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Persistent Lipedema and Folate-Deficiency Axonal Polyneuropathy after Sleeve Gastrectomy: A Case Report

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Abstract **Case Report**

Lipedema is a chronic, underdiagnosed disorder of adipose tissue that almost exclusively affects women and is characterized by painful, disproportionate fat accumulation in the limbs. Bariatric surgery, although effective for weight loss, does not consistently improve lipedema and may promote deficiency-related neurological complications. We report the case of a 27-year-old woman who presented, four months after sleeve gastrectomy for morbid obesity (preoperative BMI 42 kg/m²), with bilateral lower-limb edema, distal cyanosis, and neuropathic pain, in a context of irregular postoperative follow-up and poor adherence to supplementation. Examination revealed typical lipedema and a distal "sock-and-glove" sensory deficit. Electroneuromyography demonstrated a length-dependent axonal polyneuropathy, and laboratory testing revealed folate deficiency. A multidisciplinary strategy was implemented: neuropathic pain management (pregabalin, low-dose amitriptyline), correction of deficiencies (folate and a reinforced bariatric multivitamin supplement), and conservative treatment of lipedema (compression and manual lymphatic drainage). At three months, the edema, skin color, and neurological symptoms had markedly improved. This case highlights that lipedema may persist or be unmasked after bariatric surgery and that inadequate nutritional follow-up exposes patients to deficiency-related axonal polyneuropathies, particularly from folate deficiency. Structured, lifelong multidisciplinary monitoring is essential.

Keywords: Lipedema, Sleeve gastrectomy, Bariatric surgery, Axonal polyneuropathy, Folate deficiency, Micronutrients.

INTRODUCTION

Lipedema is a chronic disorder of adipose tissue distribution, almost exclusively affecting women, characterized by symmetrical, painful, diet-resistant enlargement of the limbs that spares the feet [1]. Often confused with obesity or lymphedema, it remains underdiagnosed [1]. Sleeve gastrectomy (SG) is an effective treatment for morbid obesity. However, major weight loss does not consistently improve lipedematous disease and may even accentuate its clinical signs [2,3]. In addition, SG exposes patients to micronutrient deficiencies, a source of neurological complications such as length-dependent axonal polyneuropathies, which may occur from a few weeks to several years after surgery [4,5]. We report the association of persistent lipedema and folate-deficiency polyneuropathy after SG and discuss its practical implications.

CASE REPORT

Presentation and History

A 27-year-old woman was hospitalized for lower-limb symptoms that had appeared two months earlier, four months after SG performed for morbid obesity (preoperative BMI 42 kg/m²). She reported progressive, painful swelling of both legs with distal bluish discoloration, together with burning and "sock-like" paresthesia. She denied any dyspnea, unilateral pain, or foot edema. Postoperative follow-up had been irregular, with non-adherence to the prescribed supplementation. Her history included polycystic ovary

syndrome (PCOS) with irregular menstruation, with no known diabetes, thyroid, or neurological disease and no edema-inducing medication.

Clinical Examination

General condition was preserved, with stable hemodynamic parameters. Cardiovascular (normal transthoracic echocardiography) and abdominal (soft abdomen, clean scars) examinations were unremarkable. Examination of the lower limbs (Figure 1) revealed bilateral edema with distal "sock-like" cyanosis and symmetrical, non-pitting thickening from the upper thighs to the ankles, with a "bracelet" sign (sparing of the feet). The skin was tender on palpation. Neurological examination showed a distal "sock-and-glove" sensory deficit without motor deficit, with preserved deep tendon reflexes and pain-related limitation of movement.

