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Caso clínico

Peroneal palsy after bariatric surgery; is nerve decompresion always necessary?

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Abstract

We present two patients who underwent successful bariatric surgery and developed peroneal nerve palsy six months after the procedure. This is an unusual complication which determines a significant functional limitation, mainly because of foot drop, and its presence may be a hallmark of excessive and rapid weight loss. We discuss possible pathogenic mechanisms and therapeutic options, and we emphasize the important role of an adequate nutritional management, in order to avoid the need for a surgical nerve decompression.

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Key words: *Peroneal neuropathy. Bariatric surgery. Neurological complications. Foot drop.*

PARÁLISIS DEL NERVIO PERONEO TRAS CIRUGÍA BARIÁTRICA; ¿LA DESCOMPRESIÓN QUIRÚRGICA ES SIEMPRE NECESARIA?

Resumen

Se presentan dos pacientes sometidos a cirugía bariátrica que desarrollaron una neuropatía del nervio peroneo común seis meses después de la intervención. Se trata de una complicación poco frecuente que supone una importante limitación funcional, y su aparición puede ser indicativa de una pérdida de peso demasiado rápida y excesiva. Se discute la patogenia y las posibles alternativas terapéuticas, y se destaca el papel esencial del correcto manejo nutricional, con el fin de evitar la necesidad de descompresión quirúrgica nerviosa.

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Palabras clave: *Parálisis peronea. Mononeuropatía peroneo. Cirugía bariátrica. Complicaciones neurológicas. Pie caído.*

Abreviaturas

"BS": bariatric surgery; "PN": peroneal neuropathy. "BMI": body mass index; "%WL": percentage weight loss; "%EWL": percentage excess weight loss.

Introduction

The rise in the number of patients undergoing bariatric surgery (BS) has led to an increase in the incidence and recognition of medical complications associated to weight loss and nutrient deficiency.[1] Several reports have described alterations of the nervous system in 1.3-16% of cases, including both central and peripheral involvement, and, in the majority of cases, vitamin deficiency was the main causal mechanism.[2-5]

Among the complications affecting the peripheral nervous system after BS, peroneal neuropathy (PN) is unusual.[2] We present two cases of PN, which developed shortly after successful BS. We suggest a causal association and propose treatment alternatives.

Case reports

Case 1

A 30 year-old woman, with a long-term history of obesity without associated comorbidities, underwent vertical gastric gastroplasty with a body mass index (BMI) of 43.6 kg/m^2. She then followed a 4-week period of hyperproteic oral fluids and two months later, correct tolerance to solid diet was reached. Vitamin and

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Table I
Weight, BMI, %WL, %EWL and %EBMI at different times during the first postoperative year of case 1 and case 2 and a reference case-series ("ref")

	Time (months)						
	0	*1*	*3*	*6*	*ref*	*12*	*ref*
Case 1 ♀							
Weight (kg)	123.1	112.1	95.2	78.8		70.3	
BMI (kg/m^2)	43.6	39.8	33.8	27.9		24.9	
%WL		8.94	22.7	36.0	25.7	42.9	31.0
%EWL		18	45	71	47.7	85	59.2
%EBMI		21	53	84	55.0	100	69.8
Case 2 ♂							
Weight (kg)	130.5	116.3	106.2	84.3		75.1	
BMI (kg/m^2)	41.7	36.7	33.5	26.6		23.7	
%WL		20.9	28.6	35.4	29.3	42.5	37.2
%EWL		24	41	78	59.2	93	71.4
%EBMI		30	49	90	60.0	108	77.0

BMI: Body mass index; %WL: Percentage weight loss; %EWL: Percentage excess weight loss.
Ref: Reference mean values, for age- and sex- matched controls from the cohort of BS performed in our center.

mineral supplements were introduced starting at the immediate postoperative period. Weight and BMI during the first postoperative months decreased quickly (table I) due to body-image dissatisfaction and obsessive behavior, which led her to reduction in food intake below advised recommendations, to only 600-800 kcal/day. Psychiatric evaluation evidenced an eating disorder not otherwise specified and subclinical anorexia nervosa, restrictive type. She was started on sertraline 100 mg/day and fluoxetine 5 mg/day. At 6-months' follow-up, she presented with right lower-limb paresthesias, foot drop, and frequent stumbling. Physical examination revealed hypoesthesia and inadequate extension of the right foot (strength 0/5), but reflexes were maintained. Laboratory data at this time were: total proteins 6.9 g/dL, serum albumin 4.3 g/dL, prealbumin 20.3 mg/dL, serum iron 108 ug/dL, folic acid 3.5 ng/mL (2-20), vitamin B12 220 pg/mL (120-900), zinc 72 ug/dL (60-150), selenium 92 ug/L (60-120), copper 112 ug/dL (70-140), vitamin 25-hydroxi-D 33 ng/ml (30-100), retinol 0.48 mg/L (0.43-0.67), ratio vitamin E/cholesterol 5.92 mg/g (5-12). Electromyogram evidenced focal right peroneal mononeuropathy at the fibular head. Diet was improved and routine oral vitamin and mineral supplementation was maintained. The patient reached 85 kg one year later, which has been her stable weight thereafter, and neurological symptoms disappeared since then.

