L-Carnitine Supplementation could improve Clinical Symptom but not effect on C- reactive Protein and Malondialdehyde in Obese Women with Knee Osteoarthritis: A Double Blind Randomized Controlled Trial

FARNAZ BAGHBAN
Shahid Sadoughi University of Medical Sciences and Health Services
Mahdieh Hosseinzadeh (hoseinzade.mahdie@gmail.com)
Shahid Sadoughi University of Medical Sciences and Health Services Yazd Research and Clinical Centre for Infertility https://orcid.org/0000-0001-7482-2494
Hassan Mozaffari-Khosravi
Shahid Sadoughi University of Medical Sciences and Health Services
Ali Dehghan
Shahid Sadoughi University of Medical Sciences and Health Services
Hossein Fallahzadeh
Shahid Sadoughi University of Medical Sciences and Health Services

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Abstract

Backgrounds:

L-carnitine decreases oxidation and inflammation by reducing the fatty acid in plasma and using oxygen in ATP synthesis. So, knee osteoarthritis (KOA) can be improved by reducing apoptotic chondrocytes. The aim of this study was to compare the effect of L-carnitine supplementation along with low calorie diet and low calorie diet alone on improvement of KOA among obese women. In other words, we aimed to investigate the additional effect of L- carnitine on improvement of KOA among obese women who received low calorie diet.

Methods

76 obese women with KOA were randomly assigned into two low-calorie diet groups; the first one received the 1000 mg L-carnitine (LCG) and with the second group took the placebo (PLG) (n = 38). Anthropometry indices, body composition, lipid profile, C- reactive Protein (CRP), Malondialdehyde (MDA), and the Western Ontario and McMaster Universities Arthritis Index (WOMAC) WOMAC index were assessed at the baseline and after 12 weeks.

Results: At the end of the study, the LCG showed significant improvements compared with the PLG in the total WOMAC scores (17.41 ± 9.81 compared with 23.50 ± 12.02 (P = 0.024)); physical function (11.15 ± 6. 56 compared with 15.61 ± 8.17 (P = 0.014)); BMI (-1.21 ± 0.84 kg/m^2 compared with − 0.79 ± 0.70 kg/m^2 (P = 0.027)). However, MDA, total cholesterol, fat and visceral fat mass decreased only in LCG significantly, but not in the PLG. Other variables did not have any significant changes.

Conclusion: Oral administration of 1000 mg L-carnitine in 12 weeks could improve WOMAC score and BMI, but not affect CRP, MDA, and lipid profile. Further trials with higher doses and longer durations, are required to verify our results.

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Background

Osteoarthritis (OA), as one of the most common kinds of arthritis has been described as a progressive degenerative joint disease [1]. Knee osteoarthritis (KOA) causes chronic pain, disability, and morbidity that imposes enormous burden on health and social care systems globally [2]. Age, obesity, gender (women) and genetic were reported as the most important risk factors for the development of KOA [3].

Higher oxidative stress contributes to the pathogenesis of knee osteoarthritis**.** Oxidative stress is produced from the presence of lipid peroxidation products, such as oxidized low-density lipoprotein (ox-LDL) in all cell types such as chondrocytes. This oxidizer results in pain and physical disability in OA, since it breaks down the matrix components and chondrocytes and degenerates cartilage [4]. Malondialdehyde (MDA) is also a specific aldehydic product of lipid peroxidation that elevated in plasma

and synovial fluid of OA patients [5]. Also, recent studies have suggested that local inflammation has an important role in the pathogenesisand progression of OA [6]. C-reactive protein (CRP), as a systemic biomarker for inflammation increases in OA patient [7].

