Acute Painful Polyneuropathy After Bariatric Surgery

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ABSTRACT

The prevalence of morbid obesity is increasing worldwide, which over the past decade, has resulted in an increase in the number of obese patients undergoing bariatric surgery. All surgical procedures carry a varying degree of risk; however, bariatric surgery is specifically associated with a number of metabolic, nutritional, and neurological complications. The neurological complications include encephalopathy, peripheral neuropathy, myelopathy, plexopathy, and radiculoneuropathy. This case report involves an 18-year-old female who underwent successful bariatric surgery for morbid obesity. Postoperatively, the patient developed severe painful weakness of the lower limbs and was found to have diffuse sensory motor polyneuropathy. It was concluded that sensory-motor polyneuropathy was the most likely cause of the patient’s weakness. Reporting this case is important as the author believes that the consequences of bariatric surgery may not be well-known to both patients and surgeons.

Key words: Bariatric surgery, morbid obesity, peripheral neuropathy

INTRODUCTION

Obesity is reaching pandemic proportions and, therefore, the frequency of bariatric surgery is increasing. As a result, along with other known complications, neurological complications are also being reported more frequently. Physicians need to be able to recognize these complications as early recognition, together with successful clinical management, can alleviate suffering and prevent long-term disabilities.

It has been reported that neurological complications of bariatric surgery occur in approximately 16% of cases. These complications can be as a result of the failure of mechanical or anti-inflammatory mechanisms, but are primarily due to nutritional deficiencies, with Vitamin B₁₂, thiamine, folate, and Vitamins D and E being the most common deficiencies.[1] Complications include peripheral neuropathy, myelopathy, myotonic syndrome, burning.
feet syndrome, radiculopathies, lumbosacral plexopathy, and Wernicke-Korsakoff’s encephalopathy. However, the most commonly described neurological manifestation is peripheral neuropathy, which is the result of vitamin and micronutrient deficiency.

Reporting this case is important because the author believes that the consequences of bariatric surgery may not be well-known to both patients and surgeons. The objective of this case report is to improve the awareness of both doctors and patients on the adverse consequences of bariatric surgery.

**CASE REPORT**

An eighteen years old, unmarried, Saudi female, underwent bariatric surgery. Three months after the procedure, she complained that she had experienced weakness in both lower limbs for a period of 3 weeks. This was associated with a burning sensation, which had started on the soles of her feet and progressively over a period of 2 weeks had increased until it reached her thighs, making it difficult for her to stand and walk without support. Although this weakness did not affect her upper extremities, she eventually became bed bound and dependent on her family members for her daily needs. She also reported that she had suffered from dizziness and intermittent mild blurring of vision. Initially, she also had suffered from vomiting, which had improved with medication. She did not report a history of headaches, double vision, confusion, seizures, difficulty in swallowing or speaking, sphincter dysfunction, or fever.

Prior to surgery, the morbidly obese patient had a body mass index (BMI) of 41.2 kg/m² and confined to a wheelchair. The patient was living with her parents and had just completed high school and waiting for acceptance to the university. She had been previously diagnosed with hypothyroidism, for which she was taking 100 μg of thyroxine daily. Other than this, her history and family history were unremarkable, with no history of smoking.

She underwent gastric sleeve surgery in July 2014. After surgery, she was primarily on a liquid diet. The patient did not take the prescribed multivitamins and lost a total of 26 kg. At the time of presentation, the patient had normal vital signs. She was conscious with normal language functions and oriented to time, place, and persons, with a mini mental state examination score of 30/30. Her cranial nerves were intact. Concerning her motor system, bulk and tone were normal and symmetrical. However, the lower extremities were tender to touch. Muscle power was 5/5 in upper limbs, proximally as well as distally, and 2/5 in both lower limbs. She had generalized hyporeflexia with bilateral flexor plantar responses. She was found to have hyperesthesia in her soles, feet, and legs. The remainder of her sensory examination was normal. Mild nystagmus was seen in all directions, but finger nose coordination was intact, and there was no dysdiadochokinesia.

Routine tests were conducted, including complete blood count, fasting blood sugar, fasting lipid profile, liver function tests, renal function tests, creatine phosphokinase, and urine analysis, which were all within normal limits. Ca²⁺ was 9.6 mg/dl and PO₄ was ≤4.25 mg/dl. The Vitamin B₁₂ level was 1803 pg/ml, and erythrocyte sedimentation rate was 36 mm in 1 h. Serum thyroid stimulating hormone was 0.161 (low), T₃ was normal, and T₄ was 13.91 (high). Her folic acid level was significantly low at 1.3 ng/ml (3.1-20.5 ng/ml,

![Figure 1](image1.png)

**Figure 1:** Nerve conduction study of left peroneal nerve showing reduced compound motor unit action potentials suggestive of axonal loss

![Figure 2](image2.png)

**Figure 2:** Electromyogram of left tibialis anterior muscle showing denervation
deficient <3.5 ng/ml). The magnetic resonance imaging of the brain and lumbosacral spine was normal. Electromyography and nerve conduction study showed axonal sensory-motor peripheral neuropathy affecting the lower limbs with acute denervation and neurogenic motor unit action potentials. Figure 1 shows the results of the nerve conduction study of the peroneal nerve. Figure 2 reveals that there was significant denervation of the left tibialis anterior muscle.

On the basis of these clinical findings and investigations, she was diagnosed as having sensory-motor polyneuropathy secondary to folate deficiency. Folate deficiency is one of the causes of post bariatric surgery polyneuropathies. She was prescribed multivitamin supplements including folic acid and other micronutrients.

