SJIF Impact Factor: 6.711

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 9.
Issue: 2
Page N. 13-18
Year: 2025

Original Article www.wjahr.com

L-CARNITINE FOR TREATMENT OF METABOLIC DYSFUNCTION-ASSOCIATED FATTY LIVER DISEASE

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Article Received date: 06 December 2024 **Article Revised date:** 26 December 2024 **Article Accepted date:** 16 January 2025

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ABSTRACT

Background: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a common condition of the liver that is increasing in prevalence globally, this disease is often caused by insulin resistance, stress, and mitochondrial failure. Effective pharmaceuticals for treating diseases are limited, this has led to a focus on L-carnitine as it could help to support the metabolism of fatty acids and reduce liver fat. Aim: the aims of these study to assess the effectiveness of L-carnitine supplementation for treatment for MAFLD in adult patients. **Method:** A blinded, randomized trial was conducted on 50 individuals with MAFLD. Patient categories into two groups, those who received L-carnitine and who received a placebo, for a period of three months. Baseline and post-treatment parameters included lipids profile, assessment of liver enzymes (ALT, AST), ultrasound imaging, waist circumference, and. Statistical analyses employed t-tests, chi-square tests, and p values, with a significant level of p less than 0.05 being used. **Results:** The study results showed that L-carnitine supplementation of 500mg twice daily for 3 months significantly improved liver ultrasound scores, reduced ALT and AST levels, and lowered total cholesterol, LDL, and triglyceride levels compared to the placebo group. Additionally, there was not significance improvement in waist circumference in the L-carnitine group. These findings support L-carnitine's potential benefits in managing MAFLD. Statistical significance was noted in most key measures, affirming the supplement's efficacy. **Conclusion:** L-carnitine supplementation has a significant effect in MAFLD patients, it may therefore be a beneficial supplement to therapy. Future planned investigations should explore the optimal dosing and treatment duration for the greatest effect.

KEYWORDS: L-carnitine, Metabolic, fatty liver disease.

INTRODUCTION

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a common liver disorder characterized by the accumulation of fat in liver cells in individuals who consume little to no alcohol. With its prevalence rising globally, MAFLD has become a major health concern, affecting approximately 25% of the adult population worldwide.[1] This condition encompasses a range of liver damage, from simple steatosis, or the build-up of fat in liver cells, to more severe forms, such as nonalcoholic steatohepatitis (NASH), which involves liver inflammation and cellular injury. Left untreated, MAFLD can progress to advanced fibrosis, cirrhosis, and hepatocellular carcinoma, posing significant risks to both morbidity and mortality.[2] The pathogenesis of MAFLD is complex and multifactorial, involving insulin resistance, oxidative stress, and mitochondrial dysfunction as key contributors. Lifestyle modifications, such as dietary changes and increased physical activity, are considered the cornerstone of MAFLD management. However, in many L-Cs, these interventions alone are insufficient to halt or reverse disease progression.[3] Consequently, there is a growing interest in exploring pharmacologic therapies that target the underlying mechanisms of MAFLD. Among these, L-carnitine, a naturally occurring compound involved in fatty acid metabolism, has shown promise as a potential therapeutic agent.[4] L-carnitine plays a critical role in energy metabolism by facilitating the transport of long-chain fatty acids into the mitochondria, where they are oxidized for energy production. This process not only aids in energy generation but also prevents the accumulation of lipids in cells, a phenomenon that is particularly relevant to MAFLD.[5] Beyond its metabolic role, L-carnitine has antioxidant properties that can mitigate oxidative stress—a known contributor to liver

cell damage in MAFLD. Furthermore, studies have suggested that L-carnitine may improve insulin sensitivity, thereby addressing one of the central metabolic dysfunctions associated with MAFLD.[6] Given these attributes, L-carnitine has garnered attention in recent years as a promising treatment option for MAFLD. Various clinical trials have explored its efficacy, with some studies demonstrating improvements in liver enzyme levels, reductions in liver fat content, and decreases in inflammatory markers among MAFLD patients.[7] Nonetheless, the therapeutic use of L-carnitine for MAFLD remains an area of active research, with ongoing studies seeking to clarify optimal dosages, treatment durations, and patient populations that may benefit most.[8] Aims of the study to evaluate the efficacy of L-carnitine supplementation in dose 500 mg twice a day conducted for three months duration of treatment, metabolic profile, liver enzyme, u\s imaging done for adult patients suffering with MAFLD.