L-carnitine (4-N-trimethylammonium-3-hydroxybutyric acid) plays an important role in transferring the long-chain fatty acids from the inner mitochondrial membrane to the peripheral tissues [9]. L-carnitine, by reducing the fatty acid in plasma and using oxygen in synthesis of ATP, can decrease oxidation and inflammation [8]. So, by reduction of apoptotic chondrocytes in OA cartilage, KOA can be improved [10]. Some studies showed decrease of carnitine concentration in blood and tissues in the rheumatoid arthritis [11]. Animal study also demonstrated that L-carnitine can reduce KOA symptoms [12]. Earlier studies showed that L-carnitine can significantly reduce CRP and MDA in coronary [29, 30] and hemodialysis patients [31]. Meta-analysis also showed that L-carnitine supplementation significantly reduced the levels of CRP [13], cholesterol and low-density lipoprotein cholesterol (LDL-C) in diabetic patients [14]. Recent study also reported that 750 mg of L-carnitine supplementation had no effect on serum lipid profile and hs-CRP in women with KOA [15, 16]. However, a study on hemodialysis patients reported no significant effect on oxidative stress [32]. The results of studies over the effect of L-carnitine on lipid profile, CRP, and some other oxidative indices are controversial. Furthermore, rare studies evaluated the effect of this supplementation on KOA. So, we targeted at evaluating the effect of oral L-carnitine supplementation on CRP, MDA, lipid profile, WOMAC index, as well as anthropometry and body composition measures in obese women with KOA.

Moreover, it was confirmed that low calorie diet had a significant effect on KOA [17, 24] [17]. Therefore, it seems that both L-carnitine supplementation and weight loss diet are beneficial for OA, but previous studies were conducted on the effect of L-carnitine supplementation and did not include the received calories. The aim of this study was to compare the effect of L-carnitine supplementation along with low calorie diet and low-calorie diet alone on improvement of KOA among obese women. In other words, we aimed to investigate the additional effect of L-carnitine on improvement of KOA among obese women who received low calorie diet.

Methods

Study participant

This double blind parallel randomized controlled trial was conducted according to the CONSORT guidelines. Participants included 76 KOA women recruited from the Khatam Al-Anbia Super Specialty clinic at the Department of Rheumatology, Yazd, Iran. To be eligible for inclusion, women had to be over 45 years old, have a body mass index (BMI) in the range of 25-35 kg/m^2, and have diagnosed KOA according to the clinical classification of KOA (18). The exclusion criteria were: having former or planned knee-joint replacement, being under pharmacologic treatment for obesity, having any history or active presence of other rheumatic diseases, using any nonsteroidal anti-inflammatory drug (NSAID),

consuming ≤ 20 percent of the supplements, consuming multivitamin, minerals, or other nutritional supplements, and having severe liver, kidney, or heart diseases.

Randomization and intervention

This study was a double blind randomized controlled trial. Patients, who met the study criteria, were assigned to L-carnitine group (LCG) or placebo group (PLG) through randomization lists made by a computerized random-number generator and simple randomization process in a ratio of 1:1. The L-carnitine group received 1 g/d L-carnitine daily for 12 weeks. The placebo group received a placebo according to the same regimen and the same duration. The placebo pills contained inactive ingredients with no therapeutic activity and had an identical appearance. These tablets were produced by Karen Pharmaceutical & Nutrilife Co., Yazd, Iran. As a double-blind study, the bottles were labeled A and B respectively for the placebo and drug by the factory but neither the patients nor the research team were aware of the codes. Every month, patients received a bottle of tablet containing 30 tablets. Compliance with the medication was monitored by the research personnel using pill counts and patients' self-reporting. Participants who did not consume more than 30 percent of their supplements were eliminated from the analysis. All participants followed a low- calorie diet. A registered dietitian estimated the energy expenditure for each patient through Harris-Benedict formula using the individual activity factor [19]. The recommended composition of the dietary regimen was 50% to 60% carbohydrates, 15% to 20% proteins, and less than 30% total fat. Initially, a dietitian completed the 3-day food recall for all participants and visited patients every month to check their compliance with the diet according to the patients' feedback and 24-hour food recall. At the baseline, physical activity during the past week was assessed using the long version of International Physical Activity Questionnaire (IPAQ). Patients were also asked not to change their level of activity during the study.

