

# Rare Neurological Complications After Sleeve Gastrectomy

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## Abstract

**Background** Bariatric surgery is considered to be the most effective treatment of morbid obesity and improvement of obesity-related comorbidities, such as type II diabetes. However, both peripheral and central neurological complications can occur after bariatric surgery. Such complications tend to occur more frequently after bypass surgery than after sleeve gastrectomy (SG). The objective of this study was to identify the patients that presented post-operative neurological complications after undergoing SG and describe the incidence, presentation, and management of these complications. **Methods** This was a retrospective study of 592 cases of SG performed between 2009 and 2014 with a special focus on patients who presented neurological complications.

**Results** Of the 592 SG cases, only seven (1.18 %) patients presented neurological complications. All patients had uneventful post-operative course, but all reported feeding difficulties, accompanied by severe dysphagia, and rapid weight loss, with a mean weight loss of 35 kg (30–40 kg) 3 months after SG. All patients were readmitted owing to neurological symptoms that included paresthesia, abolition of deep tendon reflexes of the lower limbs, muscle pain, and motor and sensitive deficits in

some cases. There were two cases of Wernicke's encephalopathy. All patients were treated for neuropathy secondary to vitamin B1 deficiency and had a significant improvement and/or resolution of their symptoms.

**Conclusions** Neurological complications after SG are rare and are often preceded by gastrointestinal symptoms, rapid weight loss, and lack of post-operative vitamin supplementation. Re-hospitalization and multidisciplinary team management are crucial to establish the diagnosis and initiate treatment.

**Keywords** Sleeve gastrectomy · Vitamin B1 deficiency · Peripheral neuropathy · Wernicke's encephalopathy

## Introduction

Morbid obesity has been recognized by the World Health Organization as a systemic disease. If it remains untreated, morbid obesity can trigger the development of numerous comorbidities, including hypertension, diabetes mellitus, obstructive sleep apnea, osteoarthritis, and infertility, as well as increase the risk of developing certain types of cancer [1]. Conservative management, including lifestyle changes, dietitian follow-up, and psychotherapy, is essential for the treatment of this disease. However, patients are unlikely to achieve management goals based on lifestyle changes only [1]. Bariatric restrictive surgeries, such as sleeve gastrectomy (SG) and gastric banding, have been widely used to manage morbid obesity. Although these surgeries are known to cause less metabolic derangements, post-operative food intake difficulties could lead to vitamin deficiency that can potentially lead to severe neurological complications [2–5]. In fact, some previously published reports described cases of peripheral neuropathy and even Wernicke's encephalopathy, which were directly linked to vitamin deficiency after SG. The most common symptoms included paresthesia, motor deficit, ataxia,

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confusion, and in some cases, full-blown Wernicke's encephalopathy. To the best of our knowledge, this is the largest series of neurological complications after SG reported thus far. This study aimed to identify the patients that presented post-operative neurological complications after undergoing SG and describe the incidence, presentation, and management of these complications.

## Materials and Methods

This was a 5-year retrospective review of 592 patients who underwent SG at a specialized bariatric center between 2009 and 2014. The patients were selected to undergo SG if they met the criteria defined by the French National Authority for Health (body mass index (BMI) greater than 40 kg/m<sup>2</sup> or greater than 35 kg/m<sup>2</sup> with associated obesity-related comorbidities) [6]. All patients received both written and oral information regarding the procedure, and all provided informed consent before undergoing the surgical procedure. The following data were collected: general patient demographic characteristics (sex and age), preoperative BMI, post-operative course, BMI on re-hospitalization for neuropathy symptoms, symptoms on presentation (i.e., paresthesia, abolition of deep tendon reflexes, ataxia, and Wernicke's encephalopathy), treatment received, and progression of the neuropathy. Weight loss results were expressed as the change in BMI, percentage of excess weight loss (%EWL), and percentage of excess BMI loss (%EBMIL). Patients who had neurological complications after SG (Neurologic Complications Group) were compared to those who did not present any complications (SG cohort).