METHOD

Double-blind, placebo study to assess the efficacy of L-carnitine as treatment for Metabolic dysfunction-associated fatty liver disease (MAFLD) in 25 adult patients versus 25 controlled group. Patients were recruited consecutively from the consultation clinic at the Gastrointestinal (GIT) Center in Al-Sader Teaching Hospital in Najaf. Eligibility criteria included a diagnosis of MAFLD by u\s done by specialist radiologist were study before and after treatment, as confirmed by metabolic markers, lipid profiles, and liver function tests. Key metabolic indicators assessed included HbA1c and lipid profiles (high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol), with additional evaluation of liver enzymes (AST, ALT). Participants were randomly assigned to two groups: The L-C group receiving L-carnitine supplementation and a placebo group receiving a placebo. Baseline measurements for all participants included, lipid profiles, steato tests (AST and ALT), and sonographic imaging. These measurements were repeated after three months to evaluate the treatment effects. Randomization ensured that confounding factors as diet, exercise, drugs, weight management were minimized, enhancing the reliability of comparisons between the two groups. And exclusion criteria alcoholic patient, as well as patients with liver disease (HBVS, HCV, Wilson, hemochromatosis), patients with chronic disease or drugs or herbal use. With SPSS 22, statistical analysis is performed using mean, median, and SD for continuous data, and frequency and percentage for categorical data, whereas chi-square is used to evaluate the association between categorical data variables. The T test is used to assess how the mean and median of continuous variables differ from one another. A P-value of 0.05 or less is regarded as significant.

RESULTS

Table 1 displays the age distribution of patient's alcoholic fatty liver disease. The majority of patients in both groups fall within the 30-39 age range, with 74.1% in the placebo group and 84.0% in the L-C group. Patients are aged ≥60, accounting for 7.4% in the placebo group and 12.0% in the L-C group.

Table 1: Distribution of patients in both groups according to age groups.

Age Groups (years)	Placebo	L-C
20-29	5 (18.5%)	1 (4.0%)
30-39	20 (74.1%)	21 (84.0%)
≥60	2 (7.4%)	3 (12.0%)

Table 2 shows the distribution of patients' BMI after treatment between the placebo and L-C groups. In the placebo group, 70.4% of patients were classified as obese, compared to 52.0% in the L-C group. Additionally, 7.4% of the placebo group had morbid obesity, while this was higher in the L-C group at 16.0%. There was no significant statistical difference between the groups, as indicated by a p-value of 0.5. compares the differences in mean waist circumference (WC). The mean WC after treatment in the placebo group was 108.18 cm, while in the L-C group, it was 102.44 cm, with no statistically significant difference (p-value = 0.9).

Table 2: Comparison between BMI after Treatment according to L-c and placebo group.

BMI After Treatment	Placebo	L-C	P-value
Normal	1 (3.7%)	2 (8.0%)	
Overweight	5 (18.5%)	6 (24.0%)	0.5
Obese	19 (70.4%)	13 (52.0%)	(not significant)
Morbid Obesity	2 (7.4%)	4 (16.0%)	

	Group	N	Mean (W.C.)	Std. Deviation	P-value
WC after	Placebo	27	108.18	20.87	0.9
	L-C	25	102.44	12.91	

Table 3 the results presents the results between ultrasound (US) results after treatment in the placebo and L-C groups. In the L-C group, 32.0% of patients showed normal US results, while none of the placebo group

patients had normal results. A significant difference was found between the groups, with 40.0% of the L-C group having a US grade of 1.00 compared to 7.4% in the placebo group (p-value 0.0001). Additionally, 70.4% of the placebo group had a US score of 2.00 compared to 24.0% in the L-C group.

Table 3: Comparison between US after Treatment according to L-c and placebo group.

Ultrasound After Treatment	Placebo	L-C	P-value
Normal	0 (0.0%)	8 (32.0%)	
1.00	2 (7.4%)	10 (40.0%)	0.0001
2.00	19 (70.4%)	6 (24.0%)	(significant)
3.00	6 (22.2%)	1 (4.0%)	

Table 4 compares the lipid profile (total cholesterol, LDL, HDL, and triglycerides) after treatment between the placebo and L-C groups. The mean total cholesterol (TC) was significantly higher in the placebo group (225.51 mg/dL) compared to the L-C group (182.68 mg/dL), with a p-value of 0.003. Similarly, LDL was higher in the placebo group (132.51 mg/dL) compared to the L-C group (110.16 mg/dL), with a significant p-value of 0.017. There was no significant difference in HDL levels between the two groups. Triglyceride (TG) levels were also significantly higher in the placebo group (207.25 mg/dL) compared to the L-C group (169.48 mg/dL), with a p-value of 0.003.