Outcome Measurements

The following variables were assessed at the baseline and 12 weeks after the start of treatment: primary outcome included WOMAC, CRP and MDA. Secondary outcome was LDL-C, total cholesterol (TC), high density lipoprotein-cholesterol (HDL-C), triglycerides (TG), BMI, fat mass, free fat mass, as well as waist and hip circumference.

In order to conduct the laboratory tests, 5 mL of venous blood samples was obtained after the patients had fasted for 8 hours overnight. Immediately after the centrifugation (3000g, 10 min), serum samples were produced from the collected blood samples. They were then frozen at -20 ºC, stored at -70 ºC, and measured at the same time. The total TC, HDL-C, and TG were later measured using Pars Ammon kit (Iran). LDL-C was then calculated using Friedewald's equation (20). Serum CRP and serum MDA concentrations were measured through enzyme-linked immunosorbent assay kits and thiobarbituric acid reactive substances Zellbio kit (Germany), respectively.

The patients' weights were also recorded on a portable digital scale (Omeron BF511, Japan) to the nearest 0.1 kg. Participants were in light clothes and stood on the scale without help. Fat mass and free

fat mass were measured with this scale. Further, height was measured in standing position using an audiometer fixed on a straight wall to the nearest 0.1 cm. Waist circumference (WC) was recorded to the nearest 1 centimeter using non-stretch plastic tape placed midway between iliac crest and lowest rib while participants were in standing position. Hip circumference was also measured over the largest part of buttocks with the accuracy of 1 cm. BMI was also calculated as weight (kg) divided by height squared (m^2). To assess the clinical symptoms Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) were used. Patients filled out the Persian version of WOMAC index [21] that consists of 24 questions (pain, stiffness, and physical functional). Items should be answered on a Likert scale: none (0), mild (1), moderate (2), severe (3), or extreme (4). For physical activity, the Persian version of IPAQ was applied [22]. The continuous score shows the weekly energy expenditure expressed in MET-min/week (metabolic equivalent-minutes). Individuals were classified into three categories of 'inactive', 'moderately active', and 'highly active' using the categorical classification.

Sample size

Power calculations were conducted based on pain scores of 72 women with KOA who participated in a previous trial [23]. On the basis of an assumed 10% dropout rate, we estimated that a total sample size of 76 patients (38 patients per group) would provide 80% of the power to detect a 2.6 pain score difference between the PLG and LCG.

Statistical analysis

Statistical analyses were carried out using SPSS (version16). The normal distribution of variables was tested by Kolmogorov-Smirnov test. Differences in patients' anthropometrics, WOMAC scores, and hematological measurement data between the PLG and LCG were analyzed by Student's t-test or the Mann–Whitney rank sum test for parametric and non-parametric continuous variables, respectively. The paired t-test or Wilcoxon signed rank test was used to analyze the data within each group before (baseline) and after the intervention (week 12). Analysis of covariance (ANCOVA) was used to identify the differences between the two groups after adjusting for change weight. Results were considered statistically significant at $P < 0.05$. Normal data were indicated by means ± standard deviations (SD) and the non-normal scores were presented in median and inter quartile range (IQR). The dietary information was analyzed with N4 software (Nutritionist: version 4.0; Tinuviel Software, Warrington, United Kingdom).

Efficacy and tolerability assessment

For the safety, all participants were interviewed every month for any signs of L-carnitine toxicity or other adverse problems considering the diet, including serious illnesses or hospitalizations.

Results

Study participant characteristics

The sampling and trial profiles are summarized in Figure 1. The baseline characteristics of these patients are shown in Table 1. Participants included 76 women with a mean age of 54.73 ± 7.41, BMI of 32.65 ± 5.60 kg/m^2, and percentage of body fat of 44.42 ± 5.90%. Median and IQR baseline of CRP was 3.52 ± 4.33 mg/dL. Anthropometric, lipid profile, CRP measures, physical activity, and diet composition such as total energy, protein, fat, and carbohydrate intake, as well as education and occupational status did not differ between groups at the baseline of the study (P > 0.05).

