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Clinical Trial

Effects of L- Carnitine Supplementation on Nitric Oxide Levels in Pemphigus Vulgaris Patients: A Randomized, Double-Blind Clinical Trial

Running Title: L-carnitine increase NO in patients with PV

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Abstract

Background and objective: Blisters on the human skin and mucous membranes are a hallmark of the extremely rare autoimmune disease known as Pemphigus vulgaris (PV). Elevated levels of reactive oxygen species (ROS) are linked to this condition. Nitric oxide (NO), produced by endothelial cells, is a key signaling molecule that contributes to vascular function and can act as an antioxidant by neutralizing reactive oxygen species in specific biological contexts. A substance that resembles a vitamin, L-carnitine, positively influences antioxidant levels. This study aimed to investigate the impact of L-carnitine supplementation on serum nitric oxide levels in patients with Pemphigus vulgaris.

Method and materials: This clinical trial included a total of 46 patients with Pemphigus Vulgaris, aged between 30 and 65 years. Participants were randomly divided into two groups: one receiving L-carnitine (n = 23) and the other a placebo (n = 23). Each group took either 2000 mg of L-carnitine or placebo tablets daily. The intervention was conducted over 8 weeks, with serum L-carnitine and nitric oxide (NO) levels assessed both before and after the intervention.

Results: At baseline, the intervention and control groups did not differ statistically significantly in terms of age, weight, height, and BMI ($p > 0.05$). By the end of the study, patients in the L-Carnitine group showed a significant increase in serum L-Carnitine levels (from 74.56±36.36 to 97.49±41.27, $p<0.001$) and in nitric oxide (NO) concentration (from 202.37±14.59 to 242.98±20.63, $p=0.006$) In contrast, the placebo group did not show any significant changes in either parameter ($p>0.05$).

Conclusion: A daily intake of 2 g of L-carnitine for 8 weeks in patients with PV has positive effects on reducing oxidative stress and increasing serum nitric oxide levels.

Keywords: Pemphigus vulgaris, L-carnitine, Nitric oxide, Antioxidant, Oxidative stress

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Background

Pemphigus vulgaris (PV) is a rare, chronic autoimmune disorder that affects the skin. The formation of intraepithelial blisters and erosions is the primary feature of this condition, initiated by pathogenic autoantibodies that target specific membrane proteins in keratinocytes (1). The frequency of Pemphigus vulgaris cases is 0.5 to 4 cases per 100,000 people, with higher prevalence observed among individuals of Mediterranean descent and Jewish populations in contrast to other ethnic groups and regions. (2). Pemphigus vulgaris is more commonly found in individuals in their 40s and 50s, and some studies report a higher prevalence in women. (3). According to Davatchy and other research, Pemphigus vulgaris has an incidence rate of one case per 100,000 persons in Iran, with an average age of onset of 42 years and a mortality rate of 6.2% (4).

Increased levels of circulating autoantibodies (mostly IgG and infrequently IgA) that target elements of the desmosome complex, particularly the proteins Desmoglein 1 and 3, are a contributing factor in the pathophysiology of Pemphigus. This antibody response disrupts cell-cell junctions, allowing interstitial fluid to penetrate and leading to blister formation in the skin (5, 6). Although numerous studies have been conducted, the etiology of the disease remains unclear. However, a correlation has been observed between genetic susceptibility and the prevalence of Pemphigus vulgaris in specific populations (7). While a genetic predisposition is necessary for developing Pemphigus vulgaris, it is not sufficient on its own, as there are additional independent risk factors that contribute to the onset of the disease. Consequently, the influence of environmentally induced factors appears to be critical for triggering the disease initially (as initiating factors) and for sustaining it over time (as perpetuating factors) (8). Environmental factors encompass various elements, including certain medications, viral infections, physical factors, allergen exposure, and specific foods. (9). The aim of treatment for pemphigus vulgaris, similar to other autoimmune blistering diseases, is to reduce the formation of blisters, promote healing, and decrease the medication required to manage the disease. Although immunosuppressive and corticosteroid medications have reduced the overall mortality rate, a significant number of deaths in these patients are now linked to the side effects of these treatments, such as a decline in the concentration and activity of enzymatic and non-enzymatic antioxidants (10, 11).

L-carnitine (LC), also known as L-3-hydroxytrimethylamminobutanoate, is a vitamin-like compound that is primarily absorbed from meat and dairy products. In mammals, it is produced from the essential amino acids lysine and methionine in various organs, including the liver, kidneys, skeletal muscles, heart, and brain (12). L-carnitine acts as an antioxidant and antiperoxide by neutralizing hydroxyl radicals, superoxide anions, and hydrogen peroxide and also by inhibiting the formation of hydroxyl radicals during the Fenton reaction. (13).

