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CLINICAL RESEARCH

Levocarnitine supplementation for management of hypertriglyceridemia in patients receiving parenteral nutrition

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Abstract

Background: Levocarnitine deficiency has been observed in patients receiving parenteral nutrition (PN) and can cause or worsen hypertriglyceridemia. The objective was to characterize use of levocarnitine supplementation in PN and evaluate its effect on triglyceride levels in hospitalized adults.

Methods: This retrospective, single-center study included patients with triglyceride levels ≥175 mg/dl while receiving PN who had a subsequent reduction in lipid injectable emulsion dose. A piecewise linear regression was used to evaluate trends in triglyceride levels before and after the intervention, defined as initiation of levocarnitine in PN for the levocarnitine group, or reduction in lipid injectable emulsion alone for the control group.

Results: Two hundred sixty-one patients who received PN had an elevated triglyceride level and lipid injectable emulsion dose reduction, of which 97 (37.2%) received levocarnitine in PN. The median (IQR) levocarnitine dose added to PN was 8.0 (5.7–9.9) mg/kg. Triglyceride levels at 30 days post-intervention did not differ between groups (125 vs 176 mg/dl, $P = .345$). The addition of levocarnitine to PN was associated with a significantly greater rate of reduction in triglyceride levels pre-intervention to post-intervention compared with a reduction in lipid injectable emulsion alone (–11 vs –3 mg/dl per day; 95% CI, –15 to –2; $P = .012$).

Conclusion: In hospitalized adults with hypertriglyceridemia who had a lipid injectable emulsion dose reduction, the addition of levocarnitine in PN was not associated with a difference in triglyceride levels at 30 days; however, a greater rate of improvement in pre-intervention to post-intervention triglyceride levels was observed.

KEYWORDS
carnitine, hypertriglyceridemia, intravenous lipid emulsions, parenteral nutrition

INTRODUCTION

Carnitine is an essential carrier molecule in long-chain fatty acid metabolism, facilitating the transport of fatty acids from the cytosol into the mitochondria. Fatty acids then undergo beta oxidation, which produces energy used in tissues throughout the body. Approximately three-fourths of carnitine stores are obtained through the diet from animal products such as red meat and dairy. Carnitine is also produced endogenously in the kidney, liver, and brain, with renal tubule resorption leading to the conservation of carnitine.[1]

Carnitine deficiency can occur due to decreased carnitine synthesis or intake or increased carnitine requirements or loss.[1] In a state of carnitine deficiency, fatty acid oxidation is diminished leading to conversion to triglycerides. This can lead to hypertriglyceridemia,[2] defined as triglyceride levels ≥175 mg/dl by the American Heart Association and American College of Cardiology (AHA/ACC).[3] Previous studies have shown that patients receiving parenteral nutrition (PN) have decreased serum carnitine concentrations likely due to decreased dietary intake.[4,5]

Levocarnitine, the L-isomer and active form of carnitine, may be administered in PN to adult patients with hypertriglyceridemia. The effect of levocarnitine supplemented outside of PN on triglyceride levels has been studied with mixed results in patients with end-stage renal disease on intermittent hemodialysis[6–11] given their risk of carnitine deficiency due to removal of carnitine through dialysis and decreased synthesis by the kidneys.[12] However, studies evaluating the effect of levocarnitine supplementation in PN on triglyceride levels are lacking. Additionally, there is no established recommended dietary allowance for levocarnitine, and no standardized dosing regimen of levocarnitine in PN currently exists.[13] The purpose of this study was to characterize the use of levocarnitine in PN and evaluate its effect on triglyceride levels in hospitalized adult patients.

METHODS

Study design

This was a retrospective, observational, single-center cohort study performed in a tertiary referral center from August 2016 to July 2019. Patients ≥18 years of age were included if they had a triglyceride level ≥175 mg/dl and a reduction in the dose or frequency of lipid injectable emulsion in PN while receiving PN during their index hospital admission. The triglyceride level cutoff was chosen in accordance with AHA/ACC criteria for moderate hypertriglyceridemia. Patients received a 3-in-1 PN solution with 20% lipid injectable emulsion (Nutrilipid; B. Braun Medical Inc, Bethlehem, PA). Patients with familial hypertriglyceridemia, inborn errors of metabolism managed by the genetics nutrition service, a gap in PN administration >5 days, or administration of levocarnitine outside PN were excluded. The study received Institutional Review Board approval with waived informed consent. Demographic and medical information related to PN and levocarnitine administration, triglyceride levels, actual body weight, renal replacement therapy, triglyceride-altering medications prior to and during admission, and past medical history were evaluated.

