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Sequential Management of Stage 3/Type III Lipedema With a Ketogenic Dietary Phase Followed by Adjunctive Photothermal Biomodulated Autologous Platelet-Rich Plasma: A Descriptive Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: Dr. Isabel Bové Farré is co-founder of Nesai Smart Technology S.L.

Patient: Female, 47-year-old
Final Diagnosis: Stage 3/Type III lipedema
Symptoms: Adipose tissue accumulation involving the hips, buttocks, thighs, and peri-knee region • lobular adipose prominences and folds over the thighs and around the knees • pain elicited by palpation • skin dimpling • symmetrical disproportionate enlargement of the lower body
Clinical Procedure: —
Specialty: Dermatology
Objective: Unusual setting of medical care
Background: Lipedema is a chronic adipose tissue disorder associated with pain, tissue fibrosis, and functional impairment. Evidence supporting nutritional strategies and adjunctive local treatments remains limited.
Case Report: A 47-year-old woman with stage 3/type III lipedema and obesity underwent an individualized ketogenic, carbohydrate-restricted, normoprotein dietary phase for 7 months. This was followed by a 4-month local intervention phase including 4 monthly sessions of multipuncture administration of autologous platelet-rich plasma after ex vivo photothermal biomodulation with blue light (467 nm, 2 J/cm^2, 37 °C, 10 min). Body composition was assessed by bioelectrical impedance analysis, fat thickness by ultrasonography, pain by visual analog scale, and patient-reported global aesthetic improvement at the final visit. Over 11 months, body weight decreased from 103.4 to 72.0 kg, body mass index from 37.1 to 25.8 kg/m^2, fat mass from 47.2 to 23.2 kg, and visceral fat level from 12 to 6. Subcutaneous, preperitoneal, and omental fat thickness also decreased. Pain decreased from 9/10 at baseline to 5/10 at final assessment; patient-reported global aesthetic improvement was 5/5, and no adverse events were documented.
Conclusions: Most objective changes occurred during the dietary phase. During the local intervention phase, the diet continued to be modified, and body weight decreased further, confounding attribution of second-phase outcomes specifically to photothermal biomodulated platelet-rich plasma. Pain was not assessed at the end of the dietary phase. Therefore, the independent contribution of photothermal biomodulated platelet-rich plasma to second-phase outcomes cannot be determined. This case should be interpreted as a descriptive report rather than as evidence of efficacy, mechanism, or additive benefit.

Keywords: Lipedema • Low-Level Light Therapy • Platelet-Rich Plasma • Ultrasonography
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954921

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Introduction

Lipedema is a chronic, symmetrical, and progressive condition characterized by disproportionate accumulation of subcutaneous adipose tissue, predominantly in women [1,2]. It is usually diagnosed clinically and may be associated with pain, tissue tenderness, easy bruising, functional limitation, and impaired quality of life [3]. Conservative management commonly includes compression, exercise, manual or decongestive therapies, and symptom-directed supportive care, while surgery may be considered in selected patients [1-3]. With disease progression, tissue nodularity, fibrosis, contour irregularity, microvascular fragility, and possible lymphatic involvement can contribute to symptoms and functional burden [4]. Lipedema features provide the clinical context for multimodal supportive management; however, the rationale for adjunctive photothermal biomodulated platelet-rich plasma (PTBM-PRP) remains exploratory and should be distinguished from established lipedema pathophysiology.

Nutritional approaches have been explored as supportive strategies in lipedema, including ketogenic or low-carbohydrate dietary interventions [5,6]. The available evidence suggests that such approaches may reduce weight, body mass index, circumferences, and pain in some patients, although protocols vary and long-term effects require further study [7]. Non-surgical multimodal management has also been described in a 5-case series, including a patient with stage 3 lipedema treated with an anti-inflammatory diet followed by a ketogenic diet and other supportive measures [8].