Nitric oxide (NO) is synthesized in endothelial cells by three different forms of nitric oxide synthase (NOS) (14). Due to its chemical properties as a nitrogen-centered free radical, nitric oxide (NO) can scavenge reactive oxygen species, serving as a potent

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antioxidant that protects the body from oxidative stress and damage induced by these reactive species. (15). Various studies have shown that the use of corticosteroids leads to an increase in oxidative stress among patients with Pemphigus vulgaris (PV) (11, 16). Consequently, the present study was conducted to assess the impact of L-carnitine supplementation on serum nitric oxide (NO) and L-carnitine levels in patients with Pemphigus vulgaris (PV).

Materials and methods

Study participants

Patients with pemphigus vulgaris who visited the dermatology department at Razi Hospital in Tehran were recruited for this study in a blinded manner. To be enrolled in this study, patients were required to be between 30 and 65 years old, have a body mass index (BMI) of ≤ 35 kg/m^2, take corticosteroid medication in combination with methotrexate, azathioprine, or CellCept, and have a history of at least one year of PV disease. The exclusion criteria were life-threatening complications due to LC supplementation, change in the dose or type of medication, incomplete questionnaires, and <90% compliance with the intervention protocol. The sample size was determined according to a study by BJ Lee. (17) Regarding the effects of L-carnitine supplementation on antioxidant enzyme activities, the sample size was initially calculated to include at least 19 cases in each group, with a confidence level of 95% and a power of 80%. To account for a potential dropout rate of 20%, the sample size was subsequently increased to 23 cases in each group. The study protocol was approved by the Ethical Committee of the Tehran University of Medical Sciences (IR.TUMS.REC.1394.1992). This study was also registered in the Iranian Registry of Clinical Trials (IRCT2015062322769N4), and all participants were informed about the study's details and provided written informed consent.

This study was designed as a randomized, double-blind, placebo-controlled trial. Patients were assigned to two groups using permuted block randomization with a random code, receiving either 2000 mg of L-Carnitine or placebo tablets. To maintain the double-blind nature of the study, the containers with placebo and L-Carnitine pills were categorized into two groups, A and B, by someone not involved in the research. For eight weeks, the experimental group (n = 23) received 2000 mg of L-carnitine daily in two equal doses, administered at breakfast and dinner. The control group (n = 23) received a placebo with the same schedule and duration. Throughout the trial, participants were told not to change their regular eating patterns, physical activity levels, medication types, or dosages. Weekly phone calls were made to check on any adverse effects of taking L-carnitine supplements and to make sure they were taking the pills as prescribed.

Using a Seca scale (Seca, Clara 803) with an accuracy of 100 g, participants' body weight was recorded at the start and finish of the intervention while they were fasting, voiding, wearing minimal clothes, and without shoes. A wall-mounted height meter (Seca 206, Germany) was used to measure participants' height while they were standing, without shoes, with their knees and heels against the wall, looking straight ahead, and with their shoulders in a normal position. The measurement had an accuracy of 1.0 cm.

Dietary intake data were collected using a 24-hour dietary recall method before and after the intervention, assessing intake over three consecutive days (including two weekdays and one weekend). The amounts listed for each food item were converted to grams using a domestic scale guide and analyzed using the Nutritionist IV software program (version 4.1, First Databank Division, The Hearst Corporation, San Bruno, CA, USA).

Blood collection and biochemical measurements

Fasting venous blood samples (10 mL) were collected at both the start and the end of the study. After centrifugation for 10 minutes, serum samples were separated from the blood and stored at −80°C until biochemical analysis. The serum levels of nitric oxide (NO) and L-Carnitine (LC) were measured using Enzyme-Linked Immunosorbent Assay (ELISA) kits from SHANGHAICRYSTAL DAY BIOTECH CO., LTD (Cat. No: E1510Hu) and Zell Bio GmbH, Germany (Cat. No: ZB-3426-R9648), following the suppliers' instructions. The assay range for NO analysis was 2 µmol/L to 600 µmol/L, with a sensitivity of 1.12 µmol/L.

Statistical analyses

IBM-SPSS Modeller software (version 16, IBM-SPSS Inc., Chicago, IL) was used for statistical analysis. The results are presented as mean ± standard deviation (SD). Statistical significance was defined as a p-value of less than 0.05. An independent-sample t-test was used for intergroup comparisons, and a paired t-test was used to evaluate differences between variables before and after the intervention.

