ORIGINAL CONTRIBUTIONS



Neurological Complication After Laparoscopic Sleeve Gastrectomy: Foot Drop

Ozan Şen¹ · Fatih Can Karaca² · Ahmet Türkçapar¹

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Abstract Background The relationship between rapid weight loss and peroneal nerve entrapment neuropathy (PNEN) was shown in various series following bariatric surgery. Thus, we aimed to determine the occurrence of PNEN in our patients who underwent laparoscopic sleeve gastrectomy (LSG) and to reveal the factors contributing to this complication.

Methods We evaluated our series of 635 patients in terms of neurological symptoms following laparoscopic sleeve gastrectomy, retrospectively. We recorded the preoperative data, laboratory vitamin and nutrient levels, weight loss, electromyography (EMG) findings, and treatment modalities of these patients.

Results Seven out of 635 patients developed foot drop as a result of PNEN after bariatric surgery. The mean total weight loss for these patients was 50.6 kg in 6 months, and 63 kg in 12 months. In the laboratory analyses, we did not detect any signs of vitamin deficiency. EMG findings confirmed the diagnosis.

Conclusion We demonstrate that rapid weight loss is correlated with the risk of foot drop incidence as a result of PNEN.

Keywords Bariatric surgery · Weight loss · Drop foot · Neurological complications

Introduction

Laparoscopic sleeve gastrectomy (LSG) is a minimally invasive bariatric surgery procedure aiming to reduce gastric space and appetite. The complications associated with sleeve gastrectomy are rare; however, bariatric surgery procedures in total might cause some complications such as postoperative bleeding, leaks at the stapler line, and stenosis [1]. Besides other or these complications, neurological complications can be observed after bariatric surgery. The incidence of neurological complications after bariatric surgery varies between 5 and 16%, and most of them are due to vitamin deficiency in the majority of patients [2–4]. Entrapment of the peroneal nerve is an important neurological complication causing numbness in the foot, loss of strength, and limited foot ankle dorsoflexion and may be observed in some patients following the bariatric surgery. Sitting cross-legged is one of the activities that most

Fatih Can Karaca drckaraca@yahoo.com

- ¹ Türkçapar Bariatrics, Center for Obesity Surgery, Istanbul, Turkey
- ² Bilgi University, Faculty of Health Sciences, Istanbul, Turkey

patients who lose weight after bariatric surgery like to do since they were not able to prior to surgery. Loss of fat tissue in the proximal head of the fibula in people with rapid weight loss increases the sensitivity of the peroneal nerve to external compression, providing a more superficial track for the nerve, offering a relationship between peroneal nerve entrapment neuropathy (PNEN) and weight loss [5]. Although some patients' symptoms improve with conservative treatment options, surgical decompression of the nerve is often required in cases with foot drop.

The aim of this study was to determine the incidence of PNEN in our patients who underwent LSG and to reveal the factors contributing to this complication.

Materials and methods

We performed LSG to 635 patients between January 2012 and February 2018 in our clinic. The mean body mass index (BMI) of the patients was $43.9 \pm 7.2 \text{ kg/m}^2$. The mean age was 38.1 ± 11.2 years, and 355 out of 635 patients were female (56%). We examined the neurological complications following LSG in our series, retrospectively.

The BMI, operative time, surgical procedure, and equipment used during the surgery were noted. Vitamin and nutrient levels, weight loss, complications, and/or complaints were recorded during the mean follow-up period of 49.5 ± 16.5 months. Daily doses of multivitamin and mineral tablets were prescribed to all patients for 3 years after the LSG procedure (Solgar VM-2000 tablets). Expanded laboratory analysis of vitamins and EMG examination were also performed for the patients with neurological complications.

The study was approved by the institutional review board, and the data were collected in accordance with the principles of the Declaration of Helsinki. The patients were informed about the possible complications and technical details of the surgery, and written informed consent was obtained from each patient.

Results

We observed PNEN in seven of our patients who underwent LSG (1.1%). In laboratory analysis, we did not detect any signs of nutritional and vitamin deficiency or insufficiency in any of the cases. One of these patients with PNEN was a superobese patient with a BMI of 63 kg/m². This patient was re-operated on for leakage due to acute gastric stenosis on postoperative day 7. The patient underwent 5 h of gastric by-pass operation, and acute PNEN developed on the day after the operation. In all of our bariatric surgeries, we prefer the reverse Trendelenburg position where the legs are opened on both sides, and we use surgical legs that can be mounted on the operating table to supply the position (Yellowfins ® Elite Stirrups); thus, the surgeon is positioned between the two legs during surgery. In this patient, we concluded that PNEN developed as a result of compression of the peroneal nerve by the surgical stirrup legs during the long operation period. The diagnosis of PNEN was confirmed by EMG. The patient recovered without sequelae after 2 months with rehabilitation therapy for PNEN.