Blood lipids and lipoproteins, CRP and MDA

No significant decrease was observed in the LDL-C, HDL-C, TG and CRP concentrations in either the LCG or PLG compared with the baseline. In the LCG, however, there was a significant decrease in TC (226.74 ± 50.55 to 212.24 ± 41.39 mg/dL, P= 0.021) and MDA (33.32 ± 36.09 to 26.14 ± 22.22 mg/dL, P= 0.035) after treatment but not in PLG. No significant difference was observed between the LCG and the PLG in lipid profiles, CRP, and MDA concentrations (Table 2).

Anthropometry and body composition

Compared with the baseline, weight, BMI, as well as the waist and hip circumference decreased significantly in both groups after 12 weeks of intervention (P= 0.001) (Table 3). Furthermore, results showed significant reduction in visceral fat and fat mass in LCG (P = 0.001), but not in PLG. However, no significant differences were observed between the LCG and the PLG. The mean of weight change was 4.49% in the LCG, while it was 2.43% in the PLG (P = 0.05). changes of BMI were significant between the two groups (-1.21 ± 0.84 kg/m^2 compared with -0.79 ± 0.70 kg/m^2; P = 0.027).

WOMAC index

Compared with the baseline results, decrease of pain, stiffness, physical function, and total scores were significant in both groups after 12 weeks of treatment (P= 0.001). Physical function improved significantly (P= 0.014) in the LCG with a mean of 11.15 ± 6.56 compared with PLG with a mean of 15.6 ± 8.2. Furthermore, the patients in the LCG had significantly lower total scores (17.41± 9.81 versus 23.50 ± 12.02) than those in the PLG (P= 0.024). However, mean of changes were not significant (Table 2).

Tolerability

Both L-carnitine and placebo were well tolerated in all patients. In the group treated with L-carnitine, one patient complained of skin dryness and two complained of slight stomachache. In the PLG, two patients complained of skin dryness and three complained of stomachache.

Discussion

This randomized placebo-controlled trial examined the effect of oral l-carnitine (1000 mg/d) consumption compared with placebo in combination with low calorie diet for 12 weeks in overweight or obese women with KOA. Results showed significant improvements in physical function, total score WOMAC, BMI, and

weight. However, MDA, total cholesterol, fat and visceral fat mass decreased only in LCG significantly, but not in the PLG. CRP and other lipid profiles did not change significantly.

One of the important risk factors of KOA is obesity. Studies confirmed that weight loss intervention can improve pain and physical function [17, 24]. Therefore, it seems that both L-carnitine supplementation and weight loss diet are beneficial for OA. To the best of our knowledge, this is the first study investigating the effect of L-carnitine supplementation along with low calorie diet compare low calorie diet alone on improvement of KOA among obese women.