Results

Patient characteristics

Figure 1 summarizes the trial profiles and the sampling process. One participant was eliminated from each group out of the 46 who signed up for the study because they stopped taking the intervention for personal reasons or due to non-compliance with study procedures. The final analysis included participants who demonstrated good adherence to L-carnitine consumption and who did not experience any severe symptoms or adverse effects.

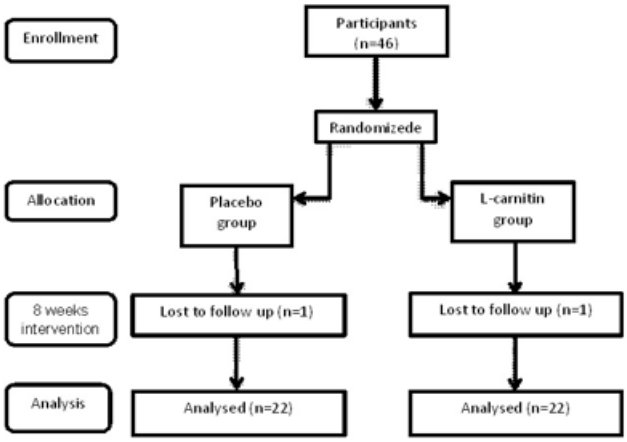

Table 1. Baseline characteristics of research participants

Variable	L-Carnitine group (n = 22)	Placebo group (n = 22)	P-value*
sex	-	-	
men(*n*)	8	7	
Women (*n*)	14	15	
Age (y)	41.36±2.12	41.55±2.17	.953
Weight (kg)	77.32±13.66	72.42±9.50	.137
Height (cm)	164.9±6.95	164.56±10.25	.076
BMI (kg/m ^{2})	28.35±4.39	26.80±3.40	.136

Values are means ± SD.

P < .05 was accepted as significant

* P values **show** a comparison between groups at baseline (independent-sample t-test)

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Table 1. presents the baseline characteristics of individuals in both groups. There were no statistically significant differences in age, height, weight, or BMI (p>0.05).

The effects of L-carnitine supplementation on serum levels of L-carnitine and nitric oxide (NO) at baseline and after eight weeks are presented in **Table 2**.

Table 2. Serum LC and NO concentration and dietary intake of Vitamin C and Protein at baseline and after 8 weeks

Variable		L-Carnitine group (n =22)	Placebo group (n =22)	P-value
L-carnitine (nmol/mL)	Baseline After 8 wk Change P-value #	74.56±36.36 97.49±41.27 21.97±16.54 <.001	77.82±36.64 75.77±36.78 -1.95±5.91 .12	.79 .09 <.001
NO (µmoL/L)	Baseline After 8 wk Change P-value	202.37±14.59 242.98±20.63 61.40±13.12 .006	250.46±27.32 231.32±18.98 -19.14±15.76 .238	.130 .679 .006
Vitamin C (mg)	Baseline After 8 wk P-value	79.36±14.16 115.98±20.23 .122	131.37±62.13 106.33±106.33 .710	.423 .685
Protein (gr)	Baseline After 8 wk P-value	66.54±19.31 74.25±24.93 .13	69.44±18.33 65.40±17.78 .37	.59 .16

Values are means ± SD unless otherwise indicated.
P < .05 was considered significant.
*P values show comparison between groups (independent-sample t)
#P values show comparison within groups (paired t-test)

Before the intervention, no significant differences were found in the concentrations of L-carnitine and NO between the two groups. However, compared to the control group, the changes in serum L-carnitine and NO levels in the L-carnitine group were statistically significant ($p < 0.001$ and $p = 0.006$, respectively). Furthermore, within the L-carnitine group, statistically significant differences were observed when comparing pre-and post-intervention values for serum L-carnitine and NO ($p < 0.001$ and $p = 0.006$, respectively).

Additionally, **Table 2** presents the average daily dietary intake of protein and vitamin C in both groups before and after the intervention. The intra-group and inter-group comparisons revealed no statistically significant differences in daily protein and vitamin C intake throughout the study ($p > 0.05$).

Discussion

To the best of our knowledge, this is the first study to investigate the effects of L-carnitine supplementation on serum nitric oxide (NO) levels in patients with primary valvular disease (PV). The findings suggest a notable increase in serum L-carnitine levels (from 74.56±36.36 to 97.49±41.27) and NO levels (from 202.37±14.59 to 242.98±20.63) following L-Carnitine administration. Furthermore, these changes reached statistical significance, with p-values of <0.001 and 0.006, respectively.