**DISCUSSION**

The frequency of bariatric surgery has increased significantly over the past decade as the prevalence of morbid obesity is increasing worldwide. While this procedure is considered both safe and effective, bariatric surgery presents a distinctive set of risks. Injuries have been reported on all levels of the nervous system, including the central, peripheral, and enteric nervous system. The most likely etiology is nutritional deficiency, which includes Vitamin B<sub>12</sub>, B<sub>1</sub>, E, and D along with copper, zinc, and folate. Common neurological complications are presented in Table 1. Although bariatric surgery to address morbid obesity is one of the most invasive treatments, it is becoming the most commonly practiced and the treatment of choice recommended by doctors and asked for by morbidly obese patients. In some cases, the risk factors are not fully discussed, and sometimes, the benefits and risks are not logically weighed. The rise of bariatric surgery has introduced a variety of previously unrecognized complications, particularly neurological complications.

Bariatric surgery is recommended for patients who have a BMI >40 kg/m<sup>2</sup> with no obesity-related co-morbidities, or for patients with BMI >35 kg/m<sup>2</sup> with co-morbid conditions such as hypertension, obstructive sleep apnea, diabetes mellitus, or dyslipidemia who have failed to lose weight through conservative treatment. The most commonly described complications are peripheral neuropathy and Wernicke-Korsakoff’s encephalopathy. In 1987, Abarbanel et al. reported that 4.6% patients who underwent bariatric surgery experienced some form of neurological complications that became apparent between 3 and 20 months after surgery.

The most immediate neurological complications are the result of involvement of the enteric and peripheral nervous system directly due to the surgical procedure. These complications include peripheral mononeuropathy as meralgia paresthetica, plexopathy, gastroparesis, excessive vagal stimulation, and possibly acquired achalasia. Dr. Singh in a brief review of the literature, conservatively estimated the neurological complication rate to be somewhere between 5 and 10%. In 2004, Thaisetthawatkul et al. published a retrospective case-control study of patients treated at the mayo clinic and found that 16% of patients developed some form of peripheral neuropathies.

Unfortunately for this patient, who was seeking to improve her physical image, the bariatric surgery resulted in a debilitating complication affecting her quality of life with peripheral neuropathy identified as the cause of rapidly progressive painful weakness of the lower limbs. However, Vitamin B<sub>12</sub> deficiency, which is considered to be the most common cause, was not found in this patient. Vitamin B<sub>1</sub> and folate deficiencies are also considered as important causes of painful peripheral neuropathy in early postoperative period (between 3 weeks and 3 months

### Table 1: Specific neurological complications associated with vitamin deficiencies after bariatric surgery

| Vitamin/nutrient | Incidence (%) | Complications |
|------------------|---------------|---------------|
| Vitamin A        | 10            | Xerophthalmia, night blindness, and decreased immunity |
| Vitamin B<sub>1</sub> | Common        | Wernicke’s encephalopathy, Korsakoff syndrome, and beriberi (dry/wet) |
| Vitamin B<sub>12</sub> | <1% symptomatic | Myelopathy, neuropathy, dementia, and depression |
| Folate           | 30-70         | Macrocytic anemia and fatigue may aggravate B<sub>12</sub> deficiency |
| Vitamin D        | 1-10          | Myopathy |
| Vitamin E        | Rare          | Peripheral neuropathy, myopathy |
| Copper           | Rare because undiagnosed | Myelopathy, sensory ataxia |
| Vitamin B<sub>2</sub> | 14            | Burning feet syndrome |
| Vitamin B<sub>6</sub> | 17            | Polyneuropathy |
after the procedure). Folic acid deficiency was the cause identified in our patient.

Different clinical patterns of peripheral neuropathy after bariatric surgery have been described in the literature. A review of case reports identified 60 patients with peripheral neuropathy after bariatric surgery. Among these, the most common presentations were diffuse peripheral neuropathy (67%) which was found in this case, followed by mononeuropathies (30%), mainly meralgia paresthetica. However, peroneal nerve palsy has also been reported after weight loss.

It has been proven that malnutrition can become a significant problem in patients who have undergone bariatric surgery. Surprisingly, obesity itself is a preexisting risk factor for malnutrition, and it is estimated that 20-30% of obese patients have micronutrients deficiency even prior to surgery. After surgery, prolonged vomiting, changes in dietary pattern, bacterial overgrowth, altered flow of the biliopancreatic fluid, and bypass of the duodenal absorptive surface area, all contribute to the risk of complications from malnutrition. Because of the lifelong risk, bariatric surgery patients must continue to take vitamin supplements, undergo routine physician visits, and have scheduled laboratory tests. Patients should be followed by a multidisciplinary team whenever possible.

This patient was started on adequate vitamin and micronutrients supplements and was encouraged to have a proper balanced liquid diet. The endocrinological consultation was sought for deranged thyroid functions, and the dose of thyroxine was readjusted. Gabapentin was given for neuropathic pain, after which her symptoms improved, and her weakness gradually decreased. The patient can now walk independently without any support.

CONCLUSIONS
Bariatric surgery is associated with a number of neurological complications. Patients undergoing bariatric surgery should be counseled about all possible complications associated with the procedure to ensure compliance with dietary advice, particularly that related to nutritional supplements. A preoperative multidisciplinary approach and postoperative regular physician follow-up can reduce the frequency, duration, and intensity of neurological complications in these patients.

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Conflicts of interest
There are no conflicts of interest.

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