## Statistical Analysis

Continuous quantitative variables with a normal distribution are presented as mean  $\pm$  standard deviation. Categorical

variables are expressed as frequencies (%). Differences between groups were evaluated using parametric and nonparametric tests as appropriate ( $\chi^2$  test for categorical variables and analysis of variance with post hoc analysis or Kruskal-Wallis test for quantitative variables). Statistical significance was defined as a *p* value < 0.05. All analyses were performed using JMP 10.0® (SAS Institute Inc, Cary, NC, USA).

## Results

Of the 592 patients who underwent SG at our institute during the study period, we identified only 7 (1.18 %) patients with neurological complications after SG with a mean delay of 4.6  $\pm$  3.4 (range 3–12) months. All patients, but one, were female (six patients, 86 %). They all had an uneventful immediate post-operative course. None of these patients had vitamin deficiencies (vitamins B1, B6, B9, B12, and D) preoperatively. They did not receive vitamin supplementation in the preoperative or immediate post-operative phases. All patients reported difficulties in oral food intake, accompanied by severe dysphagia, gastro-esophageal regurgitation, and vomiting. They all had rapid weight loss within the first few months of the surgery with a mean weight loss of 35.8  $\pm$  11.1 and a percentage of excess weight loss (%EWL) reaching 63.8  $\pm$  6.9 (Table 1). One patient had a chronic fistula at the gastro-esophageal junction, hindering the ability to eat. Consequently, this patient experienced rapid weight loss with a %EWL of 78.6 % in the first post-operative year. When compared with patients who did not have neurological complications, age, sex, height, initial weight and BMI, and excess weight loss were comparable (Table 2). However, a statistically significant difference was found in the %EWL and %BMI loss 3 months after surgery. Patients in the neurological complications group experienced extreme weight loss in a short period of time.

**Table 1** Demographic characteristics and weight loss data of the patients who had neurological complications

	Gender	Age (years)	Height (m)	Initial weight (kg)	Pre-SG BMI (kg/m <sup>2</sup> )	Weight on presentation (kg)	BMI on presentation (kg/m <sup>2</sup> )	% excess BMI loss	Time to presentation (months)
Pt. 1	F	47	1.54	143	60	87	36.6	67	4
Pt. 2	F	20	1.65	120	46.5	88	32	67.4	4
Pt. 3	F	46	1.74	109	36	83	27.5	77.3	4
Pt. 4	F	37	1.6	97	38	67	26	92	2
Pt. 5	F	27	1.68	114	40.5	84	29.7	69.6	2
Pt. 6	M	29	1.87	240	68.6	120	34.3	78.6	12
Pt. 7	F	31	1.58	117	46	76	30.4	74.3	4

BMI body mass index, SG sleeve gastrectomy

**Table 2** Comparison between neurological complications patients and SG cohort

	Neurological complications ( <i>n</i> = 7)	SG cohort ( <i>n</i> = 585)	<i>p</i> value
Baseline			
Age (years)	33.8 ± 10	38.1 ± 11.9	0.38
Sex ratio M/F, <i>n</i> (%)	1 (14.3)/6 (85.7)	107 (18.3)/478 (81.3)	1
Weight (kg)	134.3 ± 48.6	121.6 ± 21.7	0.85
Height (cm)	166.6 ± 11.2	165.9 ± 8.9	0.92
BMI (kg/m <sup>2</sup> )	47.7 ± 12.2	44.1 ± 6.6	0.71
Ideal weight (kg)	62.8 ± 10.1	62.2 ± 8.1	0.92
Excess weight (kg)	71.4 ± 42.3	59.4 ± 18.5	0.78
Postoperative (3 months)			
Weight (kg)	80.8 ± 8	103.2 ± 19.5	0.001
Weight loss (kg)	35.8 ± 11.1	20 ± 15.4	0.0004
% excess weight loss	63.8 ± 6.9	34.7 ± 26.9	0.0002
% excess BMI loss	74 ± 10.3	39.8 ± 30.9	0.0002