In recent decades, substantial evidence has emerged linking oxidative stress to Pemphigus vulgaris (PV) disease. (11, 18) Reactive Oxygen Species (ROS) are among the most significant factors involved in the inflammatory process (18). It has been reported that the skin is particularly susceptible to exposure to high levels of reactive oxygen species (ROS). (19). Several studies have indicated an elevated production of reactive oxygen species (ROS) in patients with

Pemphigus vulgaris, which is attributed to damaged epithelial cells and heightened neutrophil activity (18, 20). The use of immunosuppressive and corticosteroid medications is likely associated with an increase in oxidative stress. (11, 16). Although using these drugs as part of a treatment plan has decreased overall mortality, patients have experienced several adverse side effects. (10). The decrease in the quantity and activity of antioxidants, both enzymatic and non-enzymatic, is one of the most important adverse effects (11) that is linked to a rise in ROS levels. (18).

Nitric oxide synthase (NOS) enzymes in endothelial cells produce nitric oxide (NO), a molecule known for its antioxidant properties. (21). Nitric oxide is thought to regulate oxidative processes, the production of cytotoxic oxygen species, and the scavenging of free radicals via stimulating guanylate cyclase. Furthermore, NO is known to scavenge hydroxyl radicals produced by ferrous compounds and to have protective effects against lipid peroxidation. (22). Some studies have explored the impact of immunosuppressive drugs on serum nitric oxide (NO) levels. For example, Yazici et al.'s study showed that corticosteroid treatment in nasal polyposis tissue reduced the expression of NOS (23). Immunosuppressive drugs were shown to suppress the expression of NOS in vitro investigation by Attur et al. Because of this restriction, the generation of nitric oxide (NO) was significantly reduced. (24) Additionally, oxidative stress can have detrimental effects on vascular walls by inactivating nitric oxide (NO) through the formation of superoxide anions, leading to endothelial dysfunction (25). Similarly, our study's findings indicated that in the placebo group, NO concentration decreased after 8 weeks (from 250.46±27.32 to 231.32±18.98). However, this decrease was not statistically significant; the brief intervention likely contributed to it. In contrast, Siebra et al.'s investigation revealed that patients with Pemphigus vulgaris (PV) had considerably higher serum NO levels than controls; however, these changes did not show significant differences between the active and inactive forms of the disease. Additionally, this study employed a range of testing techniques, utilizing the Griess reaction to quantify the amounts of total serum nitrate and nitrite as indicators of NO generation. (26).

Considering the high levels of oxidative stress in Pemphigus vulgaris patients and the antioxidant effects of nitric oxide, it appears beneficial to enhance the serum concentration of this enzyme in PV patients. In our study, NO levels increased significantly following LC supplementation. This finding aligns with prior in vitro studies, such as those by Calo et al., who demonstrated that L-carnitine enhances NOS gene and protein expression following incubation with L-carnitine. (27). Similar to the current investigation, two well-established clinical trials have shown how LC supplementation affects serum nitric oxide (NO) concentration and have identified a favorable association between LC and NO levels after supplementation. (28, 29). In one of these studies, 26 healthy men received either 3 or 4 grams of LC mixed with fruit juice, followed by a treadmill test conducted one hour later. The findings showed that, compared to when the same patients were examined with placebo fluids, the plasma levels

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of nitrate and nitrite (NOx) increased considerably (28). Similarly, Koozehchian et al. demonstrated that elevated serum NO levels occurred following L-carnitine intake during endurance training. Our findings extend this evidence to patients with PV, highlighting the potential of LC in reducing oxidative damage in this autoimmune condition.

Notably, no serious adverse events were reported, and participants did not experience side effects such as nausea or vomiting, consistent with previous studies that confirm its safety profile (30). Therefore, LC at a dose of 2000 mg/day is well tolerated and beneficial in improving antioxidant capacity by elevating serum NO levels in patients with PV.

However, some limitations should be acknowledged. The study duration was relatively short, and the sample size was limited. Further studies with larger cohorts and extended follow-up periods are necessary to validate these findings and explore long-term outcomes, including clinical markers of disease activity.

In conclusion, L-carnitine supplementation significantly improved serum nitric oxide levels in patients with Pemphigus vulgaris, indicating enhanced antioxidant defense. These results, in line with prior evidence from both healthy populations and other inflammatory settings, suggest a potential therapeutic benefit of LC in oxidative stress management in PV.

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