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[Mariangela Rondanelli](#) , [Clara Gasparri](#) , [Alessia Moroni](#) , [Elisa Genovese](#) , [Eugenio Marzio Valentini](#) , [Giorgia Leone](#) , [Simone Perna](#) ^{*} , [Giuseppe Mazzola](#)

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Keywords: sarcopenic obesity; body composition; multidisciplinary residential program



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Article

The Effects of a Multidisciplinary Residential Program on Risk Factors of Sarcopenic Obesity: An Open Label Trial Study in a Cohort of Institutionalized Italian Obese Older Adults

Mariangela Rondanelli ¹, Clara Gasparri ², Alessia Moroni ², Elisa Genovese ²,
Eugenio Marzio Valentini ², Giorgia Leone ² and Simone Perna ^{3,*} Giuseppe Mazzola ²

¹ Department of Public Health, Experimental and Forensic Medicine, University of Pavia, 27100 Pavia, Italy

² Endocrinology and Nutrition Unit, Azienda di Servizi alla Persona "Istituto Santa Margherita",
University of Pavia, 27100 Pavia, Italy

³ Division of Human Nutrition, Department of Food, Environmental and Nutritional Sciences (DeFENS),
Università Degli Studi di Milano, 26900 Milan, Italy

* Correspondence: simone.perna@unimi.it

Abstract: Background. Sarcopenic obesity, characterized by excess fat and reduced muscle mass/function, is linked to chronic inflammation and metabolic dysfunction. **Methods.** This study assessed a 2-month multidisciplinary residential program (MRP) for improving clinical and functional outcomes in 61 institutionalized obese Italian adults (mean age 60; 36 women, 25 men; BMI ≥ 30 with metabolic comorbidities). The MRP included personalized nutrition, physical activity, and psychological support. Outcomes included anthropometric, biochemical, body composition, and physical performance measures (via Short Physical Performance Battery [SPPB]), with sarcopenia risk evaluated using EWGSOP2 criteria. **Results.** Post-intervention, significant improvements were observed in SPPB scores (+0.93 units, $p < 0.001$), weight (-6.4 kg), BMI (-2.45 kg/m²), fat mass (-3.9 kg), visceral adipose tissue (-314.2 g), and fat-free mass index (-285.54 g; all $p < 0.01$). Glycemic control improved, with reductions in fasting glucose (-16.4 mg/dL), HbA1c (-0.81%), insulin (-2.77 mcU/mL), and HOMA-IR (-0.95; $p < 0.05$). Lipid profiles also improved: total cholesterol (-21.32 mg/dL), LDL (-12.10 mg/dL), and triglycerides (-39.07 mg/dL; all $p < 0.001$). **Conclusion.** The MRP effectively enhanced body composition, metabolic health, and physical function, underscoring its potential as a preferred strategy for managing sarcopenic obesity in institutional settings.

Keywords: sarcopenic obesity; body composition; multidisciplinary residential program

1. Introduction

Obesity represents a major global health concern, affecting both industrialized and developing countries with epidemic proportions. According to the 2022 European Regional Obesity Report published by the World Health Organization (WHO), 29% of European adults and nearly one in three children are either overweight or living with obesity [1]. In obesity, there is an increase in the accumulation of adipose tissue mainly at the visceral level [1]. The inflammatory process that occurs in visceral adipose tissue is closely related to insulin resistance, since inflammation interferes with the normal function of insulin, contributing to impaired glucose regulation in the body. Furthermore, inflammation is often associated with abnormal tissue restructuring and can progress to fibrosis [2]. Obesity, therefore, causes physiological alterations that can cause chronic inflammation in the body, negatively influencing various systems such as vascular, immune, metabolic, hormonal and bone. This condition of chronic inflammation can inhibit muscle protein synthesis, favouring the development of sarcopenia, characterized by the progressive loss of muscle mass and strength. In particular, excess adipose tissue increases the production of cytokines, further aggravating muscle alterations [3].