Data in the table are presented as mean ± SD, unless otherwise indicated  
*BMI* body mass index, *M* male, *F* female, *SG* sleeve gastrectomy, *SD* standard deviation

**Neurological Symptoms**

Neurological symptoms included paresthesia of upper and lower extremities (LE), dysesthesia, and in some cases abolition of the deep tendon reflexes of the LE (Table 3). Two patients developed symptoms consistent with Wernicke’s encephalopathy (WE). These symptoms were memory loss, generalized fatigue and sensorimotor deficit. After their readmission to the hospital, when these patients were questioned, both noted that they had started presenting symptoms of peripheral neuropathy a few weeks prior to the development of WE symptoms. One of these patients had been examined at the emergency department of another hospital for these symptoms, but no proper investigation and treatment were given. Thus, the symptoms worsened and the condition progressed to WE.

**Workup and Treatment**

All patients underwent an upper gastrointestinal series that showed no functional stenosis. None of the patients had a history of alcohol abuse. All patients underwent vitamin measurements (vitamins B1, B6, B9, B12, and D) at the time of presentation with neurological complications and before beginning vitamin supplementation. However, some of these measurements were performed at an outside laboratory and the results were delayed; thus, we did not wait for these results before initiating the vitamin therapy to avoid worsening of the neurological symptoms and to prevent progression to WE. Only two patients were found to have low serum thiamine (vitamin B1) level and follow-up measurements after treatment showed improvement of these values concomitant with reversal of symptoms (Table 3). All patients were evaluated by a neurologist. All of them underwent an electromyogram,

**Table 3** Vitamin values preoperatively (A), on presentation (B), and 1-week post-treatment (C) for patients with neurological complications after sleeve gastrectomy

	Vitamin B1 (nmol/L)			Vitamin B6 (µg/L)			Vitamin B9 (µg/L)			Vitamin B12 (ng/L)		
	A	B	C	A	B	C	A	B	C	A	B	C
Pt. 1	117	65	105	36.8	4.7	21	11	4.5	6.8	690	363	>2000
Pt. 2	95	78	164	31	7.5	9.1	16	17.8	22	710	613	908
Pt. 3	145.3	74.5	107	26.2	5.8	17.1	9.2	4	6.8	690	640	728
Pt. 4	98.7	81	103	41	11.4	26.8	7.6	3	3.3	889	480	1546
Pt. 5	189.6	88	141.3	28.1	10.1	27.4	11.3	7.1	12.9	567	472	699
Pt. 6	139	64.9	111	41.4	5.2	19.7	15.2	5	12	789	388	690
Pt. 7	176.4	70.8	137.3	43	7.6	29.3	16.2	9.1	13.5	803	762	843

Vitamin B1 normal range, 67–200 nmol/L; vitamin B6 normal range, 5–50 µg/L; vitamin B9 normal range, 3–18 µg/L; vitamin B12 normal range, 200–950 ng/L

which was consistent with sensitive axonal peripheral polyneuropathy. The two patients who had symptoms of WE underwent an MRI; however, no radiologic findings of WE were observed.