This study showed a significant decrease in BMI and weight in the LCG. In agreement with our findings, a meta-analysis reported that L-carnitine might be effective for weight loss in adults [25]. Another study that supported this hypothesis indicated that L-carnitine can decrease weight, BMI, as well as waist and hip circumference [26]. In other words, studies assessing the effect of L-carnitine on other diseases didn't report significant effects on anthropometry measures [14, 27]. In our study fat mass and visceral fat mass were not differences significantly between two groups. Similar to our results, no significant change was observed in fat mass of healthy people who consumed L-carnitine with exercise for 8 weeks [27]. On the contrary, 2000 mg L-carnitine among with hypo-caloric diet could reduce fat mass in diabetic patients [28]. The doses of L-carnitine supplementation in the mentioned study were two times higher than those of our study that might explain the discrepancy between results. L-carnitine leads to weight loss by oxidizing fat and decreasing the serum levels of leptin. The serum level of leptin is proportional to the body fat mass. Thus, the level of leptin drops by decrease of the adipose tissue mass [29]. Moreover, obesity is a chronic inflammatory disease that causes lipid peroxidation by abnormal production of pro-inflammatory factors such as IL-6 and CRP as well as release of free fatty acids from adipose tissue [4]. This study concluded that, no significant differences were observed in CRP and MDA between the LCG and the PLG. In consistent with our results, 750 mg/d of L-carnitine supplementation on women with KOA didn't show any significant change on MDA and hs-CRP [15,16]. Confirming this issue, supplementation with oral consumption of 1000 mg/d L-carnitine can significantly reduce CRP and MDA levels in coronary patients [30, 31]. These results were similar to another study in which propionil L-carnitine was injected into hemodialysis patients [32]. Furthermore, a study on hemodialysis patients with hyper lipo-proteinemia reported that 1000 mg/d of oral L-carnitine could reduce inflammation but didn't affect oxidative stress [33]. Moreover, a meta-analysis confirmed that L-carnitine can reduce CRP [13]. In comparison with the current research, CRP reduction in this study may be due to injection of L-carnitine and longer duration of the study.

Our results, which are consistent with the literature [15, 34], showed that oral administration of L-carnitine didn't lead to any significant difference in lipid profile. In consistent with the present findings, two meta-analyses indicated that L-carnitine decreases serum LDL-C [35] and cholesterol [14]. L-carnitine is an essential cofactor that helps transfer of fatty acids into mitochondria and causes incorporation of long-chain fatty acids into the β oxidation cycle to produce acetyl-CoA. L-carnitine helps oxygen entrance into the tri carboxylic acid (TCA) cycle to synthesize ATP. This process decreases the concentration of oxygen and reduces formation of reactive oxygen species [36].

However, our study showed significant improvement in physical function and total score in WOMAC questioner. In the same line with our results, one study indicated 750 mg of oral L-carnitine supplementation have significant improvement in pain intensity and patients' global assessment of disease status on KOA [23]. A review article indicate that more than 2 g L-carnitine can reduce stiffness in movement, the pain after prolonged movement, and disturbed sleep due to the pain [37]. L-carnitine may have an additional effect on the improvement of KOA. In other word, L-carnitine may have a greater impact on the clinical symptoms of KOA by decreasing the fat mass and causing a significant reduction in WOMAC score. It seems this effect are independent of inflammatory and lipid profile modification [15,23]. After adjusting for the weight reduction during the study, the results remained significant, which indicates that these effects were independent of weight change. This may show the direct effect of the L-carnitine.

The strength of our study was comparing the effect of L-carnitine supplementation along with low calorie diet on improvement of KOA among obese women for the first time; their adherence to the diet was also monitored every month. Moreover, L-carnitine appeared to be well tolerated by participants. The limitation of the present study was that we did not evaluate the serum L-carnitine levels. Long-term studies with higher doses of L-carnitine and measurement of other indicators such as inflammation factors and MMP would help to clarify further this realm.

Conclusions

According to the findings, Oral administration of 1000 mg L-carnitine in 12 weeks could improve WOMAC score and BMI, but not affect CRP, MDA, and lipid profile. Further trials with higher doses and longer durations, are required to verify our results.

Abbreviations

CRP, C- reactive protein; MDA, malondialdehyde; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index; LCG, L-carnitine group; PLG, placebo group; OA, Osteoarthritis; KOA, Knee Osteoarthritis; ROS, reactive oxygen species; LDL-C, low density lipoprotein cholesterol; BMI, body mass index; NSAID, nonsteroidal anti-inflammatory drug; IPAQ, International Physical Activity Questionnaire; TC, total cholesterol; HDL-C, high density lipoprotein-cholesterol; TG, triglycerides

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics committee of The Shahid Sadoughi University of Medical Sciences, School of Public Health Iran IR.SSU.SPH.REC.1395.45. All patients signed and approved the informed consent. This study is also registered with the code of IRCT2017011932026N2.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

M H created the study concept and design and edited the manuscript, F B collected data, and prepared the manuscript; H F provided statistical analyses, A D managed subjects and edited the manuscript; H M-K was involved in the design of the study, and edited the manuscript.