Sarcopenia is a condition previously defined as the gradual decline in muscle mass that occurs during aging. Over the years, more comprehensive definitions for sarcopenia have been proposed, including diagnostic criteria and functional measures. In 2019, the EWGSOP2 enriched the definition, considering sarcopenia as a muscle disease associated with increased risks of adverse outcomes such as falls, frailty, physical disability and a worse quality of life, characterized by a progressive and generalized loss of mass and skeletal muscle strength. Sarcopenia, therefore, is today considered as a muscle disease characterized by low muscle quantity and quality, to be considered serious when it is confirmed by reduced muscle strength and low physical performance [4].

When the first symptoms of sarcopenia appear, an individual may already be below the threshold of poor physical performance and, probably, even above the threshold of disability. However, both genetic and lifestyle factors can accelerate muscle deterioration and lead to functional impairment and disability. In fact, interventions such as proper nutrition and exercise appear to delay or even reverse this process [5]. The primary goal should therefore be to prevent or delay sarcopenia by maximizing and preserving muscle mass and strength during youth and young adulthood, maintaining it during middle age, and minimizing loss in later life [4].

When obesity and sarcopenia combine, with a complex interplay between muscle, adipose tissue, hormonal changes, inflammation, oxidative stress and lifestyle factors, the specific condition of sarcopenic obesity develops [6]. Furthermore, inadequate dietary behaviours, such as insufficient protein intake, and lack of physical activity can contribute to accelerating the progression of these conditions [7]. In this context, therefore, an imbalance occurs between lean mass and fat mass, with the latter exceeding the weight that the lean mass can support, determining functional limitations mainly related to the mechanical action of obesity, which overwhelms the support capacity provided by the muscle tissue. This affects body composition, resulting in reduced exercise tolerance and decreased muscle strength [8].

Given this background, The aim of this study was to evaluate the efficacy of a multidisciplinary residential program (MRP) in improving clinical and functional outcomes associated with risk of sarcopenia in a cohort of Italian obese patients.

2. Materials and Methods

2.1. Study Design and Population

This on open label study conducted at the Metabolic Rehabilitation Unit of Azienda di Servizi alla Persona, Istituto Santa Margherita, University of Pavia (27100 Pavia, Italy) in which both participant and researcher were aware of assigned treatment.

The study design was approved by the ethics committee of the University of Pavia, and an individual written informed consent was obtained from each participant. Data were gathered from 1 January 2021 to 1 September 2023. All the methods were performed in accordance with the CONSORT guidelines [9].

Eligible participants were aged >18 years with BMI ≥ 30 Kg/m² with one or more with metabolic comorbidities (type 2 diabetes mellitus, dyslipidaemia, high blood pressure, hyperuricemia, etc...) and the MRP lasted about two months.

2.2. Multidimensional Residential Program Interventions

Nutritional Intervention

Body weight reduction was induced by a low-energy mixed diet (55 % carbohydrates, 30 % lipids and 15 % proteins) providing 600 kcal less than individually energy requirements based on the measured TEE. The energy content and macronutrient composition of the diets adhered to the nutritional recommendations of the American Diabetes Association [10, 11]. These diets were designed to achieve weight losses of 0.5–1 kg per week; this type of diet is considered to be a low-risk intervention [12].

Individual diet plans were drawn up for each subject by the research dietitian. To optimize compliance, dietary instructions were reinforced each week by the same research dietitian. Each consultation included a nutritional assessment and weighing.

Patients were administered with vitamin D supplement only if they presented a value of 25-hydroxyvitamin D (25OHD) <30 ng/ml in blood tests at the beginning [13]. No other vitamin supplements were provided.

Physical Activity

The exercise program was based on the physical activity recommendations for adults proposed by the World Health Organization [14], on progression models in strength and aerobic training for healthy adults. Since there is limited information regarding the ideal exercise model for morbidly obese adults, we will combine strength and aerobic training (i.e., a concurrent training protocol), as previous findings in obese adults displayed important benefits when both strength and aerobic exercise are implemented in the same session [15] of 60 minutes of five days a week and more than 10.000 steps per day.

Physical activity was individualized and conducted every day by each subject with the help of qualified and properly trained physiotherapist.

Psychological Support

Psychological management is based on the enhanced cognitive behaviour therapy (CBT-E) approach, that is considered the most valid methodology for the treatment of eating disorders [16].