Treatment consisted of aggressive multivitamin and mineral replacement therapy. This was achieved by means of intravenous administration of 1 gr of vitamin B1 (Bevitine, Laboratoires DB PHARMA, La Varenne-St-Hilaire), 500 mg of vitamin B6, 1000 µg of vitamin B12, 1 ampoule of a multivitamin preparation (Cernevit®: 3500 IU retinol, 5.5 µg cholecalciferol, 10.2 mg alpha-tocopherol, 125 mg ascorbic acid, 3.51 mg vitamin B1, 4.14 mg riboflavin, 4.53 mg pyridoxine, 6 µg cyanocobalamin, 414 µg folic acid, 17.25 mg pantothenic acid, 69 µg biotin, 46 mg nicotinamide; Clintec-Baxter, Paris, France), and 1 ampoule of Decan® (10 mg zinc gluconate, 0.48 mg copper gluconate, 15 µg chromium chloride, 25 µg ammonium molybdate, 0.2 mg manganese gluconate, 1 mg ferrous gluconate dihydrate, 1.47 µg cobalt gluconate, 70 µg sodium selenite, 1.45 mg sodium fluoride, and 1.53 µg sodium iodide; Aguetan, Lyon, France). Patients were maintained on this regimen throughout all their hospitalization period and were transitioned to intramuscular injections of vitamin B1 and B12. Additionally, oral multivitamin supplementation was prescribed once discharged from the hospital for a period of at least 6 months. To cover their daily caloric needs during the hospitalization, patients were fed by peripheral parental nutrition. In the case of the patient with the chronic gastric fistula, the patient was fed by enteral nutrition through a feeding naso-jejunosomy.

## Outcomes

All patients responded to the aggressive vitamin therapy with resolution of the majority of their symptoms within a few months of therapy. Residual sequelae are detailed in Table 4.

## Conclusions

Neurological complications of bariatric surgery are rare. The largest longitudinal surgical series reported that only 1.3 % (133/9996) of patients experienced such complications [3]. Abarbanel et al. [2] described that neurological complications developed 3 to 20 months after gastric restrictive surgery in 4.6 % of their 500 patients. Their symptoms included chronic or subacute symmetric polyneuropathy, acute severe polyneuropathy, burning feet syndrome, myotonic syndrome, posterolateral myelopathy, and Wernicke-Korsakoff encephalopathy. The highest incidence, however, was recorded in a controlled retrospective study from the Mayo Clinic that described 16 % of peripheral neuropathy among patient undergoing bariatric surgery [4]. Most of the other data available in the literature about neurological complications after bariatric surgery in general and SG particularly consist of case reports. Our study was tailored to help identify the incidence of neurological complications among patients undergoing SG, to describe the presentation and to propose a strategy to manage and prevent such complications in the future. Similarly to the abovementioned studies, the incidence of neurological complications in our study was found to be 1.18 % after a mean delay of  $4.6 \pm 3.4$  (range 3–12) months.

The predominant clinical features preceding the development of these complications were gastrointestinal symptoms that included dysphagia, gastro-esophageal reflux and recurrent vomiting, all leading to a rapid and excessive weight loss within the first 3 months after SG. In fact in our series, weight loss and %EWL values at 3 months were significantly higher in the group that developed neurological complications when compared with the group without symptoms. The recurrent vomiting episodes and the excessive weight loss are perhaps the main cause of the depletion of the vitamin reserves. Therefore, these should be considered as potential risk factors

**Table 4** Symptoms and residual sequelae of patients who developed neurological complications after sleeve gastrectomy

	Sensitive	Motor	Central	Sequelae
Pt. 1	LE paresthesia	LE motor deficit	Ataxia	Paresthesia of feet
	LE dysesthesia	Abolition of LE DTR	Wernicke's	
Pt. 2	LE paresthesia	None	None	Paresthesia of feet
	LE hypoesthesia			
Pt. 3	LE paresthesia	LE motor deficit	None	Paresthesia of feet
		Abolition of LE DTR		
Pt. 4	Abdomen hypoesthesia	None	None	None
	LE paresthesia			
Pt. 5	LE paresthesia	None	None	None
Pt. 6	UE paresthesia	UE motor deficit	Ataxia	UE Paresthesia
	LE paresthesia	LE motor deficit	Wernicke's	LE motor deficit
	LE dysesthesia	Abolition of LE DTR		Abolition of LE DTR
Pt. 7	LE paresthesia	None	None	Paresthesia of feet