Platelet-rich plasma and platelet-derived extracellular vesicles have been investigated in other contexts for tissue repair and vascular or lymphatic signaling [9]. Ex vivo PTBM-PRP with blue light has been proposed as a method to modify extracellular vesicle release. However, evidence in lipedema remains indirect and should not be interpreted as proof of clinical efficacy [10-12].

This case report describes the chronological course of a patient with stage 3/type III lipedema who was treated with a ketogenic dietary phase followed by a local intervention phase with 4 sessions of local multipuncture administration of autologous PTBM-PRP. The objective was to describe the clinical management, measurements, safety observations, and interpretive limitations.

Case Report

A 47-year-old woman presented with a clinical diagnosis of stage 3/type III lipedema. The diagnosis was made clinically. At baseline, she showed bilateral and broadly symmetrical disproportionate enlargement of the lower body, with adipose tissue accumulation involving the hips, buttocks, thighs, and peri-knee regions, pronounced contour irregularity, skin dimpling, and lobular adipose prominences and folds over the thighs and around the knees, findings considered compatible with the recorded stage 3 morphological classification (**Figure 1**). According to the baseline clinical record, adipose tissue involvement extended from the hips to the ankles, corresponding to a type III distribution. At baseline, the patient reported pain elicited by palpation, although its anatomical distribution was not documented. Obesity was present concomitantly; however, the painful and disproportionate lower-limb



Figure 1. Posterior and lateral clinical photographs at baseline, before the dietary phase, and at the final assessment after the local intervention phase.

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Table 1. Changes in body composition, ultrasonographic measurements, pain, and patient-reported global aesthetic improvement across the 11-month clinical course.

	Dietary phase			Local intervention phase		Overall change (baseline to final assessment)	
BIA	**Baseline**	**End of phase**	**Percentage change**	**End of phase**	**Percentage change**	**Δ**	**Percentage change**
Weight (kg)	103.4	74.8	-27.7	72.0	-3.7	-31.4	-30.4
Fat mass (kg)	47.2	25.7	-45.6	23.2	-9.7	-24.0	-50.8
Fat mass (%)	45.6	34.3	-24.8	32.2	-6.1	-13.4	-29.4
Lean mass (kg)	56.2	49.1	-12.6	48.8	-0.6	-7.4	-13.2
Muscle mass (kg)	53.4	46.6	-12.7	46.3	-0.6	-7.1	-13.3
Total water (kg)	40.9	34.7	-15.2	34.3	-1.2	-6.6	-16.1
Water (%)	39.6	46.4	17.2	47.6	2.6	+8.0	20.2
Bone mass (kg)	2.5	2.5	0.0	2.5	0.0	0.0	0.0
Visceral fat level	12	7	-41.7	6	-14.3	-6	-50.0
Body mass index	37.1	26.8	-27.8	25.8	-3.7	-11.3	-30.5
Ultrasonography							
Subcutaneous fat (mm)	32	14	-56.3	11	-21.4	-21	-65.6
Preperitoneal fat (mm)	4	4	0.0	3	-25.0	-1	-25.0
Omental fat (mm)	20	8	-60.0	7	-12.5	-13	-65.0
Steatosis (Yes/No)	No	No	-	No		No change	
Clinical							
VAS pain	9/10	-	-	5/10		-4 points; attribution by phase not possible	
GAIS	-	-	-	5/5		Very much improved	

Note: Δ indicates absolute change or difference between the corresponding start and end values, while % change indicates relative percentage change from the corresponding start value. For variables already expressed as percentages, Δ represents percentage-point change.
Abbreviations: Δ, change or difference; BIA, bioelectrical impedance analysis; VAS, visual analog scale; GAIS, Global Aesthetic Improvement Scale.
Body composition parameters were measured using BIA, and visceral fat level was graded from 1 to 20. Values are reported at baseline, at the end of the dietary phase, and at the final assessment after the local intervention phase. Phase-specific relative percentage changes were calculated as [(end value - start value)/start value] × 100. Overall change from baseline to final assessment is presented as both absolute change (Δ = final value - baseline value) and relative percentage change. For variables expressed as percentages, Δ represents the percentage-point difference, whereas the Percentage Change columns represent the relative percentage change in the percentage value.