Psychological support during the MRP had the dual purpose of defining the presence of eating disorder and providing psychoeducation and strategies for adhering to the new diet. Individual interviews have been carried out weekly with the aim of reducing psychopathology, if present, investigating the factors of maintenance of the disorder and carrying out a cognitive restructuring. In addition, multidisciplinary group meetings are held with an expert dietitian to identify functional strategies for managing the diet once back home.

2.3. Measured Outcomes

Handgrip Test

The hydraulic hand dynamometer (Jamar 5030 J1, Sammons Preston Rolyan, Bolingbrook, Illinois, USA) was used to assess the handgrip strength of the muscles in accordance with established methods, and the accuracy was 0.6 N. With the elbow by the side of the body and the arm at right angles, the subject applies an isometric contraction while holding the dynamometer in the hand that will be examined. Muscle strength was assessed at times t0 and t1.

Short Physical Performance Battery (SPPB)

Physical performance was assessed using the a) SPPB, which comprised gait speed and balance (three different tests that assess the ability to stand with the feet together in the side-by-side, semi-tandem, and tandem positions); each component was scored from 0 (not possible) to 4 (best performance). This assessment was performed at t0 and t1.

Biochemical Analysis

Blood samples were collected at baseline and at the end of the treatment. In particular, nutritional status, lipid profile, glycaemic profile and status of inflammation were assessed.

Serum iron, lipids, uric acid, creatinine, and calcium were measured by enzymatic-colorimetric assay (Abbott Laboratories). PCR, Transferrin, Apo A1 and Apo B were determined by immunoturbidimetry (Roche). ESR was measured by the Westergren method using a DIESSE Analyzer, blood electrolytes by indirect ISE potentiometry (Abbott Laboratories), ionized Calcium by

selective electrode potentiometry, Insulin by Electro-chemiluminescence immuno-assay (ECLIA) (Roche Diagnostics). Blood glucose, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were analysed by Enzymatic UV Assay (Abbott Laboratories) and CBC by differential blood cell counter. Insulin resistance was evaluated using the Homeostasis Model Assessment (HOMA) [17].

Anthropometric Assessment

Anthropometric parameters, such as body weight, waist and hip circumference were measured weekly during recovery period. Body weight was measured to the nearest 0.1 kg, using a precision scale; participants wore light clothing, no shoes, and a standardized method was used [18]. The waist was measured at the midpoint between the top of the hip bone (iliac crest) and lowest rib, using a standardized method.

Body Composition Assessment by Double X-Ray Densitometry

Body composition (fat free mass, fat mass, visceral fat mass) was determined by dual-energy X-ray absorptiometry (DXA), using a Lunar Prodigy DXA (GE Medical Systems). In vivo CVs were 0.89% for whole body fat (fat mass) and 0.48% for FFM. The Skeletal Muscle Index (SMI) was taken as the sum of the fat-free soft tissue mass of arms and legs divided by height². Whole body and fat free mass (FFM) were divided by height squared to obtain FFM index (FFMI). FFM depletion was defined as having whole-body FFMI below the 5th centile for age- and gender-matched healthy subjects [19]. Visceral adipose tissue (VAT) volume was estimated using a constant correction factor (0.94 g/cm³). The software automatically places a quadrilateral box, which represents the android region, outlined by the iliac crest and with a superior height equivalent to 20% of the distance from the top of the iliac crest to the base of the skull [20]. Subcutaneous abdominal fat was defined as the difference between android fat and visceral fat. The in vivo CVs were 0.89% and 0.48% for FM) and FFM, respectively [21].

2.4. Statistical Analysis

Analyses were performed using IBM SPSS ver. 26.0. Descriptive statistics are presented as means and standard deviations for continuous variables. Normality of data was evaluated through Kolmogorov-Smirnov test. Pre post intervention changes were assessed by means of paired-t test. Statistical significance was accepted for p<0.05.

3. Results

A total of 61 obese patients (mean age 60.00 ± 13.48 years old) completed the study (36 women and 25 men). The baseline characteristics are reported in Table 1.

Table 1. Baseline characteristics of the sample.

