⁹ Some, if not all, of these cases were likely due to vitamin B₁ deficiency. Shortly after this article was published, several reports linked the appearance of Wernicke encephalopathy with surgery for morbid obesity.³⁰⁻³⁵ As late as 2000, some authors considered Wernicke encephalopathy a very rare complication of gastric surgery for morbid obesity³⁶; however, there are at least 30 cases in the world literature. This complication is almost always associated with severe intractable vomiting. It may be seen relatively soon after surgery, usually within 8 to 15 weeks,³⁷ but occasionally as early as 6 weeks.³⁸ An increased susceptibility to Wernicke encephalopathy in women has been suggested.³⁷

Peripheral neuropathy often, but not invariably, accompanies Wernicke encephalopathy following surgery for morbid obesity. Peripheral neuropathy with or without Wernicke encephalopathy that is attributable to vitamin B₁ deficiency and has occurred following surgery for morbid obesity has been referred to as *bariatric beriberi*.³⁹ It seems to be more common than encephalopathy and may also occur within 6 weeks of surgery,⁴⁰ although intervals exceeding 3 years have been reported.⁴¹ The neuropathy predominantly affects the lower limbs and is both sensory and motor, with variable involvement of each. While it may progress for years when untreated, rapid progression over intervals as short as 3 days may mimic Guillain-Barré syndrome.^{41,42} Electrophysiological studies reveal that this neuropathy is axonal in nature, with markedly reduced amplitudes of compound motor action potential and sensory nerve action potentials, especially in the lower extremities.⁴¹

Vitamin B₁ deficiency can be confirmed by assessing the vitamin B₁ pyrophosphate effect in erythrocyte transketolase studies.⁴³ The clinical constellation coupled with the response to administration of parenteral thiamine, especially with the features of the encephalopathy, may prove sufficiently diagnostic. Additionally, magnetic resonance imaging of the brain may show characteristic abnormalities, in particular, hyperintense signal abnormalities on T2-weighted images in the dorsomedial thalamic nuclei, periaqueductal gray matter, and mammillary bodies.^{44,45} Both the encephalopathy and peripheral neuropathy of vitamin B₁ deficiency may occur despite oral supplementation with thiamine, because emesis may preclude effective absorption. Substantial functional recovery typically occurs within 3 to 6 months of the initiation of therapy⁴¹; however, neurological recovery may be incomplete,⁴⁶ particularly if the nature of the disorder is not recognized promptly. Physical therapy for the peripheral neuropathy is also recommended.⁴⁰

OTHER NUTRITIONAL AND METABOLIC DISORDERS

Absorption of vitamin B₁₂ is complex and requires the presence of intrinsic factor derived from gastric parietal cells, acid gastric pH, and absorption in the ileum. Bariatric surgery may interfere with several of these mechanisms. As liver stores of vitamin B₁₂ are sufficient to allow for years of dietary insufficiency, these features may not appear for long periods of time. A low serum vitamin B₁₂ level has been observed in as many as 70% of pa-

tients undergoing gastric bypass surgery, and vitamin B₁₂ deficiency has been observed in more than 30%.⁴⁷

The prototypical neurological disorder occurring with vitamin B₁₂ deficiency is subacute combined degeneration, in which the peripheral nerves and posterior columns of the spinal cord are chiefly affected. Numerous neurologic symptoms and signs have been associated with vitamin B₁₂ deficiency, including paresthesias, loss of cutaneous sensation, weakness, decreased reflexes, spasticity, ataxia, incontinence, loss of vision, dementia, psychoses, and altered mood.⁴⁸ Subacute combined degeneration has been reported after partial gastrectomy^{49,50}; however, the infrequency of disorders related to vitamin B₁₂ deficiency has suggested to some investigators that vitamin B₁₂ deficiency is seldom clinically relevant in the post-gastric bypass patient.⁵¹

Low plasma folate levels are seen in up to 42% of persons undergoing gastric bypass surgery and followed up for 3 years.⁵² Folate deficiency with an attendant peripheral neuropathy would not be unexpected. However, the literature does not suggest that it is a common problem, and some investigators argue that it is not clinically relevant.⁵¹ As with vitamin B₁₂, oral folic acid supplementation seems effective in maintaining levels within the reference range.⁵³

Niacin deficiency and pellagra has occurred after gastroplasty.⁵⁴ This syndrome is characterized by symmetric rash on sun-exposed areas with hyperkeratosis, hyperpigmentation, and desquamation. Glossitis, diarrhea, fatigue, hallucinations, and encephalopathy are also features of the disorder.