Data availability statements: The datasets generated during and analyzed during the current study are available from the corresponding author on reasonable request.

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Authors' Information:

Farnaz Baghban[1], Mahdieh Hosseinzadeh [1*], Hassan Mozaffari-Khosravi[1], Ali Dehghan[2], Hossein Fallahzadeh[3]

[1] Department of Nutrition, School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

[2] Rheumatology, Department of Internal Medicine, Shahid Sadoughi Hospital, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

[3] Department of Biostatistics and Epidemiology, Research Center of Prevention and Epidemiology of Non-Communicable Disease, Faculty of Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

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Tables

TABLE. 1. Selected baseline characteristics of study participants

	L-carnitine group (n = 34)	Placebo group (n = 36)	p[1]
Age (y)	55.01± 7.12	54.43 ± 7.80	0.737
BMI (kg/m2)	33.03± 6.67	32.10 ±4.29	0.471
Height(m)	1.55 ± 0.10	1.56 ± 0.55	0.481
Weight(Kg)	78.69 ± 10.86	78.90 ± 12.17	0.937
Waist circumference (Cm)	105.13 ± 9.04	105.92 ± 10.57	0.728
Hip circumference (Cm)	115.63 ± 9.37	117.87 ± 10.56	0.332
Physical activity†	548.01 ± 1131.4	540.01 ± 620.25	0.432
Energy (kcal)	1426.21 ± 344.91	1377.72 ± 406.44	0.577
Carbohydrate (g)	174.76 ± 37.21	177.38 ± 66.75	0.834
Fat (g)	60.83 ± 24.77	56.19 ± 27.55	0.443
Protein (g)	50.52 ± 19.34[2]	50.63 ± 22.17[2]	0.455
Education			
Illiterate	3 (7.9)	1 (2.6)	0.29
Elementary school graduate	18 (47.4)	20 (52.6)	
Middle and high school graduate	13 (34.2)	14 (36.8)	
University graduate	4 (10.5)	3 (7.9)	
Occupational status			0.42
housewife	30 (78.9)	34 (89.5)	
employee	3 (13.9)	2 (5.3)	
Retired	5 (7.2)	2 (5.3)	

Mean ± SD (all such values except protein).

[1] Determined with the use of independent samples t tests for differences at baseline between L-carnitine and placebo groups

[2] Median and IQR (for no normally distributed variables).

TABLE. 2. Lipids, CRP concentration and WOMAC score changes in patients treated with placebo and L-carnitine before and after 12 weeks of treatment

		L-carnitine group (n = 34)	P[2]	Placebo group (n = 36)	P[3]	P[4]
L cholesterol g/dL)	before	136.13 ± 43.71	0.240	126.92 ± 35.33	0.380	0.316
	after	130.97 ±29.46		122.94 ± 44.18		0.372
	change	-7.68 ± 37.07		-3.05 ± 20.59		0.525
DL cholesterol g/dL)	before	64.97 ± 26.35	0.434	64.08 ± 19.60	0.413	0.867
	after	62.26 ± 11.35		67.94 ± 15.24		0.083
	change	-3.85 ± 8.35		2.5 ± 18.12		0.265
glycerides g/dL)	before	185.53 ± 92.50	0.23	193.63 ± 80.05	0.888	0.684
	after	169.15 ±73.81		193.44 ± 78.60		0.188
	change	-20.50 ± 97.72		1.50 ± 63.18		0.265
tal cholesterol g/dL)	before	226.74 ± 50.55	0.021	220.71 ± 42.70	0.259	0576
	after	212.24 ± 41.39		210.89± 61.00		0.914
	change	-18.47 ± 44.33		-8.94 ± 46.77		0.385
P(mg/dL)	before	3.40 ± 2.51[2]	0.142	3.9 ± 3.92 [1]	0.682	0.618