Variable	Mean ± ds
Age (years)	60.00 ± 13.48
Height (m)	1.61 ± 0.11
Weight (kg)	106.5 ± 16.89
BMI (kg/m ²)	41.1 ± 5.71
Handgrip (kg)	25.49 ± 12.05
SMI	9.18 ± 1.20
SPPB	8,48 ± 2.69

Abbreviations: BMI, body mass index; SMI, skeletal muscle index; SPPB, short physical performance battery.

Table 2. Risk factors for sarcopenia at baseline, according to the reference cut-offs for the diagnosis of sarcopenia (EWGSOP2).

	SMI	Handgrip test	Chair test	Handgrip test AND Chair test	Handgrip test OR Chair test
Cut-off	M < 7 kg/m ² F < 5,5 kg/m ²	M < 27 kg F < 16 kg	> 15 sec	M < 27 kg F < 16 kg; > 15 sec	M < 27 kg F < 16 kg; > 15 sec
Men (n)	0	1	3	3	7
Women (n)	0	1	13	12	26
Total (n)	0	2	16	15	33
Total (%)	0%	3.28%	26.23%	24.59%	54.1%

Abbreviations: SMI, skeletal muscle index.

Table 3. Risk factors for sarcopenia at the end of the recovery, accordingly to the reference cut-offs for the diagnosis of sarcopenia (EWGSOP2).

	SMI	Handgrip test	Chair test	Handgrip test AND Chair test	Handgrip test OR Chair test
Cut-off	M < 7 kg/m ² F < 5,5 kg/m ²	M < 27 kg F < 16 kg	> 15 sec	M < 27 kg F < 16 kg; > 15 sec	M < 27 kg F < 16 kg; > 15 sec
Men (n)	0	1	4	1	6
Women (n)	0	6	10	8	24
Total (n)	0	7	14	9	30
Total (%)	0%	11.48%	22.9%	14.75%	49.18%

Abbreviations: SMI, skeletal muscle index.

As shown in Table 4, all the anthropometric and body composition variables significantly changed between pre and post recovery. Particularly, weight, BMI, body circumferences as well as fat mass, fat free mass and VAT improved significantly ($p < 0.001$ or $p < 0.01$). In concern to physical performance, only SPPB values increased significantly after the recovery ($p < 0.001$).

Regarding blood parameters, as there was a significant improvement of the overall values between pre and post recovery, it is noteworthy to mention how such intervention enhanced glycaemia ($p < 0.05$), glycosylated hemoglobin ($p < 0.01$), insulinemia ($p < 0.05$), total cholesterol ($p < 0.001$), LDL ($p < 0.001$) and triglycerides ($p < 0.001$) levels.

Table 4. Anthropometric parameters, body composition parameters and physical function parameters at the beginning and at the end of the recovery.

Variable	Pre (mean $\pm$ ds)	Post (mean $\pm$ ds)	Δ change (CI95: lower; upper)	P value
Weight (kg)	106.50 $\pm$ 16.89	100.1 $\pm$ 15.98	-6.40 (-7.132; -5.662)	0.001
BMI (kg/m ²)	41.14 $\pm$ 5.71	38.69 $\pm$ 5.60	-2.450 (-2.706; -2.193)	0.001
Arm circumference (cm)	36.84 $\pm$ 4.65	35.44 $\pm$ 3.83	-1.395 (-2.357; -0.432)	0.007
Calf circumference (cm)	42.12 $\pm$ 4.45	41.07 $\pm$ 3.53	-1.053 (-1.895; -0.211)	0.017
Waist circumference (cm)	125.88 $\pm$ 10.61	120.22 $\pm$ 10.83	-5.664 (-6.403; -4.925)	0.001
Hips circumference (cm)	127.612 $\pm$ 12.504	123.89 $\pm$ 12.41	-3.718 (-4.474; -2.961)	0.001
Total mass (kg)	106.50 $\pm$ 16.89	100.1 $\pm$ 15.98	-6.40 (-7.13; -5.66)	0.001
Fat mass (g)	49411.12 $\pm$ 10603.74	45504.46 $\pm$ 10428.60	-3906.66 (-4574.46; -3238.86)	0.001
Fat mass (%)	48.61 $\pm$ 6.89	46.67 $\pm$ 7.26	-1.94 (-2.44; -1.43)	0.001
Fat free mass (g)	51976.34 $\pm$ 10394.07	51174.89 $\pm$ 9799.68	-801.46 (-1335.31; -267.61)	0.004
FFMI	19889.81 $\pm$ 2279.42	19604.27 $\pm$ 2192.67	-285.54 (-487.31; -83.78)	0.006
VAT (g)	2701.29 $\pm$ 1272.15	2387.1 $\pm$ 1098.90	-314.19 (-452.08; -176.30)	0.001
Handgrip (kg)	25.49 $\pm$ 12.05	26.14 $\pm$ 11.52	1.117 (-0.303; 2.536)	0.121
SMI	9.179 $\pm$ 1.20	9.07 $\pm$ 11.23	-0.106 (-0.29; 0.08)	0.248
SPPB (score)	8.48 $\pm$ 2.69	9.41 $\pm$ 2.28	0.934 (0.55; 1.32)	0.0001