distribution was considered not fully explained by generalized obesity alone. The available records did not document easy bruising, edema, distal foot involvement or cuffing, Stemmer sign, functional limitations, or dedicated venous or lymphatic imaging. She had no allergy to topical anesthetics and no other active disease reported at presentation. Before this treatment course, she had tried dieting but had not received medication or supplemental micronutrition specifically for lipedema.

At baseline, body weight was 103.4 kg, body mass index (BMI) was 37.1 kg/m², visual analog scale (VAS) pain was 9/10, and ultrasonography showed no steatosis. Baseline bioelectrical impedance analysis (BIA) and ultrasound values are detailed in **Table 1**.

The case was reported retrospectively, with written informed consent from the patient for the publication of clinical data and photographs. It did not require ethical approval in accordance with local or national guidelines.

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Therapeutic Intervention

The patient was treated in 2 consecutive clinical phases: a dietary phase of variable duration, which lasted 7 months in this case (from April 17, 2024, to November 18, 2024), followed by a local intervention phase, which lasted approximately 4 months (from November 18, 2024, to March 10, 2025). The overall clinical course lasted approximately 11 months (**Figure 2**).

Dietary Phase

During the dietary phase, the patient followed an individualized ketogenic, carbohydrate-restricted, and normoprotein dietary intervention aimed at reducing visceral and omental fat. Protein intake was calculated as approximately 1 g/kg of BIA-derived muscle mass, with recommended protein sources including fish, chicken, and turkey. Protein supplementation of 15 g/day was used to support protein targets and was discontinued during the later phase. At baseline, BIA-derived muscle mass was 53.4 kg, corresponding to an approximate protein target of 53 g/day; a 15 g/day protein supplementation was used to help meet this target and was discontinued during the later phase. The dietary intervention was adjusted during follow-up according to tolerability, body composition, and clinical assessments.

The available retrospective records did not include a prespecified daily caloric target, carbohydrate threshold, ketogenic ratio, ketone-monitoring schedule, formal adherence method, or structured exercise prescription. During this phase, the patient was encouraged to remain physically active but did not follow a structured exercise program.

During this phase, pain was assessed at baseline using the 10-point VAS, weight was controlled weekly, and other variables were assessed at baseline and at the end of the dietary phase. Some variables, detailed in **Figure 2**, were assessed using an electrical bioimpedance analyzer, Tanita T6360 (Tanita Corporation, Tokyo, Japan), which operated at a single frequency of 50 kHz, in accordance with the standard specifications for this type of equipment. Measurements were conducted under controlled conditions, using the same device and by the same operator. All variables were assessed at the same hour (in the morning to reduce circadian variability), after urination, with strenuous physical exercise avoided for 12 hours prior to the measurement, and with no metal objects worn during the assessment. Following the manufacturer's instructions, the patient stood barefoot on the device platform and held the electrodes. The skin of the soles was cleaned, and a standardized posture was maintained throughout the assessment (legs and arms were positioned at 45° relative to the midline of the body and at 30° from the chest, respectively).

Ultrasonography assessments, listed in **Figure 2**, were conducted using a LOGIQ S8 Ultrasound System (GE Healthcare, Chalfont St Giles, UK). The device was equipped with a 9L-D broadband linear transducer (operating frequency 3-10 MHz/12 MHz). B-mode and Doppler settings were optimized to examine hypodermic structures. Subcutaneous fat measurements were taken at the trochanteric zone, and preperitoneal and omental fat thicknesses were measured at the level of the fourth lumbar vertebra, where the abdominal aorta bifurcates into the iliac arteries.

