Guillain-Barré-like Syndrome after Bariatric Surgery

by NATALIA BODUNOVA, MD; DANIIL DEGTEREV, MD, PhD; NATALIA SUPONEVA, MD, PhD; EVGENIY ZORIN, MD, PhD


ABSTRACT
Macro- and micronutritional deficiencies may occur in some bariatric patients post surgery, which can result in serious neurological complications. In this article, the authors describe the case of a patient who developed polyradiculoneuropathy, a variation of acute inflammatory demyelinating polyneuropathy or Guillain-Barré syndrome, following Roux-en-Y gastric bypass surgery. They discuss the importance of identifying symptoms of polyneuropathy after bariatric surgery and given recommendations for treatment.

KEYWORDS
Bariatric surgery, Roux-en-Y gastric bypass, complications, acute postgastropastic polyradiculoneuropathy, Guillain-Barré-like Syndrome, neurological complications

INTRODUCTION
Bariatric surgery has been shown to be the most effective treatment for obesity in the long term, with the gold standard being the Roux-en-y gastric bypass (RYGB), a malabsorptive procedure that has consistently shown excess bodyweight loss (eBWL) of 70 to 80 percent.1 Macro- and micronutritional deficiencies, however, may occur in some bariatric patients post surgery, which can result in serious neurological complications.1 Acute post-gastric reduction surgery (APGARS) neuropathy is a term used to describe such complications that have been linked to bariatric surgery.2 We present a case of a patient who developed polyradiculoneuropathy (PRN) following RYGB surgery that resembled Guillain-Barré syndrome.

PRN ETIOLOGY
PRN is a condition in which polyneuropathy (PNP) and polyradiculopathy occur together.3,4 Polyneuropathy is a general degeneration of peripheral nerves that spreads toward the center of the body. Polyneuropathy is a serious neurological complication that has been associated with bariatric surgery with a frequency varying between 0.66 and 6.2 percent.5,6 Polyradiculopathy is an inflammatory disorder that affects the spinal nerve roots.

Symptoms of PNP after bariatric surgery include pain in the feet or lower back with ascending paresthesias that may progress to leg weakness associated with ataxia, areflexia, and vibration and proprioceptive sensory loss.7 These symptoms resemble AIDP, the most common form of GBS—a disorder in which the body’s immune system attacks part of the peripheral nervous system—with the exception that patients with PNP tend to have axonal degeneration, rather than demyelination, and normal CSF protein levels.1 In the bariatric patient, the onset of PRN is believed to be associated vitamin B1 deficiency.8 Changes in the immune system after bariatric surgery have also been linked to the development of PRN, and in most cases, incidents of PRN occur 2 to 4 weeks after an episode of acute viral or bacterial infection in posturgery bariatric patients.9

CASE REPORT
A 34-year-old man with morbid obesity (body mass index [BMI] 46.2kg/m2) underwent laparoscopic RYGB in February 2014 (Table 1). The operation and initial postoperative period were uneventful. Within two months postoperatively, his weight loss amounted to 43kg. In early April 2014, the patient reported feeling influenza-like symptoms with a low-grade fever. He was treated with doxycycline. Three weeks later, he developed nausea and vomiting after meals. He was admitted to the hospital, and work-up that included blood and urine analysis revealed no abnormalities. An upper gastrointestinal (GI) study showed gastroenterostomatus patency with good flow of contrast. The patient was given sulpiride 200mg daily, and in a week, symptoms regressed. The patient’s health improved and he was discharged from the hospital after two weeks.