Abbreviations. BMI, body mass index; FFMI, fat free mass index; VAT, visceral adipose tissue; SMI, skeletal muscle index; SPPB, short physical performance battery. * In bold: value with $p < 0.05$.

Table 5. Blood chemistry parameters at the beginning and at the end of the recovery.

Variable	Pre (mean ± SD)	Post (mean ± SD)	Δ Change (95% CI)	P-value
Folate (ng/mL)	7.475 ± 4.18	7.38 ± 13.07	−0.10 (−2.96; 2.76)	0.919
Vitamin B12 (pg/mL)	437.33 ± 184.54	506.08 ± 207.58	+68.75 (−122.41; 259.91)	0.335
Iron (mcg/L)	89.95 ± 33.71	107.08 ± 23.70	−17.13 (−25.53; −8.73)	0.001
Transferrin (mg/dL)	258.15 ± 43.70	241.58 ± 43.26	−16.57 (−27.89; −5.25)	0.006
Vitamin D (ng/mL)	24.96 ± 11.91	26.86 ± 13.68	+1.90 (−2.95; 6.75)	0.338
ESR (mm/h)	27.46 ± 20.35	26.09 ± 15.03	−1.37 (−7.16; 4.43)	0.635
CRP (mg/dL)	0.61 ± 0.57	0.48 ± 0.45	−0.13 (−0.32; 0.05)	0.144
Glycaemia (mg/dL)	108.51 ± 52.26	92.11 ± 14.38	−16.40 (−30.98; −1.81)	0.028
HbA1c (%)	7.10 ± 1.63	6.29 ± 0.92	−0.81 (−1.31; −0.30)	0.004
Insulin (mcU/mL)	15.54 ± 8.65	12.77 ± 7.85	−2.77 (−5.12; −0.41)	0.023
HOMA-IR	4.03 ± 2.83	3.08 ± 1.92	−0.95 (−1.75; −0.15)	0.022
Uricemia (mg/dL)	6.01 ± 1.69	6.24 ± 1.90	+0.23 (−0.05; 0.52)	0.108
BUN (mg/dL)	39.75 ± 14.13	40.24 ± 16.66	+0.49 (−1.80; 2.78)	0.669
Creatinine (mg/dL)	0.92 ± 0.27	0.94 ± 0.32	+0.02 (−0.02; 0.06)	0.402
Sodium (Na, mmol/L)	138.96 ± 2.07	140.29 ± 2.24	+1.33 (0.56; 2.10)	0.001
Potassium (K, mmol/L)	4.35 ± 0.35	4.28 ± 0.46	−0.07 (−0.17; 0.03)	0.144
Chloride (Cl, mmol/L)	102.73 ± 2.53	103.37 ± 3.73	+0.64 (0.44; 1.71)	0.242
Calcium (mg/dL)	9.24 ± 0.57	9.36 ± 0.54	+0.12 (−0.03; 0.29)	0.119
Total cholesterol (mg/dL)	182.61 ± 36.07	161.29 ± 35.72	−21.32 (−30.67; −11.97)	0.001
HDL (mg/dL)	45.88 ± 10.22	40.08 ± 8.42	−5.80 (−7.38; −4.23)	0.001
Triglycerides (mg/dL)	160.77 ± 71.31	121.70 ± 39.41	−39.07 (−53.29; −24.85)	0.001
LDL (mg/dL)	108.88 ± 32.24	96.78 ± 32.01	−12.10 (−18.53; −5.67)	0.001
Apo A (mg/dL)	139.23 ± 23.69	120.51 ± 20.00	−18.72 (−23.61; −13.83)	0.001
Apo B (mg/dL)	100.06 ± 25.98	84.74 ± 21.94	−15.32 (−20.52; −10.12)	0.001
AST (IU/L)	22.28 ± 11.13	20.35 ± 11.26	−1.93 (−4.78; 0.93)	0.182