The duration of the dietary phase was not prespecified and varied according to the patient's response. In routine clinical practice, transition to the local intervention phase occurred when omental fat thickness was considered within the normal range on ultrasonographic assessment; in this patient, this occurred after 7 months.

Local Intervention Phase

During this phase, the patient's diet was modified, with protein supplementation being discontinued, some foods, including legumes, being progressively reintroduced, and other foods, such as red meat, being eliminated. The patient was instructed to eat every 5 hours, with 1 or 2 fasting days every 15 days, and to continue the same nonscheduled physical activity pattern.

This phase included 4 monthly sessions of locally injected autologous PTBM-PRP. Autologous PRP was prepared from approximately 35 mL of peripheral blood collected using a 21-G needle. Peripheral hematology testing was performed as part of routine clinical care to confirm the absence of thrombocytopenia, but these tests were not designed to characterize the final PRP product. Platelet concentration, leukocyte content, erythrocyte content, and platelet enrichment factor in the final PTBM-PRP preparation were not measured and were unavailable in the retrospective records. Whole blood was placed in citrate tubes and double centrifuged at 3000 rpm for 15 minutes to obtain approximately 20 mL of PRP per session. The corresponding relative centrifugal force (× g) was not available in the retrospective records.

Before administration, PRP underwent ex vivo photothermal biomodulation using a CE-marked device and 2 single-use 10 mL kits (MCT Unit and MCT Kit, Meta Cell Technology, Sant Cugat del Vallès, Spain). The predefined settings were 467 nm, 2 J/cm^2, 37 °C, and 10 minutes. The biological effect of this preparation step was not directly measured in the patient. Approximately 20 mL of PRP was administered per session, distributed as approximately 10 mL per lower limb. No exogenous platelet activator was added before injection. Each lower limb was divided into 2 treatment segments, with approximately 5 mL administered per segment.

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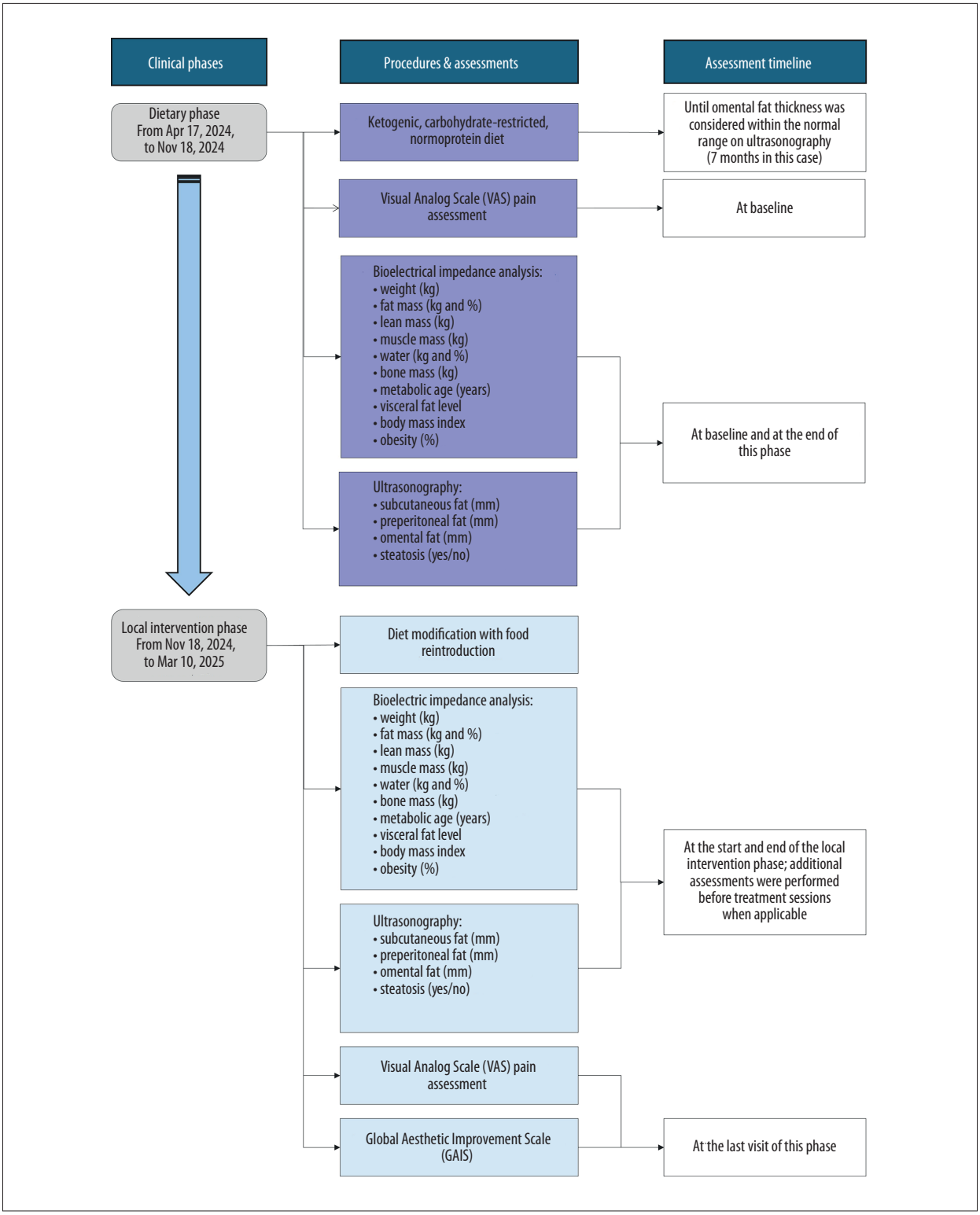