Study objectives

In patients receiving PN with hypertriglyceridemia, all of whom had a reduction in the dose or frequency of lipid injectable emulsion in PN, the primary objective was to compare trends in triglyceride levels before and after the triglyceride-altering intervention. The triglyceride-altering intervention was defined as initiation of levocarnitine in PN for the levocarnitine group, and as reduction in lipid injectable emulsion for the control group. Secondary objectives included describing patients who received levocarnitine in PN and characterizing the dosing of levocarnitine in PN. Ideal body weight in non-obese patients (BMI < 30 kg/m^2) and adjusted body weight in obese patients (BMI ≥ 30 kg/m^2) were used to analyze weight-based dosing of levocarnitine in PN using the following equations:

$$\text{Ideal body weight (kg), males} = 50.0 + 2.3 \times (\textit{height in inches} - 60)$$

$$\text{Ideal body weight (kg), females} = 45.5 + 2.3 \times (\textit{height in inches} - 60)$$

$$\text{Adjusted body weight (kg)} = \text{ideal body weight} + 0.4 \times (\textit{actual body weight} - \textit{ideal body weight})$$

$$\text{Body mass index } \left(\text{kg/m}^2\right) = \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

Statistical analysis

Descriptive statistics were used to summarize the data collected and to represent the secondary outcomes of the description of patients who received levocarnitine in PN