ALT (IU/L)	33.58 ± 22.98	33.06 ± 27.44	−0.52 (−6.62; 5.58)	0.856
GGT (U/L)	32.49 ± 20.28	21.96 ± 14.88	−10.53 (−15.44; −5.62)	0.001
Prealbumin (mg/dL)	25.25 ± 4.89	23.48 ± 4.80	−1.77 (−2.85; −0.69)	0.002
Phosphatase (U/L)	65.47 ± 17.70	62.23 ± 18.02	−3.25 (−6.69; 0.47)	0.086
Total bilirubin (mg/dL)	0.68 ± 0.26	0.58 ± 0.25	−0.10 (−0.15; −0.05)	0.001
Lipase (U/L)	23.02 ± 11.64	27.65 ± 14.87	+4.63 (1.97; 7.28)	0.001
Amylase (U/L)	49.92 ± 19.57	45.15 ± 19.99	−4.77 (−7.21; −2.32)	0.001
Homocysteine (μmol/L)	16.05 ± 4.99	14.71 ± 5.92	−1.34 (−5.47; 2.79)	0.419
Total proteins (g/dL)	6.75 ± 0.46	6.60 ± 0.45	−0.15 (−0.27; −0.04)	0.008
Albumin (%)	58.87 ± 4.11	59.08 ± 4.25	+0.21 (−0.50; 0.91)	0.561
Albumin (g/dL)	3.97 ± 0.34	3.89 ± 0.37	−0.08 (−0.16; 0.01)	0.094
Alpha-2 globulin (%)	10.26 ± 1.79	10.40 ± 1.84	+0.14 (0.02; 0.25)	0.022
Alpha-1 globulin (%)	4.05 ± 0.59	3.99 ± 0.58	−0.06 (−0.41; 0.29)	0.734
Beta globulin (%)	11.99 ± 1.60	11.80 ± 1.84	−0.19 (−0.47; 0.10)	0.188
Gamma globulin (%)	14.80 ± 2.85	14.89 ± 2.78	+0.09 (−0.20; 0.38)	0.517
Leukocytes (k/μL)	7.12 ± 1.97	6.47 ± 1.99	−0.65 (−0.97; −0.33)	0.001
Lymphocytes (n)	2.39 ± 0.90	2.34 ± 0.87	−0.05 (−0.19; 0.09)	0.488
Lymphocytes (%)	32.45 ± 7.14	35.76 ± 8.83	+3.31 (1.69; 4.93)	0.001
Erythrocytes (M/μL)	4.68 ± 0.46	4.63 ± 0.44	−0.05 (−0.11; 0.02)	0.159
Hemoglobin (g/dL)	13.70 ± 1.41	13.55 ± 1.30	−0.15 (−0.34; 0.04)	0.130
Hematocrit (%)	41.17 ± 3.60	40.89 ± 3.45	−0.28 (−0.88; 0.32)	0.359
Mean corpuscular volume (fL)	87.90 ± 4.75	88.15 ± 4.38	+0.25 (−0.33; 0.82)	0.396
Platelets (k/μL)	247.77 ± 247.77	236.58 ± 55.79	−11.19 (−21.37; −1.00)	0.032

* In bold: value with p < 0.05.

4. Discussion

This study demonstrates the effectiveness of a multidisciplinary residential program (MRP) in improving parameters associated with both obesity and sarcopenia in a cohort of hospitalized obese individuals. The observed improvements in body composition, metabolic markers, and physical

performance underscore the potential clinical impact of an integrated, team-based approach to managing patients at risk for sarcopenic obesity.