One week after hospital discharge, he developed new onset of numbness in the abdomen and a sense of “cooling” in his feet. He also reported leg weakness with numbness spreading up his legs that required him to use a walking aid. The patient ceased to move independently five days after onset of these neurological symptoms. Two weeks later, he developed numbness in the lower third portion of his forearms on both sides of his body. He was readmitted to the hospital. A neurological examination was performed using the Medical Research Council (MRC) scale for muscle testing strength.10 The MRC for muscle effort grades movement from 0 to 5, with 0 representing “no movement” and 5 representing “muscle contracts against full resistance.” The patient’s MRC results were as follows: lower paraparesis with decreased muscle strength in the legs proximal (2.5/5 MRC) and in the dorsal flexors of the toes of both feet (3/5 MRC). Deep reflexes of the legs were absent, and carpaladral reflexes were decreased. Pain sensitivity in the feet and hands was decreased. Assessment of vibratory sensations was done with a 128 Hz tuning fork with two scaled dampers ranging from 0 to 8 points (reference value ≥ 6 points). Vibration sensitivity was reduced more proximally (spina iliaca anterior superior 2.5/8 points, patella 2/8 points, ankles 5.5/8 points) on both sides of the patient’s body. There was a decrease of painful, tactile sensation on the surface of the abdomen and on the lower third portion of the chest. His hip muscles were nearly symmetrical (left side: 48cm, right side: 47cm). His bladder and bowel functions were good.

Blood and urine tests showed normal levels of iron, calcium, magnesium, potassium, sodium, total protein, total bilirubin, and creatinine levels. Magnetic resonance imaging (MRI) scans of the brain and cervical and upper thoracic levels were without pathological changes. There were increased levels of alamine transaminiade (ALT [206 U/l]), aspartate aminotransferase (AST [86 U/l]), and glucose (7.7mmol/L). Analysis of the cerebrospinal fluid showed the following: white blood cells were 2 cells/mm3 and total protein was 1.2g/L (normal range=0.15–0.45g/L). The level of thiamine was a decreased. Other vitamins (cyanocobalamin, pyrodoxine, folic acid) were being administered.

Assessment of nerve conduction velocity (NCV)10 indicated the motor fibers of tibials and peroneals nerve M-waves were not affected. NCV assessment also showed tachycardia and chrono-dispersion of F-wave latency and the disappearance of afferent component of the H-reflex in the soleus muscles. NCV in upper extremities was normal. A needle electromyography (EMG) of quadriceps femoris and ilolobal muscles on both sides of the patient’s body was performed, which indicated increased insertion activity, which increased the neuropathic recruitment ratio (>10Hz) and the average duration and axial amplitude of polyphasic and pseudopolyphasic motor unit action potentials (MUAPs). Spontaneous denervation activity was the only fasciculation (+++) present.

Based on medical history, clinical, laboratory, electrophysiological data, and the revised diagnostic criteria by Dimachkie and Barohn,11 we diagnosed the patient with acute postgastropastic PRN. We treated the patient with vitamin B complex...

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<th>TIMELINE</th>
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<tr>
<td>February 2014</td>
<td>Laparoscopic Roux-en-Y gastric bypass with uneventful postoperative period</td>
<td></td>
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<td>Uneventful recovery/postoperative period</td>
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<td>April 2014</td>
<td>Influenza-like state</td>
<td>Low-grade fever</td>
<td>Doxycycline</td>
<td>Improved</td>
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<tr>
<td>April/early May 2014</td>
<td>Nausea and vomiting after meals</td>
<td>• Blood and urine analysis: no abnormalities</td>
<td>Sulpride 200mg</td>
<td>Improved</td>
</tr>
<tr>
<td>May 2014</td>
<td>• Lower paraparesis with decreased muscle strength</td>
<td>• Upper GI study: gastroenteroanastomosis with good flow of contrast</td>
<td>• Miligammon 4mL/day</td>
<td>Some improvement</td>
</tr>
<tr>
<td>May/early June 2014</td>
<td>Acute postgastroplastic polyradiculoneuropathy or Guillain-Barré-like syndrome</td>
<td></td>
<td></td>
<td>Symptoms completely resolved within 6 months of treatment</td>
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(miligramma) 4ml per day, thiamine 500mg per day, pyridoxine 3mL per day, and ferrous sulfate 640mg per day. We decided not to treat the patient with plasma exchange and intravenous immunoglobulin (IVIG) due to the disease’s mild course and lack of respiratory disturbances.

After nine days of treatment in the hospital, the patient’s neurological symptoms stabilized. After two weeks of treatment, the patient reported feeling a release from numbness in his hands and pain in his feet. His muscle strength increased to 3.5/5 MRC in the proximal muscles of the legs, and vibration sensation improved in spinal ilica anterior superior and patella (3/8) and ankles (6/8).