	after	3.00 ± 3.05		4.40 ±5.90		0.969
	change	-0.6 ± 2.52		0.09 ± 3.72		0.383
)A(mg/dL)	Before*	33.3 ± 36.09	0.03	24.2 ± 14.7	0.36	0.36
	After	26.1 ± 22.22		21.03 ± 11.4		0.76
	change	-5.22 ± 24.98		-2.08 ± 13.5		0.22
Pain index	before	7.34 ± 3.4	0.001	8.63 ± 3.63	0.001	0.322
	after	4.71 ± 2.65		5.89 ± 3.54		0.119
	change	-2.91 ± 2.03		-2.89 ± 2.21		0.964
Stiffness	before	2.28 ± 1.72	0.001	3.02 ± 2.18	0.001	0.107
	after	1.56 ± 1.50		2.00 ± 1.74		0.261
	change	-1 ± 1.00		-1.11 ± 1.43		0.223
Physical function	before	17.82 ± 7.75	0.001	20.63 ± 8.77	0.001	0.142
	after	11.15 ± 6.56		15.61 ± 8.17		0.014
	change	-6.97 ± 3.95		-5.11 ± 5.29		0.102

Global Score	before	27.45 ± 11.44	0.001	32.29 ± 12.93	0.001	0.088
	after	17.41± 9.81				
	change	-10.59 ± 5.58		23.50 ± 12.02		0.024
				-9.11 ± 6.44		0.342

Mean ± SD (all such values).

[1] Median; IQR (all such values for non-normally distributed variables).

[2] Determined with the use of paired Student's t tests for differences between baseline and follow-up in the L-carnitine group

[3] Determined with the use of paired Student's t tests for differences between baseline and follow-up in the placebo group.

[4] Determined with the use of independent samples t tests between L-carnitine and placebo groups.

TABLE. 3. Anthropometry changes in patients treated with placebo and L-carnitine before and after 12 weeks of treatment

		L-carnitine group (n = 34)	P[1]	Placebo group (n = 36)	P[2]	P[3]
ght(Kg)	before	78.7 ± 10.86	0.001	78.91 ± 12.18	0.001	0.937
	after	75.19 ± 10.84		76.99 ± 12.70		0.527
	change	-2.76 ± 1.69		-1.95 ± 1.73		0.052
(kg/m2)	before	33.04 ± 6.67	0.001	32.10 ± 4.30	0.001	0.471
	after	31.87 ± 6.56		31.29 ± 4.56		0.669
	change	-1.21 ± 0.84		-0.79 ± 0.70		0.027
st umference)	before	105.13 ± 9.04	0.001	105.92 ± 10.57	0.001	0.728
	after	99.45 ± 11.72		102.39 ± 10.1		0.262
	change	-5.65 ± 5.85		-3.64 ± 3.37		0.081
circumference)	before	115.63 ± 9.37	0.001	117.87 ± 10.56	0.001	0.332
	after	108.44 ± 9.67		112.19 ± 10.58		0.127
	change	-6.82 ± 3.56		-5.64 ± 4.07		0.201
e fat mass (%)	before	24.00 ± 1.74	0.8	24.59± 2.64	0.531	0.253
	after	24.11 ±1.75		23.99 ± 3.78		0.865
	change	0.04 ± 0.09		-0.71 ± 3.47		0.384
mass (%)	before	45.19 ± 4.85	0.03	43.63 ± 6.77	0.915	0.251
	after	44.07 ± 4.33		43.95 ± 5.66		0.921
	change	-0.71 ± 1.83		-0.3 ± 1.93		0.360
eral fat (%)	before	10.71 ± 1.95	0.001	10.47 ± 1.82	0.644	0.587
	after	10.24 ± 2.104		10.50 ± 1.89		0.581
	change	-0.41 ± 0.49		-0.55 ± 0.752		0.320

Mean ± SD (all such values).