LE lower extremity, UE upper extremity, DTR deep tendon reflexes

for the development of neurological complications in such patients [5]. These risk factors were also described in the studies of Thaisetthawatkul et al. They found that the rate and absolute amount of weight loss, increased serum-glycosylated hemoglobin and triglycerides, prolonged hospitalization, post-operative gastrointestinal symptoms, nausea and vomiting, and malnutrition all contributed to the development of neurological complications in patients who underwent bariatric surgery. The authors concluded that a systematic multidisciplinary approach to intensive nutritional management pre- and post-operatively with frequent follow-up greatly decreased the development of peripheral neuropathy in patients that underwent bariatric surgery [4, 7].

Notably, inadequate vitamin supplementation, specifically with vitamin B1, in the presence of gastrointestinal symptoms was incriminated in the development of neurological complications [5]. Reports show that 5 to 30 % of patients undergoing bariatric surgery had subnormal serum vitamin B1 levels prior to the treatment [8, 9]. Additionally, low serum vitamin B1 levels are detected in up to 25 % of the patients within 2 years and in up to 30 % within 5 years after bariatric surgery [10, 11]. It is possible that the rapid onset of vitamin B1 deficiency in these patients occurs because vitamin B1 cannot be produced by humans or other animals. Vitamin B1 is obtained from exogenous sources, namely vegetables, whole grain, and supplemented foods. The human storage of vitamin B1 is about 25 to 30 mg; thus, vitamin B1 can be depleted within 2 to 3 weeks [12]. Patients with depleted vitamin B1 reserves are not only prone to develop symptoms of peripheral neuropathy but can progress to WE. This progression was observed in two of our patients and was supported by several case reports in the literature [5, 13–15]. In our series, none of the patients had preoperative vitamin B1 deficiency, and none of them were discharged with vitamin supplements after SG. This is probably attributable to the fact that unlike the American Society of Metabolic and Bariatric Surgeons guidelines [16], the French National Authority for Health recommendations does not advocate for any systematic supplementation after SG [6]. A relatively new French report has addressed this issue and emphasized the need to change the HAS recommendations in this regard [17].

Other mechanisms have been implicated in the development of neurological complications after bariatric surgery. These include mechanical compression and entrapment that lead to mononeuropathies. It seems that the rapid weight loss may increase the susceptibility for nerve compression secondary to loss of subcutaneous tissue and protective fat pads as well as other structural changes [3]. Others researchers suggested that inflammatory and immune mechanisms may also be involved in the development of these neurological complications [3, 4].

In our series, we attribute all the neurological complications to vitamin storage depletion resulting from a prolonged period of gastrointestinal symptoms (nausea, vomiting, and dysphagia) and rapid weight loss. All our patients responded to empiric

intravenous vitamin and mineral replacement therapy, evidenced by clear improvement and regression of their neurological symptoms as described elsewhere in the literature [3–5, 7].

Our study has its limitations, which are mostly owed to its retrospective nature. The serum vitamin levels of most patients that developed neurological complications on presentation were not indicative of deficiency. However this does not alter our conclusions. In fact, Abarbanel et al. reported that the occurrence of these complications seems to be linked to nutritional causes, although no such deficiencies were confirmed [2]. Another limitation is the fact that this study is based on a small cohort of patients because of the rarity of the findings described. Nevertheless, to date, this series is the first to describe the incidence, presentation, and management of neurological complications after SG.

Neurological complications after SG are rare and are often preceded by prolonged gastrointestinal symptoms, rapid and excessive weight loss, and a lack of vitamin supplementation and dietary follow-up. Re-hospitalization and multidisciplinary team management (surgery, endocrinology, neurology, and physiotherapy) are crucial to establish the diagnosis, initiate treatment, and halt the progression of neurological complications after SG.

#### Compliance with Ethical Standards

**Conflict of Interest** The authors declare that they have no competing interests.

**Statement of Informed Consent** For this type of study, formal consent is not required.

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