Figure 2. Timeline of the sequential clinical course, including the dietary phase (from April 17, 2024, to November 18, 2024), followed by a local intervention phase (from November 18, 2024, to March 10, 2025) with an overall clinical course of approximately 11 months. Assessments included bioelectrical impedance analysis (BIA), ultrasonography, visual analog scale (VAS), and Global Aesthetic Improvement Scale (GAIS).

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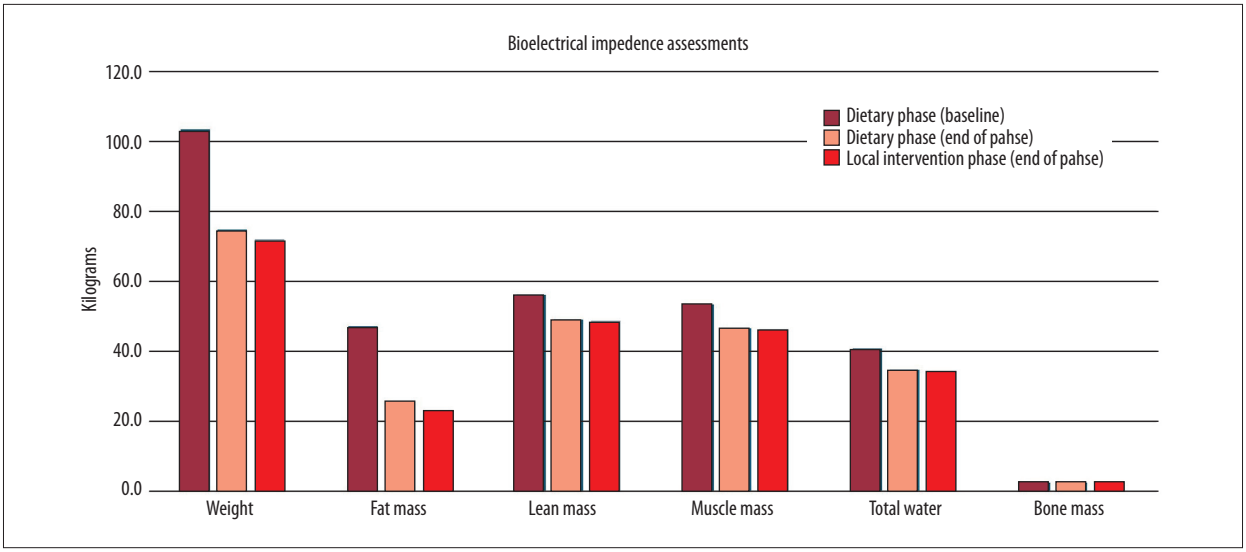