[1] Determined with the use of paired Student's t tests for differences between baseline and follow-up in the L-carnitine group

[2] Determined with the use of paired Student's t tests for differences between baseline and follow-up in the placebo group.

[3] Determined with the use of independent samples t tests between L-carnitine and placebo groups

Figures

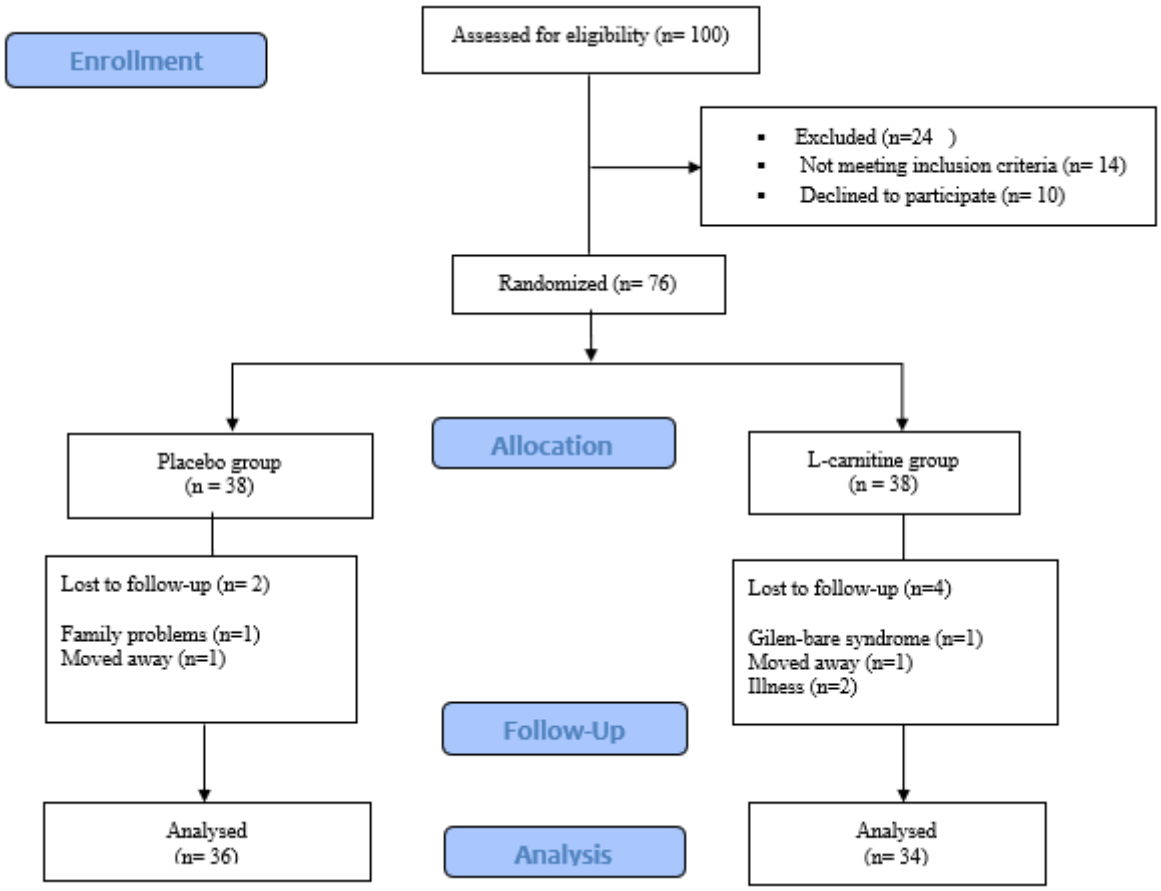

Figure 1

Participant flowchart showing numbers of participants who were recruited, were randomly assigned, dropped out, and were analyzed during the trial

Supplementary Files

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- CONSORT1ExtensionforAbstractsChecklist.doc