

# A new etiology for variant of Guillain-Barré syndrome: bariatric surgery

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## ABSTRACT

Bariatric surgery is an effective treatment for obesity. However, the number of acute or chronic neurological complications after bariatric surgery, including Guillain-Barré syndrome, is increasingly reported. We present here two cases which developed acute motor sensory polyneuropathy a couple months after bariatric surgery which rapidly progressed over the following month. Both patients used received parenteral vitamin B complex replacement after surgery. The first case responded well to intravenous immunoglobulin (IVIg) treatment. However, the second case required plasmapheresis and physical rehabilitation for recovery after IVIg treatment. It is thought that minerals, vitamins, and trace element deficiencies can develop after bariatric surgery. These deficiencies may trigger inflammatory and autoimmune mechanisms and cause acute polyneuropathies. In such cases, it should be kept in mind that immune therapies may be beneficial, as well as vitamins.

**Keywords:** Bariatric, complication, neuropathy, malnutrition, Guillain-Barré syndrome

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Obesity is a growing problem and the necessity for bariatric intervention is apparent worldwide [1-3]. Neurological complications are increasingly recognized by the number of bariatric surgeries performed. These complications such as encephalopathy, optic neuropathy, myelopathy, radiculoplexopathy, and neuropathy can involve any level of the central and peripheral nervous system. Neuropathies after bariatric surgery have been reported in 5-16% of various studies [4-6]. Neurological complications may result from immune and inflammatory mechanisms, and some authors claim that the real underlying pathology originates from micro-nutritional deficiencies, such as a lack of vitamins B and E and trace elements [5, 6]. Changes in incretins secreted from intestinal endocrine cells after gastrectomy are also blamed on a pathological mechanism [7, 8]. In addition, there are very few

studies reporting that polyneuropathy develops as a result of an autoimmune reaction after bariatric surgery [9, 10]. Although the underlying mechanisms are still debated, there is, as yet, no general consensus for treatment [10-13]. We discuss the clinical findings and treatment response of two cases developing acute axonal sensory motor neuropathy (AMSAN) which as a variant of Guillain-Barré syndrome (GBS) related bariatric surgery.

## CASE PRESENTATION

### Case 1

A 27-year-old female with 59.7 kg/m<sup>2</sup> body mass index (BMI) (height: 152 cm, weight: 138 kg), underwent a sleeve gastrectomy and was discharged



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on a special diet including oral multivitamins and mineral and protein supplements. She had also been using intramuscular 1000 mcg hydroxocobalamin, 100 mg thiamine and 100 mg pyridoxine per week. Three months after surgery, she developed a burning sensation in her feet, and lower back pain. After this, weakness in both lower limbs increased progressively. Weight loss was almost 20 kg over this duration. Her examination showed normal mental function, intact cranial nerves, distal muscle weakness (a grade of muscle strength of 4/5) with areflexia in lower extremities. Complete blood counts, vitamin B12, folate levels, and metabolic panels, electro-neuromyography (ENMG) and magnetic resonance imaging (MRI) of brain, cervical, thoracic and lumbar regions were found to be normal. Routine cerebrospinal fluid (CSF) analysis including protein, glucose, sodium and potassium levels were normal, in addition no inflammatory cell reaction was found. Her complaints got progressively worse. Over the following month she was unable to get up and walk without full physical assistance. Laboratory studies were repeated. Vasculitis panel, protein electrophoresis, and tumour markers were found negative. The new ENMG showed a severe, diffuse reduction in the amplitudes of the motor and sensory action potentials, particularly in the lower extremities. No conduction block or increase in distal latencies were noted. Needle examination showed fibrillation potentials and positive sharp waves in distal limb muscles. It was thought to be a case of AMSAN. Intravenous immunoglobulin (IVIg) treatment was started 0.4 g/kg/day for 5 consecutive days and 2 g/kg/month for 6 consecutive months. Also, treatment with intramuscular B-complex vitamins were continued. She began walking short distances after six months. Weight loss was 45 kg over the 6 month period.