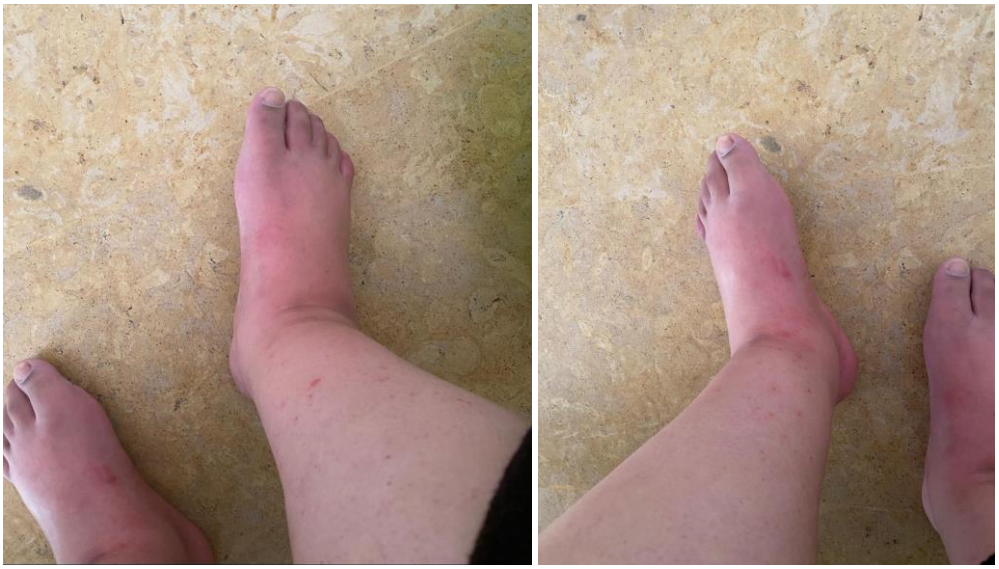

Figure 1: Clinical appearance of the lower limbs on admission (two views): bilateral edema with distal "sock-like" cyanosis and erythema, symmetrical non-pitting thickening, and sparing of the feet ("bracelet" sign)

Investigations

Venous Doppler ultrasound of the lower limbs was normal, ruling out deep vein thrombosis or significant chronic venous insufficiency; serum albumin and NT-proBNP were normal. Electroneuromyography confirmed a length-dependent sensorimotor axonal polyneuropathy of probable deficiency origin, and spinal MRI was normal. Laboratory testing (Table 2) revealed serum folate deficiency, with vitamin B12, thiamine (B1), pyridoxine (B6), copper, and zinc levels at the lower limit of normal. An elevated TSH with normal free T4, a normal cortisol level, and hormonal parameters consistent with PCOS (elevated LH, low FSH, elevated testosterone) were also noted. Blood glucose, HbA1c, renal and hepatic function, serum protein electrophoresis, and CRP were normal.

Diagnosis and Management

Two diagnoses were made: (1) lower-limb lipedema meeting clinical criteria [1], probably influenced by PCOS; and (2) a length-dependent sensorimotor axonal polyneuropathy in the setting of post-SG micronutrient deficiencies, with documented folate deficiency and other low-normal micronutrients, consistent with a possibly multifactorial deficiency neuropathy. A multidisciplinary approach was implemented, combining neuropathic pain management (pregabalin 75 mg twice daily, then dose adjustment, and amitriptyline in progressive doses), correction of deficiencies (oral folic acid 1 mg/day until normalization and strict resumption of a bariatric-specific multivitamin supplement [6]), and conservative treatment of lipedema (class II compression stockings and daily manual lymphatic drainage).

Outcome

The course was marked by progressive regression of the lipedema, a significant decrease in pain, and normalization of skin color. By day 15 of hospitalization, a marked reduction of the edema and resolution of the distal cyanosis were already observed (Figure 2). Neuropathic treatment was tapered stepwise according to tolerance. At the three-month follow-up, the patient reported a marked reduction (>70% on a visual analog scale) in pain and paresthesia, examination confirmed a clear decrease in lower-limb volume, and treatment adherence was good. Long-term nutritional and cutaneous monitoring was planned.