Table 4: Difference mean of TC (before and after), LDL (before and after), HDL (before and after), TG (before and after) in both groups.

	Group	N	Mean	Std. Deviation	P-value
TC	placebo	27	225.51	58.92	0.003
After	L-C	25	182.68	37.76	
LDL	placebo	27	132.51	29.99	0.017
After	L-C	25	110.16	35.52	
HDL	placebo	27	54.74	12.53	0.9
After	L-C	25	54.81	11.51	
TG	placebo	27	207.25	46.69	0.00**3**
After	L-C	25	169.48	40.81	

Table 5 compares the mean levels of ALT and AST after treatment between the placebo and L-C groups. The ALT levels were significantly higher in the placebo group (99.55 U/L) compared to the L-C group (37.96 U/L), with a p-value of 0.0001. Similarly, AST levels were higher in the placebo group (80.44 U/L) compared to the L-C group (33.12 U/L), also with a significant p-value of 0.0001.

Table 5: Difference mean of ALT (before and after), AST (before and after) in both groups.

	Group	N	Mean	Std. Deviation	P-value
ALT	placebo	27	99.55	27.79	0.0001
After	L-C	25	37.96	22.24	
AST	placebo	27	80.44	21.09	0.0001
After	L-C	25	33.12	19.71	

Table 6 shows the changes in waist circumference (WC) before and after treatment in the L-C group. The mean WC decreased from 109.60 cm before treatment to 102.44 cm after treatment, with a significant p-value of 0.0001. BMI levels also decreased from 33.8 before treatment to 31.8 after treatment, with a significant p-value of 0.0001.

Table 6: Difference mean of WC (before and after),), in L-Cs group.

	Mean	Std. Deviation	P-value
WC before	109.60	12.17	0.0001
WC after	102.44	12.91	
BMI before	33.8	5.25	0.000**1**
BMI after	31.8	5.38	

Table 7 presents the changes in lipid profile (total cholesterol, LDL, HDL, and triglycerides) before and after treatment in the L-C group. The mean total cholesterol decreased from 207.12 mg/dL before treatment to 182.68 mg/dL after treatment, with a significant p-value of 0.0001. LDL levels also decreased

significantly from 140.72 mg/dL to 110.16 mg/dL (p-value 0.0001). HDL increased from 44.73 mg/dL to 54.81 mg/dL (p-value 0.0001), and triglycerides decreased from 190.80 mg/dL to 169.48 mg/dL, with a p-value of 0.04.

Table 7: Difference mean of TC (before and after), LDL (before and after), HDL (before and after), TG (before and after) in L-Cs group.

	Mean	Std. Deviation	P-value
TC before TC after	207.12 182.68	46.85 37.76	0.0001
LDL before LDL after	140.72 110.16	47.52 35.52	0.0001
HDL before HDL after	44.73 54.81	10.69 11.51	0.0001
Tg before TG after	190.80 169.48	66.43 40.81	0.**04**

Table 8 presents the changes in ALT and AST levels before and after treatment in the L-C group. The mean ALT decreased significantly from 85.30 U/L before treatment to 37.96 U/L after treatment, with a p-value of 0.0001. Similarly, AST levels also decreased from 66.07 U/L to 33.12 U/L, with a significant p-value of 0.0001.

Table 8: Difference mean of ALT (before and after), AST (before and after) in L-Cs group.

	Mean	Std. Deviation	P-value
ALT before ALT after	85.30 37.96	45.16 22.24	0.0001
AST before AST after	66.07 33.12	33.81 19.71	0.0001

Table 9 shows the association between ultrasound (US) results before and after treatment in the L-C group. All patients who had a normal US result before treatment maintained a normal result after treatment. For those with a US grade of 1.00 before treatment, 83.3% continued to have a US grade of 1.00 after treatment, with a significant p-value of 0.0001. Additionally, 83.3% of patients with a US grade of 2.00 before treatment maintained the same grade after treatment.

Table 9: Comparison between US before Treatment and After Treatment.

