



A Rare Complication of Bariatric Surgery; Guillain Barre Syndrome

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Abstract

Objectives: Guillain-Barre Syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy characterized by rapidly progressive paresis and sensory disturbances. Laparoscopic Sleeve Gastrectomy (LSG), used of morbid obesity, is the standard surgical treatment method for Bariatric Surgery (BS). Neurological complications of BS are also increasing with growing obesity prevalence. We present a case of developed acute polyneuropathy after BS.

Study Design-Methods: The presentation of GBS is ascending paralysis, reduction/loss of muscle stretch reflexes, and albumino-cytologic dissociation in the CSF. The diagnosis was made using anamnesis, neurological examination, Electroneuromyography (ENMG) and laboratory findings.

Results: Neurological complications of BS are usually related to micronutrients deficiencies secondary to malabsorption after surgery. The complications are seen variable which are count encephalopathy, optic neuropathy, myelopathy, polyradiculoneuropathy, and polyneuropathy.

Conclusions: Among these, GBS appears to be a very rare complication in BS. It is important to consider the diagnosis of peripheral neuropathy, and discrimination of Guillain-Barré syndrome. Because of, their treatments are different.

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Introduction

Obesity is expressed in terms of body mass index (BMI) and is calculated by dividing the weight (kilogram) by square of the length (square meters) (kg/m²), and it's normal range is between 18,5-24,9. BMI between 25-30 is overweight, 30 or more are obese, and when BMI is over 40 morbid obese is considered [1].

Obesity is an increasing medical challenge in both developed and developing countries. The prevalence of obesity in Turkey was found to be in general 35% (in females 44%, in males 27%). In last 12 years, in Turkey obesity increases 34% in women and 107% in men [2].



The American Society for Metabolic and Bariatric Surgery point out that growing obesity prevalence, the number of patients exposing BS and associated neurological complications will also increase. BS has also been reported to reduce morbidity and mortality and obesity-related complications with BMI > 40 and BMI > 35 [3,4]. In LSG, the stomach is resected along the gastroesophageal junction, resulting in a tube shape. It has reducing nutrient absorption but decreases the absorption of vitamins and minerals, is an undesirable effect. This leads to various neurological complications, like as peripheral neuropathy [5].

Peripheral neuropathies may affect up to 16% of operated patients [6]. The most common polyneuropathies are due to deficiencies of vitamins, especially as thiamine (vitamin B1) and cyanoboamin (vitamin B12), and the inflammatory causes of post-BS polyneuropathies, include GBS is rare [4,7]. GBS can appear within a few weeks or months after surgery, and the onset of symptoms are acute. But in the other peripheral neuropathies, onset of symptoms are insidious, the onset time varies from months to years, post-surgery [4]. Peripheral neuropathies usually present years later, progress and must be separated from the polyradiculopathies.

Case Report

A 21-year-old male patient, in the first month after LSG, he had weakness in the lower extremities which was difficulty in climbing stairs within 5-10 days, and gradually realized that these complaints increased.

Medical history of the patient is only obesity and his family medical history revealed no notable characteristics.

In the neurological examination; bilateral upper extremity proximal and distal muscle strength 3/5, bilateral lower extremity proximal and distal muscle strength 2/5. The deep tendon reflexes of the patient were abolished. Routine blood tests and imaging showed no pathological findings. In addition, vitamin B12 and vitamin E, folic acid, selenium and copper levels were normal. When lumbar puncture was performed, CSF protein was detected at the border-line (40 mmHg), infectious and other parameters were normal.

The initial ENMG (Table 1) demonstrated that peroneus and posterior tibialis nerve's Compound Muscle Action Potential (CMAP) amplitudes reduced. Sensory nerve conduction studies and needle ENMG were normal. In an ENMG repeated after 10 days later (Table 1), the CMAP reduction continued, and motor axonal peripheral neuropathy was detected with ENMG findings. With his clinic and ENMG findings the acute GBS was diagnosed to the patient. He received 7 days of intravenous immunoglobulin (IVIg) therapy at a dose of 0.4 mg/kg/day. After the treatment, in his neurological examination showed recovery.

Vitamin replacement (Tiamin, folic acid, selenium, B12, B6 and D) continued to the patient.