Figure 3. Evolution of bioelectrical impedance analysis-derived weight, fat mass, lean mass, muscle mass, total water, and bone mass at baseline, at the end of the dietary phase, and at the final assessment.

Before the treatment injection, the skin was cleansed with chlorhexidine, and topical lidocaine/tetracaine cream was applied for 20 minutes. PTBM-PRP was administered using a multipuncture mesotherapy technique with a 30-G needle, approximately 0.1 mL per puncture, at approximately 0.5-cm intervals, at an angle of approximately 30 degrees, and to a depth of approximately 2 mm.

After injection, topical arnica cream was applied manually over treated areas for comfort. Safety monitoring consisted of immediate observation and 24-hour telephone follow-up after each session, including questions about pain exacerbation, bruising, bleeding, local swelling, nodules, hypersensitivity, infection, fever, or technical complications.

During the local intervention phase, body composition and ultrasonographic assessments were performed before treatment sessions when applicable, using the same devices and procedures as in the dietary phase. The patient avoided strenuous exercise for 12 hours before assessment, removed metal objects, stood barefoot on the platform, and held the electrodes as directed by the manufacturer. Ultrasonography was performed using the same device as in the dietary phase. The ultrasonographic assessments included steatosis, subcutaneous fat at the trochanteric zone, and preperitoneal/omental fat at the fourth lumbar vertebra level, corresponding to the abdominal aortic bifurcation.

Pain was assessed at baseline and at the final visit, using a 10-point VAS. Patient-reported global aesthetic improvement was assessed using the Global Aesthetic Improvement Scale at the final visit of the phase. No validated lipedema-specific quality-of-life questionnaire and no inflammatory biomarkers were collected.

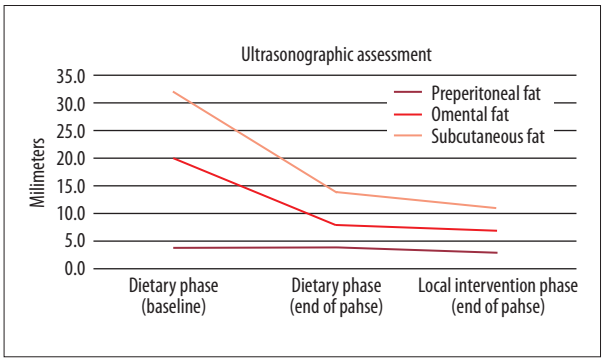


Figure 4. Evolution of ultrasound-measured subcutaneous, preperitoneal, and omental fat thickness at baseline, at the end of the dietary phase, and at the final assessment.

Treatment Outcomes

Most objective changes were already present by the end of the dietary phase. **Table 1** reports the main clinically relevant assessment points: baseline, end of the dietary phase, and final assessment after the local intervention phase. During the dietary phase, body weight decreased from 103.4 to 74.8 kg, BMI from 37.1 to 26.8 kg/m^2, fat mass from 47.2 to 25.7 kg, and visceral fat level from 12 to 7 (**Figure 3**); subcutaneous and omental fat thickness decreased from 32 to 14 mm and from 20 to 8 mm, respectively, while preperitoneal fat remained unchanged (**Figure 4**).