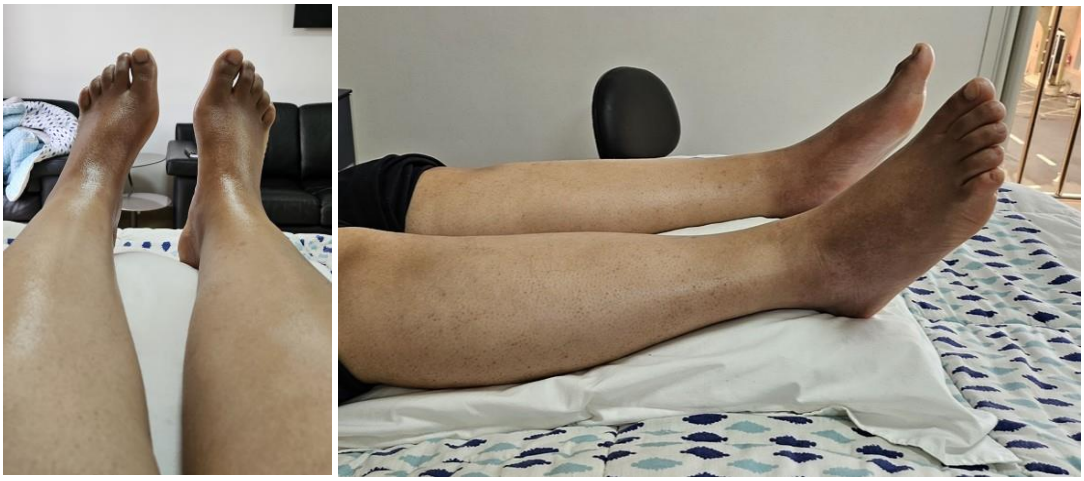

Figure 2: Clinical appearance on day 15 of hospitalization (two views): marked reduction of the edema, resolution of the distal cyanosis, and normalized skin color

DISCUSSION

Recognition of the Entities

First described by Allen and Hines in 1940, lipedema was long confused with obesity or lymphedema, a source of diagnostic delay [1]. Its recognition as a distinct entity has become established over the past two decades, alongside the characterization of its hormonal and inflammatory pathophysiology [7]. Likewise, the neurological complications of bariatric surgery, initially reported as isolated cases, have been systematized with the growth of these procedures, revealing a spectrum ranging from peripheral neuropathy to deficiency-related encephalopathy [4,8]. Massive weight loss is therefore not synonymous with resolution of all comorbidities and may even unmask underlying conditions.

Pathophysiological Interrelation

The clinical picture results from two mechanisms. Lipedema is related to adipocyte dysfunction (hyperplasia/hypertrophy), capillary fragility, and local lymphatic micro-dysfunction, influenced by sex hormones [1,7]. Bariatric surgery reduces overall fat mass but does not act on this pathological fat and may even make the limb-to-trunk disproportion more apparent [2,3]. In parallel, it can cause micronutrient deficiencies through reduced intake, vomiting, and impaired absorption, even in a so-called restrictive procedure [5,8]. Deficiency in folate and other micronutrients involved in neuronal metabolism (B12, copper, B1) may contribute to post-bariatric peripheral axonal damage [8,9].

Comparison with the Literature and Case Specificities

Our observation is consistent with recent reviews: the persistence of lipedema despite significant weight loss is increasingly reported, and bariatric surgery does not appear to be a curative treatment for lipedema, as the pathological fat may persist and require specific management [2,3]. Our case illustrates this paradigm, with the improvement in truncal obesity having "unmasked" the typical morphology of lipedema in a context of PCOS. Regarding the neuropathy, the presentation is consistent with the classic picture of a post-bariatric length-dependent axonal polyneuropathy [4,5]. Although thiamine (B1) and vitamin B12 deficiencies are the most frequently implicated, the role of folate deficiency is well documented, particularly in cases of malabsorption or inadequate supplementation [8,10]. The particularity of our observation lies in the concomitant association of the two conditions in the same patient, creating a mixed picture in which neuropathic pain and lipedema-related pain may compound one another and complicate the initial assessment. The joint improvement under comprehensive treatment supports the hypothesis of an additive contribution of the two entities.