The other six patients (3 M/3 F) with a mean age of 43 had unilateral PNEN. In three of these patients, foot drop developed suddenly. Conductive blockage of the peroneal nerve was confirmed by EMG (Fig. 1), and surgical nerve decompression treatment was applied to all three patients. The other three of the patients had less severe symptoms. The EMG view of these patients showed mild entrapment neuropathy of the fibular head of the deep peroneal nerve. They received conservative treatment, and their symptoms disappeared completely. The common feature of most patients was that the symptoms became evident on postoperative months 5–7, and all patients lost more than 80% of their excess weight. The mean excess weight loss (EWL) was $60.2 \pm 19.6\%$ within 6 months, and $81.2 \pm 24.8\%$ in 1-year follow-up in our series.

Discussion

With the increasing ratio and variety of bariatric procedures, different complications following these operations are of utmost importance due to the relatively recent history of these interventions. Herein, we present seven cases with PNEN development in our series who underwent LSG.

The ratio of neurological complications after bariatric surgery is determined as being 5–16% [2–4]. Most neurological complications originated from vitamin deficiency and usually manifest as polyneuropathy.

Among the bariatric surgical methods, LSG has become the most commonly used method in recent years [6]. Although studies suggest that LSG causes less vitamin deficiency compared with other malabsorptive methods, vitamin B12 deficiency still can be observed in 20% of the patients during the follow-up period [7, 8]. This may be due to malnutrition due to restricted solid food intake in the early postoperative period, inadequate hydrolyzation of protein in the diet as a result of hypochlorhydria of the stomach, and decreased ratio of binding to intrinsic factor due to fundus resection where the parietal cells are localized.



Fig. 1 EMG result showing the blockage of the peroneal nerve

Habitually crossing one leg over the other is known to be a risk factor for the development of PNEN due to the compression of the nerve for its superficial track as a result of the lost fat pad at the head of the fibula [9]. Therefore, those with accelerated weight loss in a short period have a higher risk for entrapment neuropathies. The relationship between peroneal neuropathy and weight loss is a well-known phenomenon. In their series of 160 patients who were operated on for persisting foot drop, Weyns et al. showed that the etiology underlying the foot drop was weight loss for 48% of the patients [10]. Nine out of 160 patients developed the condition following bariatric surgery. Another critical point in this study is that it is stated that the risk of foot drop is higher in patients with rapid weight loss in a short time. In our study, it was found that all patients lost more than 80% of their excess weight in the first 6 months, except the case with prolonged operation time. The mean EWL was $61.2 \pm 19.6\%$ within 6 months in our series; thus, we consider that the etiology underlying the PNEN in these patients might be the higher EWL ratio. Although the degree of weight loss after bariatric surgery varies between the patients, the weight is fixed at a certain point after postoperative 1-1.5 years. Losing 50-70% of excess weight after 1 year following LSG is an indicator of the success of surgery alone [11, 12]. When we consider the relationship between weight loss after bariatric surgery and PNEN, the time duration of the patient for losing excess weight is essential. In this regard, patients who lose weight faster than expected especially in the first 6 months after surgery should be paid special attention in terms of peroneal entrapment neuropathy and foot drop risk, and patients should be informed in advance about this complication.

In the evaluation of cases with peroneal neuropathy, vitamin and mineral deficiency should be excluded. Neurological symptoms due to vitamin and mineral deficiency are usually observed in the form of polyneuropathy. In our clinical practice, we recommend multivitamin supplementation for all patients after LSG for at least 5 years. Although the symptoms were unilateral in all of our patients, they still were examined for vitamin deficiency, and no deficiency or insufficiency was detected.

Additionally, the perioperative positioning of the patient is also important since these patients have excess weight applying pressure on the nerve during the operative period for hours. With the experience we have gained from this patient, we are much more cautious now about this complication when positioning patients before surgery. Besides instructing the patients to avoid crossing over the legs during the rapid weight loss period, nonsurgical treatment options such as plastic ankle foot orthosis and light braces might be beneficial [13]. EMG is crucial in confirming the diagnosis. Conductive blockage of the peroneal nerve at the head of the fibula is the main criterion for diagnosis. In this respect, it should be noted that, since EMG is not routine practice for patients during the follow-up process following bariatric procedures, many patients with mild concomitant symptoms might be overlooked.

In conclusion, PNEN due to rapid weight loss after bariatric surgery is a rare but important complication. During the postoperative follow-up period, symptoms such as numbness and loss of strength of the foot might not always be a result of nutritional or vitamin deficiency. Patients with rapid weight loss and neuropathy should be examined for nutritional and vitamin deficiency, but PNEN should be kept in mind. Patients should be informed about this complication, and preventive measures should be taken during the follow-up period.

Compliance with Ethical Standards

The study was approved by the institutional review board, and the data were collected in accordance with the principles of the Declaration of Helsinki. The patients were informed about the possible complications and technical details of the surgery, and written informed consent was obtained from each patient.

Conflict of Interest The authors declare that they have no conflict of interest.

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