During the local intervention phase, body weight decreased from 74.8 to 72.0 kg, BMI from 26.8 to 25.8 kg/m^2, fat mass from 25.7 to 23.2 kg, and visceral fat level from 7 to 6 (**Figure 3**). Ultrasonographic assessments showed additional reductions

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in subcutaneous, preperitoneal, and omental fat thickness of 3.0, 1.0, and 1.0 mm, respectively (**Figure 4**).

From baseline to the final assessment, body weight decreased by 31.4 kg, BMI by 11.3 kg/m^2, fat mass by 24.0 kg, and visceral fat level by 6 points; lean mass decreased by 7.4 kg and muscle mass by 7.1 kg, while bone mass remained stable (**Figure 3**). VAS pain decreased from 9/10 at baseline to 5/10 at the final assessment. Because pain was not assessed at the end of the dietary phase, the timing of pain improvement and the contribution of each phase cannot be determined. No adverse events, technical issues, or complications were reported during the local intervention phase. At the final assessment, the patient rated global improvement as 5/5 on the Global Aesthetic Improvement Scale.

Discussion

This case describes the sequential clinical course of a woman with stage 3/type III lipedema and obesity who underwent a 7-month dietary phase followed by a 4-month local intervention phase, including 4 monthly sessions of locally injected autologous PTBM-PRP. The main strength of the report is the chronological documentation of body composition and ultrasonographic measurements across the clinical course. Most objective changes were already present by the end of the dietary phase, before the local PTBM-PRP-containing phase. Importantly, most objective measures reported in this case, including body weight, BMI, total fat mass, visceral fat level, and abdominal fat-thickness measurements, primarily reflect generalized adiposity or body composition changes. They should not be interpreted as direct measures of improvement in lipedema-specific pathological tissue. The absence of validated lipedema-specific patient-reported outcomes, limb volume measurements, functional assessments, and quality of life instruments further limits disease-specific interpretation.

Reduction of adipose tissue burden may decrease mechanical compression on lymphatic and microvascular structures and may contribute to improvement in symptoms and skin appearance. It is important to emphasize that disproportionate fat accumulation is only one component of lipedema, which is also associated with broader pathophysiological alterations, including inflammatory and fibrotic changes [13].

Ketogenic dietary approaches have been increasingly investigated as supportive strategies in lipedema management [5,6]. In this case, the dietary phase likely contributed most to the objective improvements observed, and most changes were associated with major reductions in body weight and fat mass. However, the apparent reductions in BIA-derived lean and muscle mass should be interpreted cautiously because BIA estimates are influenced by hydration and fluid distribution and do not represent direct measurements of muscle tissue. These findings nevertheless underscore the importance of individualized nutritional supervision and functional monitoring. Conservative multimodal care may also include exercise, manual therapies, compression-based strategies, and other combined approaches depending on the clinical context [14,15]. In the present case, the multimodal approach included a PTBM-PRP injection; however, its independent contribution to the subsequent local intervention phase cannot be determined in this uncontrolled single-case study. The PTBM-PRP procedure was clinically feasible and well tolerated; however, the high patient-reported global aesthetic improvement observed at the final assessment cannot be attributed specifically to PTBM-PRP, because dietary modifications continued concurrently during the local intervention phase. The rationale for including autologous PTBM-PRP after the dietary phase was exploratory, based on proposed regenerative and signaling properties described in other contexts [10-12,16] and on the involvement of platelet-derived factors and extracellular vesicles in local tissue repair [9,17]. However, the evidence supporting this rationale in lipedema is indirect and derived mainly from non-lipedema or experimental settings. The present findings should not be interpreted as proof of mechanism, efficacy, or additive benefit of PTBM-PRP but as a descriptive case that may support future investigations in lipedema management, particularly in maintenance-oriented settings after the main metabolic improvement has been achieved.

The decrease in pain was clinically relevant to the patient but cannot be attributed to the local intervention phase, since VAS was not assessed at the end of the dietary phase. Similarly, anti-inflammatory claims cannot be made because C-reactive protein, interleukin 6, tumor necrosis factor alpha, and other inflammatory biomarkers were not measured.