Notably, the significant increase in SPPB scores indicates a marked enhancement in overall physical performance, particularly in balance, gait speed, and lower limb strength. These domains are essential in maintaining autonomy and reducing the risk of falls and functional decline in obese patients. Although the improvement in handgrip strength was not statistically significant, the trend remains clinically relevant. These findings align with the work of Paddon-Jones, who highlighted a direct correlation between muscle strength and functional independence in daily living activities [22].

The significant reductions observed in total body weight, fat mass, visceral adipose tissue (VAT), and BMI with relative preservation of lean mass confirm the efficacy of the intervention in promoting qualitative weight loss. These changes were accompanied by improvements in waist, hip, arm, and calf circumferences. Such results are consistent with Villareal et al., who demonstrated that the combination of weight loss and structured physical activity has superior benefits in improving function and reducing frailty compared to either intervention alone [23].

Of particular interest is the significant reduction in VAT and the parallel improvement in insulin resistance, as measured by HOMA-IR. These findings corroborate those of Huang et al. [24], suggesting that targeted reductions in VAT through comprehensive interventions may attenuate systemic inflammation and decrease the risk of cardiometabolic complications. From a biomolecular perspective, the reduction in VAT could downregulate the expression of proinflammatory cytokines (such as TNF- α and IL-6), mitigate oxidative stress, and improve mitochondrial function in skeletal muscle, which are all critical mechanisms implicated in the pathogenesis of sarcopenic obesity [3].

The favorable changes in glycemic parameters—including reductions in fasting glucose, HbA1c, and insulin levels—further support the metabolic benefits of the MRP. These outcomes highlight the critical role of insulin resistance in muscle degradation and the potential for multidimensional interventions to preserve muscle integrity through metabolic modulation, as reported by Hong and Choi [3]. Improved insulin sensitivity may enhance Akt/mTOR signaling pathways, essential for muscle protein synthesis, and suppress the ubiquitin-proteasome pathway responsible for muscle breakdown.

Additionally, significant improvements in lipid parameters—including reductions in total cholesterol, LDL cholesterol, triglycerides, apolipoprotein A, and apolipoprotein B—further reinforce the cardiovascular and metabolic value of the intervention. The reduction in γ -glutamyl transferase (gGT) and bilirubin levels may also serve as indirect markers of improved hepatic and muscle function, as supported by previous studies linking elevated gGT to impaired muscle quality and sarcopenic outcomes [25,26].

Although the increase in vitamin B12 levels did not reach statistical significance, this trend remains clinically meaningful, given that low vitamin B12 status has been associated with frailty and impaired physical performance in aging populations [27]. Vitamin B12 is a critical cofactor in homocysteine metabolism and may play a role in maintaining neuromuscular integrity.

Taken together, these findings emphasize the strength of a multidisciplinary hospital-based approach in addressing the multifactorial nature of sarcopenic obesity. By integrating nutritional, physical, and psychological interventions under coordinated medical supervision, this model demonstrates superior potential compared to fragmented or single-focus interventions. The hospital setting further facilitates individualized monitoring, real-time adjustment of therapeutic strategies, and a higher level of adherence and safety.

However, the present study has several limitations that should be acknowledged. Firstly, the open-label design without a control group limits the ability to attribute observed improvements solely to the intervention, as placebo effects and regression to the mean cannot be excluded. Secondly, the relatively small sample size and short duration of follow-up prevent generalization to broader populations or assessment of long-term sustainability. Finally, the lack of mechanistic biomarkers (e.g., inflammatory cytokines, muscle-specific molecular markers) limits the exploration of the biological underpinnings of the observed effects.

Future clinical applications of this work could include the implementation of similar multidisciplinary programs in community or outpatient settings, with adaptations for scalability and cost-effectiveness. Moreover, future randomized controlled trials with longer follow-up and mechanistic assessments are warranted to strengthen causal inference and to delineate the biological pathways mediating the benefits of integrated interventions in sarcopenic obesity.

In conclusion, the current findings underscore the clinical utility of multidisciplinary rehabilitation in improving body composition, functional capacity, and metabolic health in obese individuals at risk for sarcopenia. This comprehensive approach should be considered a preferred strategy in the prevention and management of sarcopenic obesity within institutional care settings.

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