We discharged the patient from the hospital after three weeks, and following discharge, the patient reported successfully walking 10 meters without support two weeks after discharge. EMG assessments were repeated five weeks after patient presented with symptoms. NCV was normal. The needle EMG of the quadriceps femoris and tibialis anterior muscles on both sides of the body showed spontaneous denervation activity like fibrillations (++), positive sharp waves (++), and fasciculations (++).

Over the next several months, the patient’s symptoms gradually improved; however, he required the assistance of a walker for six months before his symptoms completely resolved.

**DISCUSSION**

Neurological complications of bariatric interventions are mainly attributed to deficiencies in vitamins A, B1, B2, B6, folic acid, B12, D, E, and the minerals copper, and zinc. Depending on the nature of nutritional deficiency, any part of the nervous system may be involved. Neurological complications may emerge with symptoms of encephalopathy, optic neuritis, myelopathy, radiculopathy, and neuropathy. Disturbances of the peripheral nervous system are observed in 16 percent of bariatric patients during the first year following weight loss surgery. Risk factors for developing peripheral neuropathy (especially polyneuropathy) are relative to absolute weight loss, duration of gastrointestinal disorders, reduced levels of albumin and transferrin after surgery, and postoperative complications requiring hospitalization. Based on the clinical, electrophysiological, and histological findings, there are several forms of peripheral neuropathies after bariatric surgery: isolated small fiber neuropathy, axonal polyneuropathy, and demyelinating PRN. Polyneuropathy within the post-gastropastic syndrome is generally characterized by the development numbness and paresthesia in the distal first and then in the proximal parts of the lower extremities. Occasionally, disturbances in consciousness, anemic syndrome, oculomotor disturbances, psychosomatic disorders, and/or myelopathy may occur. NCV/needle EMG has revealed signs of axonopathy. The appearance of polyneuropathy is predominantly associated with a lack of vitamin B1 (thiamine) and vitamin B12. The vitamin B1 deficiency polyneuropathy is characterized by a combination with Wernicke-Korsakoff encephalopathy. Usually, Wernicke-Korsakoff syndrome is observed 4 to 12 weeks after bariatric surgery. It is characterized by severe cognitive and psychiatric disorders, confusion, and dysfunction of the cranial nerves (especially in the oculomotor group), and ataxia. In a study by Aasheim, the brain MRIs identified lesions characteristic of Wernicke-Korsakoff in 47 percent of cases. Polyneuropathy in patients with B1 deficiency may experience signs of autonomic (orthostatic hypotension) dysfunction, and heart failure. Violations of protein-cell ratio in the analysis of the cerebrospinal fluid is not characteristic of deficiency of B vitamins. It is important to note that the deficiency of other vitamins and minerals can make a significant contribution to the development of postbariatric polyneuropathy as well (Table 2).
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The pathogenesis of postbariatric polyneuropathy is represented by a combination of malnutrition, inflammatory, and immunodeficiency disorders. In our patient, we believe the anatomical positioning of the GI tract after surgery, lack of strict adherence by the patient to the nutritional recommendations post surgery, and the occurrence of an acute infection shortly after surgery likely contributed to the autoimmune aggression against the peripheral nerves, which resulted in PRN. The Guillain-Barré-like syndrome in our patient (with dysfunction of the walk) was not accompanied by a disruption to his vital functions. The symptoms began and increased over a nine-day period and then began to diminish after two weeks, without specific therapy other than nutritional supplementation. The patient was able to walk, with the help of a walking aid, one month after onset.

**CONCLUSION**

We hope this report underscores the importance of thoroughly examining and observing bariatric patients after surgery, particularly during the first year postsurgery. Early identification and intervention of nutritional deficiencies may help to reduce the occurrence of disruptions to the nervous system. We recommend the following daily regimen to all of our patients after bariatric surgery: 100mg of thiamine, 10mg of vitamin B9, 2,000mg of calcium, 3,000IU vitamin D, and 200mg of iron.

**REFERENCES**


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