The case has practical relevance as a hypothesis-generating report. It suggests that sequential management can be documented using body composition, ultrasound, pain, and patient-reported global aesthetic improvement assessments. However, future prospective studies would require standardized staging, reproducible nutritional prescriptions, interim pain measurements, biomarkers, functional outcomes, and an appropriate comparator.

The case presents several limitations. It is a single-patient retrospective case report without a control group. The retrospective records did not contain complete documentation of all elements relevant to the differential diagnosis of lipedema, including pitting edema, distal foot involvement, Stemmer sign, and dedicated venous or lymphatic imaging, limiting retrospective reconstruction of the diagnostic assessment. The dietary intervention was individualized and incompletely documented in

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the retrospective records. Specifically, the available records did not include a prespecified daily caloric intake, a carbohydrate threshold, a ketogenic ratio, a ketone-monitoring schedule, a formal adherence measure, or a structured exercise prescription. This limits reproducibility and should be considered when interpreting the observed changes. No repeatability or intraobserver variability assessment was performed for the ultrasound measurements. Therefore, the small second-phase differences in subcutaneous, preperitoneal, and omental fat thickness may fall within technical or intraobserver measurement variability and should be interpreted cautiously. No pain assessment was performed at the end of the dietary phase, preventing attribution of pain improvement to either phase. The final PTBM-PRP product was not biologically characterized. Platelet concentration, platelet enrichment factor, leukocyte content, erythrocyte content, and other compositional parameters were not available in the retrospective records, limiting reproducibility and biological interpretation of the local intervention. No inflammatory biomarkers or histologic and lymphatic functional measures were performed along the treatment course. BIA estimates may be affected by fluid distribution, and the device did not provide raw impedance parameters such as resistance, reactance, or phase angle. The numerical criterion used to define normalization of omental fat thickness was not documented in the retrospective records. Clinical photographs were obtained during routine clinical care and were not acquired using a fully standardized photographic protocol; therefore, they are presented for descriptive purposes only. Dietary prescription, PTBM-PRP sessions, and segmental injected volumes can vary between patients. No validated lipedema-specific quality-of-life questionnaire or adherence measures were used.

Conclusions

In this single retrospective case, sequential management of stage 3/type III lipedema with a ketogenic dietary phase followed by a local intervention phase, including 4 monthly sessions of locally injected autologous PTBM-PRP, was associated with reductions in weight, BMI, fat mass, and ultrasound-measured fat thickness, a lower final pain score, high patient-reported global aesthetic improvement, and no reported adverse events. Because most objective changes occurred during the dietary phase and pain was not assessed at the end of that phase, the findings should be considered descriptive and hypothesis-generating rather than evidence of efficacy, mechanism, or additive benefit of any individual component.

Department and Institution Where Work Was Done

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Statements

- Meta Cell Technology funded the article processing charge and provided medical writing and editorial support. It did not provide the device or consumables used in the patient's clinical treatment, and it had no role in patient selection, clinical decision-making, treatment administration, data collection, or outcome assessment. The authors retained responsibility for the interpretation of the clinical data, the final manuscript, and the decision to submit it for publication.

- Dr. Isabel Bové Farré is co-founder of Nesai Smart Technology S.L. This affiliation is disclosed as a potential conflict of interest. The authors declare that they received no personal fees or honoraria related to the preparation of this manuscript, and no additional financial conflicts related to this study are declared.

Patient Consent

Written informed consent for publication of clinical information and photographs was obtained from the patient.

AI Usage

Medical writing and editorial support were provided by an employee of Meta Cell Technology. Generative artificial intelligence (ChatGPT, OpenAI) was used solely to assist with language editing and editorial refinement. The authors critically reviewed and verified all content and retain full responsibility for the manuscript.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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