## Case 2

A 19-year-old male with 50.5 kg/m<sup>2</sup> BMI (height: 1.78 m, weight: 160 kg) was submitted to a sleeve gastrectomy four months previous. A liquid high protein/low carbohydrate diet was instituted and intramuscular vitamin B complex per week was administered after surgery. He had numbness and pain in his legs and hands for 2 weeks. Muscle weakness in the lower extremities started in the feet and

gradually ascended over the following days. A cranial nerve examination was normal. The predominant distal symmetric paresis was observed in the arm and leg muscles (grade of muscle strength of 3/5). Deep tendon reflexes were absent in the lower extremities. Routine blood chemistry analyses, including vitamin B12, folate levels, and vasculitis panel, were normal. An MRI of the brain, cervical and lumbar spine was normal. Routine CSF findings were also normal. ENMG showed a severe, diffuse reduction in the amplitudes of the motor and sensory action potentials, prolongation in distal latencies, and slowing of conduction velocities - particularly in the lower extremities. Needle examination showed acute denervation potentials in the muscles of the lower extremities. AMSAN has been established as a diagnosis. IVIg treatment was started at a dose of 0.4 g/kg/day for 5 consecutive days. However, his muscle strength reduced to a grade of 1/5 in the distal part of the extremities. Plasmapheresis was started 5 times daily on alternate days. In addition, treatment with intramuscular B-complex vitamins was maintained. Six weeks of physiotherapy was administered after plasmapheresis. At his last examination his neurological condition was nearly normal. He had lost 90 kg over the 6 months.

## DISCUSSION

Several bariatric procedures have been applied in the treatment of obesity, such as Roux-en-Y gastric bypass, adjustable gastric banding, and sleeve gastrectomy. Selection criteria for surgery includes BMI  $\geq$  40 kg/m<sup>2</sup> without co-morbidities. If patients have diabetes mellitus, or obstructive sleep apnea, the BMI = 35-40 kg/m<sup>2</sup> can be accepted for surgery [1-3]. However, the incidence of neurological complications is increasing with the widespread use of bariatric surgery, especially in cases of acute axonal polyneuropathies [6].

AMSAN, a variant of GBS, is characterized by acute onset of distal weakness, loss of deep tendon reflexes, sensory symptoms, and is confirmed by ENMG [12-15].

Altered diet, reduced absorption, dysmotility, loss of gastric acid and intrinsic factors are important reasons in developing complications. Among them,

B12 deficiency is the most common. A low B12 level has been reported at a ratio of 70% in these patients [4, 5]. B12 levels were normal in our patients. In addition, deficiencies in B-group vitamins (niacin, pyridoxine and especially thiamine), vitamin E, copper, zinc, selenium, and folic acid are responsible for other nutritional factors in neurologic complications [6]. A deficiency in our evaluation was the lack of analysis of other B group vitamins and trace elements as this is not a part of our work routine. It has been reported that obese patients have pre-existing micronutrient deficiencies and these patients' conditions deteriorate after surgery [6, 7]. Also, a history of repeated attacks of vomiting have been reported as a risk factor for complications in bariatric cases [4]. Our patients had already taken vitamin B supplements and had no history of repeated attacks of vomiting after surgery.

Rapid and excessive weight loss may cause malnutrition related polyneuropathy [12-15]. In the first case, BMI decreased from 59.7 kg/m<sup>2</sup> to 40.3 kg/m<sup>2</sup> in six months. In the second case, it decreased from 50.5 kg/m<sup>2</sup> to 22.1 kg/m<sup>2</sup> in six months.

It is reported that neurological complications generally developed 3-20 months after bariatric surgery. This interval may be related to immunological disturbance caused by deficits of gastric incretins, and microelements deficiencies [11-14]. In our patients, AMSAN developed 3-4 months after surgery.

Diaz *et al.* [10] showed inflammatory changes in nerve biopsy in two cases with acute polyneuropathy after bariatric surgery. They also reported good response to IVIg in these cases. Also, Chang *et al.* [11] reported one patient who after being treated with plasmapheresis no response to IVIg followed. IVIg is a gold standard treatment of autoimmune neuropathies including GBS, chronic inflammatory demyelinating neuropathy and multifocal motor neuropathy. In addition, plasmapheresis may require a second line therapy in cases which are not responding to IVIg [9]. In light of these data, we preferred IVIg as the first line treatment. However, in the second case, plasmapheresis was needed because of no response to IVIg.

Currently, there is confusion in the naming of cases with post-bariatric polyneuropathies, using

terms such as nutritional polyneuropathy, acute/subacute axonal polyneuropathy, and variants/mimics of GBS [12-14]. Using the term 'GBS variants related to malnutrition' could be more suitable in order to have a generally accepted term for these cases. Nutritional support before and after bariatric surgery, and routine biochemical evaluation at frequent intervals are very important to prevent these complications [7-10].

## CONCLUSION

Our cases showed that IVIg should be kept in mind as a reliable treatment option for GBS variants caused by bariatric surgery.

### *Informed consent*

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

### *Conflict of interest*

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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