Symptomatic hypocalcemia secondary to vitamin D deficiency after gastric bypass has been described.⁵⁵ Marinella⁵⁵ reported a patient who developed carpopedal spasms, intermittent facial twitching, and ophthalmoplegia in association with hypocalcemia years after undergoing gastric bypass surgery who responded to calcium repletion. In addition to vitamin D, patients undergoing gastric bypass surgery may also be at risk of depletion of another fat-soluble vitamin, vitamin E,⁵⁶ which is also associated with neurological manifestations.⁵⁷

A myelopathy that is clinically indistinguishable from that occurring with vitamin B₁₂ deficiency may accompany copper deficiency occurring as a consequence of bariatric surgery.⁵⁸ Kumar and colleagues⁵⁸ described a 49-year-old woman with a spastic ataxic gait, symmetrically brisk lower extremity reflexes, and loss of vibratory perception to the ankles and of pinprick and touch distally, developing 24 years after intestinal bypass surgery for obesity. Vitamin B₁₂ levels were normal, whereas, serum copper and ceruloplasmin levels were low. Clinical improvement followed the intravenous administration of copper as cupric sulfate.⁵⁸

Neurological disorders consequent to rapid fat metabolism or the result of multiple nutritional and metabolic factors have been reported but remain unproved. Feit and colleagues⁵⁹ described 2 patients who developed a severe polyneuropathy chiefly affecting position sense associated with ataxia and pseudochorea within 3 months of gastric partitioning for morbid obesity. In one patient who died, autopsy findings revealed extensive demyelination associated with extensive accumulation of

lipofuscin in anterior horn cells and dorsal root ganglia and of lipid in Schwann cells.⁵⁹ The authors suggested that a toxin from rapid fat metabolism or loss of carnitine were responsible rather than vitamin B₁ or another vitamin deficiency.⁵⁹ Similarly, Paulson and colleagues⁶⁰ described 6 patients with a clinical picture characterized by confusion, abnormal behavior, profound leg weakness, and diminished or absent muscle stretch reflexes, and, in 3, ophthalmoplegia or nystagmus. While vitamin B₁ deficiency was considered in the differential diagnosis, the authors believed that this disorder was likely the consequence of rapid metabolism of fat in obesity.⁶⁰ Other investigators have proposed that some of the neurological complications that follow bariatric surgery, such as peripheral neuropathy or psychosis, arise from multifactorial causes.⁶¹

Recurrent spells of encephalopathy characterized by confusion, behavioral abnormalities, weakness, lethargy, ataxia, and dysarthria occurring with lactic acidosis may occur after jejunoileostomy for morbid obesity.⁶² This same disorder has been described in individuals with short-bowel syndrome.⁶³ It is precipitated by high-carbohydrate diets. The neurologic symptoms occur in association with elevated concentrations of D-lactate in blood, urine, and stool.⁶² The elevated levels of D-lactate are believed to result from fermentation of carbohydrates in the colon or bypassed segment of the small bowel.⁶²

MISCELLANEOUS DISORDERS

Some neurological complications from bariatric surgery that are not ascribable to micronutrient insufficiency, although not exclusive to this setting, remain relatively unique. Unilateral lower compartment syndrome occurring in the immediate postoperative period may be observed.⁶⁴ This syndrome arises from ischemic injury to tissues in the anterior compartment with progressive increase in pressure and ultimately nerve injury. Prompt recognition and fasciotomy are essential for a favorable outcome.⁶⁴

Lumbosacral plexopathy with an asymmetric peripheral neuropathy has been reported following gastric partitioning.⁶⁵ The specific pathogenesis of this disorder has not been addressed,⁶⁵ and whether it is due to a micronutrient deficiency or other cause remains uncertain. Similarly, Thaisetthawatkul²⁴ noted plexopathy in 4 of 556 patients.

A wide variety of musculoskeletal symptoms have been reported to occur in association with gastric bypass.⁶⁶ Ginsberg and colleagues⁶⁶ described 13 patients who developed a constellation of arthritis, polyarthralgias, myalgias, and morning stiffness 3 weeks to 48 months after undergoing jejunoileal shunt surgery. These symptoms tended to be transient in nature, and the demonstration of circulating immune complexes suggested an autoimmune process.⁶⁶

CONCLUSIONS

In summary, neurological complications occurring in the setting of bariatric surgery are not uncommon. These com-

plications have been reported in as many as 5% to 10% of patients undergoing surgery for obesity. Any part of the neuraxis, including brain, cerebellum, spinal cord, peripheral nerve, and muscle, may be involved in these complications. Most of these neurological complications are the consequence of micronutrient deficiency. Physicians need to be particularly alert to Wernicke encephalopathy developing after bariatric surgery, because it is a medical emergency and demands rapid diagnosis and intervention. Physicians caring for patients who have undergone bariatric surgery should be familiar with the constellation of neurological disorders that may occur after surgery.

Accepted for Publication: January 9, 2004.

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