Nevertheless, our patient received vitamin supplements, didn't have the characteristic symptoms associated with vitamin deficiencies and these vitamin levels were measured at normal limits.

Table 1: It is a shorttable with the most important electrophysiological features

Motor NerveConductionStudy	First ENMG			Second ENMG (10 dayslater)		
	Ankle	Popliteal		Ankle	Popliteal	
Right PosteriorTibialNerve	Ankle	Popliteal		Ankle	Popliteal	
Latency (ms)	3,95	14,75		5,05	17,05	
NCV (m/s)		41,5			36,7	
Amplitude (µ V)	3350	2870		860	810	
F ResponseLatency (ms)			NP			NP
Right PeronealNerve	Ankle	Head of Fibula	Popliteal	Ankle	Head of Fibula	Popliteal
Latency (ms)	4,1	12,75	14,9	NP		14,5
NCV (m/s)		39,3	36,3			41,5
Amplitude (µ V)	1600	1100	1070			2110
F ResponseLatency (ms)			NP			NP
Right MedianNerve F ResponseLatency (ms)			NP			NP
Right UlnarNerve F ResponseLatency (ms)			25,9			NP

NP: Nopotential (potentialcould not be achieved)

Discussion

BS is also an effective treatment because of its cosmetic and resistant proof results. But neurological complications seen after BS may be irreversible [4,8]. For this reason, detection and early recognition to these neurological complications is important.

Neurological complications are evident at 3–20 months after BS and can involve nerves, spinal cord, and brain [9]. Prolonged vomiting and magnitude of weight loss, may lead to the development of neurological complications [8,9].

Inflammatory infiltrates in nerve biopsies showed that a cachexia-like situation with nutritional deficiencies may stimulate inflammatory changes and immune mechanisms for the neuropathy [4,6].

In general, Wernicke's encephalopathy, polyradiculopathy, mononeuropathy are early complications; optic neuropathy, myelopathy, myopathy and peripheral neuropathy are delayed complications, and can appear after many years [4,7].

The polyneuropathies typically described are length dependent with an axonal pathophysiology. Polyneuropathy due to thiamine deficiency which is called beriberi, is a symmetric sensory motor axonal polyneuropathy in the lower extremities. Symptoms usually develop by stages over weeks to months, sometimes a rapid progression of the neuropathy mimicking GBS has also been described. In their sural nerve biopsies show pronounced degeneration and perivascular inflammation.

In GBS, the known classical treatment should be performed. In the other peripheral neuropathies, parenteral supplementation with thiamine (100 mg daily) partially improves symptoms, physical therapy is also recommended. Polyneuropathy seen after BS is not only due to thiamine deficiency, but may be due to combined deficiencies, as many micronutrients are inadequate [1].

GBS is a rare complication of BS [10]. The relation of GBS development with surgery is not definite [1]. Its pathophysiology has been reported to be, immunogenic as in GBS or may be nutritional deficiencies and multifactorial factors. In an epidemiological study, it is reported that there is a relationship between GBS and any surgery, especially it was more association with bone and digestive organ surgery [11].

In these patients, close follow-up and support of serum levels of micronutrients and minerals after surgery is important. It is noteworthy that presented patient was not related to vitamin deficiency because he was taking vitamin replacement from the very beginning.

Early identification of neurological symptoms may help reduce the occurrence of complications. Intensive nutritional management before and after surgery with close follow up and routine screening of distinguished nutrients at 6 months and then yearly thereafter, in addition to nutritional supplementation may prevent these complications. Treatment requires intravenous vitamin supplementation. Immunomodulation with IVIg should be considered when there is suspicion of an immunologic cause.

The guidelines suggest that patients must take vitamin replacement which include 400 µg of folate (1 to 2 tablets per day), calcium citrate (1200–2000 mg/d), vitamin D (400-800 units per day), elemental iron (40–65 mg/d), vitamin B12 (300–500 µg/d) [8].

So, it is crucial to approach a well-organized multidisciplinary team management, presurgical patient counseling and stating the potential nutritional deficiencies that could lead to serious complications.

Disclosure

We as the authors of this paper, confirm that no disclose any financial and personal relationships with people or organizations that could inappropriately influence our work. We confirm that we have received a signed release form to the patient authorizing the publication of such material.

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