Clinical Implications and Perspectives

The coexistence of these conditions argues for an integrated strategy: preoperative screening for lipedema in candidates for bariatric surgery, in order to adjust therapeutic expectations; protocolized, lifelong micronutrient monitoring, in line with recommendations advocating a complete laboratory panel every 3 to 6 months during the first year and annually thereafter, including folate assessment alongside vitamin B12, thiamine, and copper [6]; and a dual clinical evaluation for any persistent lower-limb symptom after SG, seeking both a deficiency-related neuropathic cause and an exacerbation of lipedema. Future research should clarify the true prevalence of lipedema in the bariatric population, validate optimized supplementation protocols, and evaluate combined therapeutic approaches tailored to this dual pathology.

Limitations

The limitations stem from the observational nature of this report. The role of PCOS, a possible confounder for lipedema, cannot be established. The potentially multifactorial nature of the neuropathy, with several micronutrients at the lower limit, precludes attributing all symptoms to folate deficiency alone.

Finally, the specific effect of each therapeutic component in the overall improvement cannot be isolated.

Conclusion

This case highlights that lipedema may persist after sleeve gastrectomy and that inadequate nutritional follow-up exposes patients to serious neurological complications, such as folate-deficiency axonal polyneuropathy. It argues for proactive, structured, multidisciplinary post-bariatric follow-up, incorporating rigorous laboratory monitoring and a sound knowledge of lipedema, in order to prevent complications and improve patients' quality of life.

Table 1: Timeline of Clinical Events

Time after SG	Event
Month 0	Sleeve gastrectomy for morbid obesity (BMI 42 kg/m²).
Months 2–4	Irregular follow-up; poor treatment adherence.
Month 4	Onset of symptoms (edema and neuropathic pain).
Month 4.5	Hospitalization and complete diagnostic workup.
Month 4.5	Initiation of multidisciplinary management.
Month 5	Day 15 of hospitalization: marked reduction of edema and resolution of cyanosis (Figure 2).
Month 7.5	Three-month follow-up: sustained improvement.

Table 2: Micronutrient and Biochemical Workup on Admission

Parameter	Value	Reference range	Interpretation
Serum folate	1.8 ng/mL	>3.0 ng/mL	Deficiency
Vitamin B12	220 pg/mL	200–900 pg/mL	Lower limit
Thiamine (B1)	90 nmol/L	70–180 nmol/L	Lower limit
Copper	70 µg/dL	70–140 µg/dL	Lower limit
Zinc	0.65 mg/L	0.66–1.10 mg/L	Low (borderline)
TSH	6.55 µIU/mL	0.27–4.2 µIU/mL	Elevated
Free T4	1.01 ng/dL	0.93–1.7 ng/dL	Normal
LH	18.12 mIU/mL	1.7–8.6 mIU/mL (follicular phase)	Elevated
FSH	2.99 mIU/mL	3.5–12.5 mIU/mL (follicular phase)	Low
Testosterone	3.39 nmol/L	0.29–1.67 nmol/L	Elevated
Cortisol (8 a.m.)	278 nmol/L	171–536 nmol/L	Normal

Declarations

Funding: No funding was received for this work.

Conflicts of interest: The authors declare no conflicts of interest related to this article.

Ethical approval: This case report was conducted in accordance with the principles of the Declaration of Helsinki, and approval was obtained from the ethics committee of Oued Eddahab Military Hospital, Agadir, Morocco.

Informed consent: Written informed consent was obtained from the patient for the publication of this case report, the associated anonymized clinical data, and the accompanying photographs.

Author contributions: All authors contributed to the conception, drafting, and critical revision of the manuscript and approved the final version.

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