		US grades Before Treatment		
US grade after	**(1)**	**(2)**	**(3)**	**P-value**
Normal 1.00 2.00 3.00	7 (100.0%) 0 (0.0%) 0 (0.0%) 0 (0.0%)	1 (8.3%) 10 (83.3%) 1 (8.3%) 0 (0.0%)	0 (0.0%) 0 (0.0%) 5 (83.3%) 1 (16.7%)	0.0001 (significant)

DISCUSSION

The impact of L-carnitine supplementation on various health indicators in MAFLD patients, compared with the placebo group, and how these findings align or differ from similar studies. The ultrasound results show significant improvement in the L-C group compared to the placebo group, with 32% of patients in the L-C group achieving normal ultrasound findings, whereas no placebo group patients exhibited normal results. This marked improvement in liver fat reduction aligns with findings by Wang W et al. who observed that L-carnitine helps decrease liver fat deposition and inflammation.[9,10] This outcome is likely due to L-carnitine's role in facilitating fatty acid metabolism and reducing hepatic steatosis, which was similarly demonstrated in studies by Li et al. showing that L-carnitine could support the reversal of liver fat accumulation in MAFLD patients.[11] The ultrasound comparison before and after treatment within the L-C group shows a trend toward normalization in liver health, with 100% of patients with a baseline normal ultrasound result maintaining this status post-treatment. This result is supported by evidence from studies like those by Zakharova N et al. suggesting that L-carnitine can stabilize and improve liver imaging outcomes in MAFLD patients through lipid mobilization and reduction in liver fat.[12] The lipid profile analysis reveals significant reductions in total cholesterol (TC) and low-density lipoprotein (LDL) levels in the L-carnitine group, with p-values of 0.003 and 0.017, respectively. These results are consistent with similar studies indicating that L-carnitine can improve lipid metabolism. For instance, Rahbar et al. demonstrated that L-carnitine supplementation in MAFLD patients lowered LDL and triglycerides, reducing cardiovascular risk factors associated with MAFLD.[13] The L-C group also showed a slight increase

in high-density lipoprotein (HDL), though not statistically significant, mirroring the minor improvements in HDL reported by Arroyave-Ospina JC et al. in similar trials on L-carnitine's lipid-modulating effects.[14,15] L-C group before and after treatment revealed significant decreases in total cholesterol, LDL, and triglycerides and a significant increase in HDL. These results reinforce L-carnitine's lipid-lowering effect, consistent with a study by Pitti E et al. that observed improved lipid profiles among MAFLD patients. The observed TG reduction, albeit minor, aligns with similar improvements reported in studies focusing on long-term L-carnitine supplementation.[16] ALT and AST levels significantly decreased in the L-carnitine group compared to the placebo group (p-value 0.0001). This enzyme reduction suggests improved liver function and decreased liver cell damage, as L-carnitine's antioxidant properties may mitigate oxidative stress in hepatocytes. These results echo the findings of recent studies, including a trial by Anstee et al. which highlighted a reduction in ALT and AST levels in MAFLD patients treated with L-carnitine, supporting the supplement's protective effects on liver cells.[17] L-carnitine supplementation was associated with a reduction in waist circumference (WC) levels in the L-C group. The decrease in WC (p-value 0.0001) align with studies demonstrating the metabolic benefits of L-carnitine. For example, Mateus FG et al. observed that L-carnitine could helping reduce central obesity, which is crucial for MAFLD management.[18,19] The decrease in ALT and AST levels from baseline supports L-carnitine's hepatoprotective effects. This outcome resonates with findings by Nofal AE et al. who demonstrated that L-carnitine reduced oxidative damage in hepatocytes, aiding liver recovery.[20,21] So results indicate that L-carnitine positively impacts liver health, lipid profiles, liver enzymes, and some aspects of metabolic placebo in MAFLD patients, in agreement with similar studies. Differences in results across studies may relate to dosing regimens, treatment durations, and patient baseline characteristics, suggesting that optimizing these parameters could enhance therapeutic outcomes.

CONCLUSION

The study demonstrates that L-carnitine supplementation significantly improves liver function, reduces lipid levels, and enhances liver imaging outcomes in patients with nonalcoholic fatty liver disease (MAFLD). The reductions in ALT, AST, LDL, and triglycerides, along with ultrasound improvements, highlight L-carnitine's potential as an effective adjunctive therapy. These findings align with other studies indicating L-carnitine's role in mitigating MAFLD's metabolic and inflammatory factors. Further research is recommended to optimize dosing and treatment duration for maximizing therapeutic benefits in MAFLD management.

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