Case 2

A 45-year-old man, obese since the age of 4, a 15-year history of medication controlled-hypertension and smoking-habit, underwent laparoscopic biliopancreatic diversion with duodenal switch with a BMI of 40.1 kg/m^2, with no postoperative complications. Routine vitamin and mineral supplements were prescribed. At 6-months' follow-up, he achieved a complete diet, and blood pressure was controlled with only one drug. Progression of his postoperative weight is shown in table 1. Six months after BS, he presented with numbness of the right foot and *steppage* gait. He recalled a frequent habit of crossing his legs. Physical examination revealed normal reflexes and sensitivity, but diminished muscle strength (muscular balance in dorsal flexion of the foot 1/5). Laboratory data revealed normality of glucose and lipid metabolism parameters and no nutritional deficiencies, except for low 25-hydroxivitamin D: total proteins 7 g/dL, serum albumin 4.4 g/dl, serum iron 69 ug/dL, folic acid 17.7 ng/mL (2-20), vitamin B12 365 pg/mL (120-900), zinc 95 ug/dL (60-150), selenium 80 ug/L (60-120), copper 119 ug/dL (70-140), 25-OH-vitamin D 9.6 ng/ml (30-100), retinol 0.49 mg/L (0.43-0.67), ratio vitamin E/cholesterol 6.1 mg/g (5-12). Electromyogram evidenced loss of anterior tibial muscle neurons and decrease in conduction speed, which confirmed the suspicion of mononeuropathy of right peroneal nerve. Physical therapy was recommended and neurological symptoms disappeared completely six months later. Vitamin and mineral oral supplementation was maintained.

Discussion

Up to 16% of patients can develop peripheral neurologic complications following BS, according to observational series.[4-6] Peroneal neuropathy (PN), however, is unusual. In general, it may be present in up to 10-15% of patients complaining of sensorimotor symptoms such as paresthesias and foot drop,[7] but, in a large controlled study, PN was identified in only 2 of 435 patients after BS.[2] In the two cases that we report, PN

appeared approximately six months after the surgical procedure, when weight loss had been significantly greater, in comparison to age- and sex-matched controls in our center (table I). These have been the only two cases identified in our own series of almost 1,500 bariatric surgeries performed over a period of twenty years, though it is true that electromyography is not routinely carried out in the postoperative follow-up, and subclinical cases may have been missed out.

Clinical symptoms of PN comprise pain at the site of entrapment, occasional paresthesias, foot drop and a slapping gait, all of which determine a significant functional limitation[8]. Examination shows weakness of toe dorsiflexion and foot dorsiflexion and eversion, and sensory loss may affect the entire territory of the superficial peroneal nerve (that is, dorsal foot and lateral leg), or be limited to a partial location. It is important to confirm the diagnosis with electromyography, and to distinguish it from a L5 radiculopathy. The two possible etiologies, which are not mutually exclusive, that have been assumed to explain the development of this complication after BS are nutritional and vitamin deficiencies on one hand, and peroneal nerve compression due to fat-pad loss at the fibular head, on the other.[7]

The patients here reported presented with vitamin and mineral levels within the normal range using oral supplementation exclusively. Although we were not able to evaluate thiamine levels at that time, vitamin B1 was included among the prescribed supplements following BS in the immediate postoperative period.

The fact that neurological involvement was limited to the peroneal nerve, probably suggests that the main cause was extrinsic compression, since peroneal nerve is especially vulnerable to impingement because of its superficial location.[8] Excessive weight loss could determine fat-pad loss at the fibular head, and would be one of the main responsible mechanisms contributing to nerve entrapment in this clinical setting. In case number 1, the rapid and disproportionate weight loss was caused by her restrictive behavior, as it has been previously described[9]. And in case number 2, the habit of crossing legs may have contributed to unleashing neurological symptoms, as it has also been described.[3]

Vitamin deficiency, on the other hand, would be less plausible; this would be more relevant in cases of polyneuropathy. Ischemic origin was reasonably ruled out due clinical presentation and normality of glucose and lipid metabolism parameters.

Management of mononeuropathies after BS has not been thoroughly evaluated; there is not enough evidence regarding the best treatment approach, and both vitamin supplementation and surgical decompression have been proposed. To our knowledge, the majority of case-series published have used the latter as the first option.[3] However, we describe improvement of peroneal deficits in these patients using conservative and physical therapy. An adequate nutritional approach after BS has been associated to a lower rate of complications involving the peripheral nervous system.[10] But additionally, the cases that we present suggest that a suitable nutritional control may probably be helpful as well for recovery of peripheral neurological symptoms already established, by keeping vitamin and mineral levels within a normal range and, more importantly, with an appropriate caloric intake.

We remark the importance of close follow-up of patients undergoing BS in order to avoid excessive weight loss. In case this happens, development of peripheral mononeuropathies may occur, regardless of the normality of mineral and vitamin levels. PN is not frequent, but it is truly invalidating, and may be a hallmark indicating that loss of weight is occurring too rapidly and in a disproportionate amount. Early detection and nutritional intervention may avoid the need for surgical nerve decompression.

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