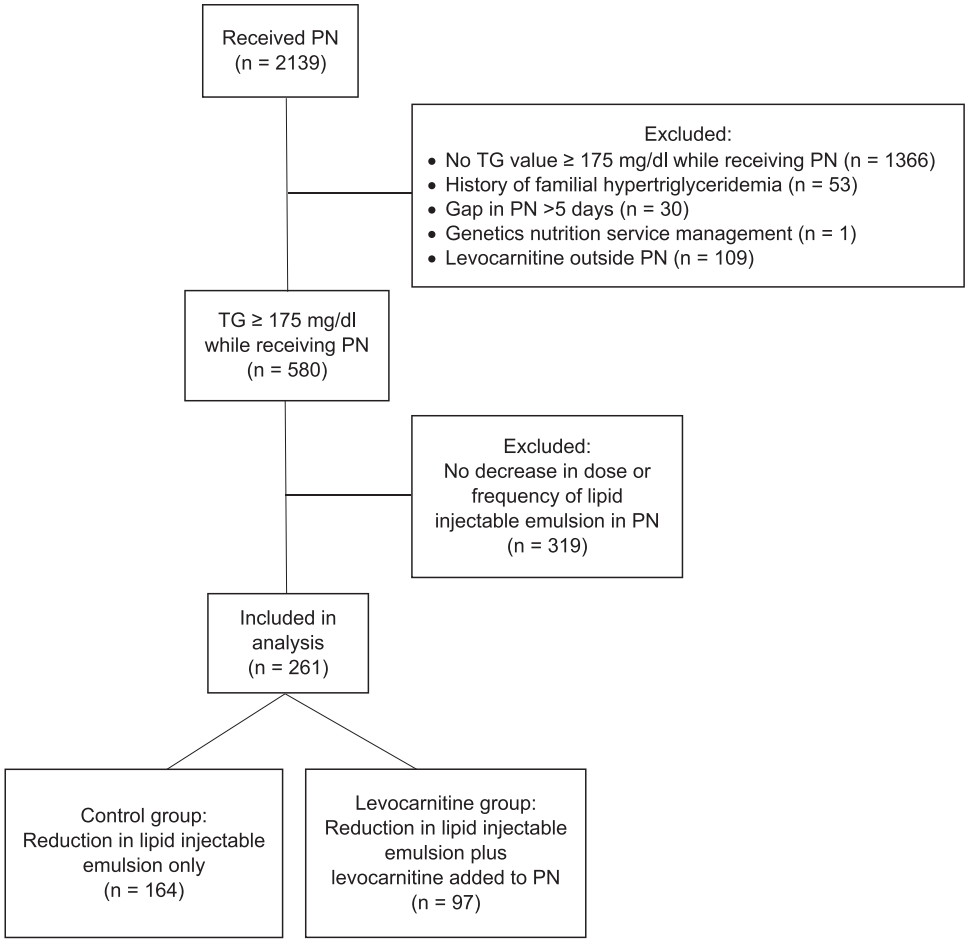


FIGURE 1 Patient selection. PN, parenteral nutrition; TG, triglyceride

as well as characterization of dosing of levocarnitine in PN. Wilcoxon rank sum and χ^2 tests were used to compare baseline characteristics between groups for continuous and categorical data, respectively. Fisher's exact test was used for categorical baseline characteristics with a frequency of less than five. A piecewise linear regression with estimated means was performed to evaluate the primary outcome of the comparison of trends before and after the triglyceride-altering intervention between groups. Covariates were added to the regression model for adjustment. An α-level of 0.05 was used for all statistical comparisons. Analyses were performed using STATA Statistical Software, version 15 (Stata Corp LP, College Station, TX).

RESULTS

Patient characteristics

There were 2139 adult patients who received PN during their hospitaladmission (Figure 1). Patients with familial hypertriglyceridemia (n = 53), a gap in PN orders >5 days (n = 30), were managed by the genetics nutrition service (n = 1), or received levocarnitine outside of PN (n = 109) were excluded. Five hundred eighty patients had a triglyceride level ≥175 mg/dl while receiving PN. Of these, 261 (45.0%) patients with elevated triglyceride levels had a reduction in the dose and/or frequency of lipid injectable emulsion and were included in the analysis. Of these patients, 97 (37.2%) patients received levocarnitine in PN and comprised the levocarnitine group, with the remaining 164 (62.8%) patients comprising the control group.

Patient characteristics are outlined in Table 1. Seventy-one percent of patients were admitted to a nonintensive care unit. Patients received PN for a median (IQR) of 15 (9–24) days. Only two patients in the study, both in the levocarnitine group, had a history of end-stage renal disease. While receiving PN, more patients in the levocarnitine group received renal replacement therapy than in the control group (17.5% vs 7.3%, $P = .011$). Almost 20% of patients overall received propofol while receiving PN, with similar use between groups (22.7% vs 17.0%, $P = .266$). Incidence of severe hypertriglyceridemia, defined as

TABLE 1 Patient characteristics

Characteristic	Overall (n = 261)	Levocarnitine group (n = 97)	Control group (n = 164)	*P*-value
Age, years, median (IQR)	61 (47–70)	61 (49–70)	61 (47–70)	.867
Female sex, n (%)	157 (60.2)	62 (63.9)	95 (57.9)	.339
Weight, kg, median (IQR)	76 (60–95)	69 (55–85)	80 (61–97)	.108
Race, n (%)				.362
White	182 (69.7)	63 (64.9)	119 (72.6)	
Black	52 (19.9)	20 (20.6)	32 (19.5)	
Other	27 (10.3)	14 (14.4)	13 (7.9)	
Unit where PN initiated, n (%)				.622
Intensive care	75 (28.7)	35 (36.1)	40 (24.4)	
Nonintensive care	186 (71.3)	62 (63.9)	124 (75.6)	
Duration of PN, days, median (IQR)	15 (9–24)	17 (10–27)	14 (9–22)	.0635
Severe hypertriglyceridemia[*], n (%)	22 (8.4)	10 (10.3)	12 (7.3)	.400
History of end-stage renal disease, n (%)	2 (0.8)	2 (2.1)	0	.137
Renal replacement therapy[†], n (%)	29 (11.1)	17 (17.5)	12 (7.3)	.011
Propofol administration[†], n (%)	50 (19.2)	22 (22.7)	28 (17.0)	.266
Triglyceride-altering medication[‡] prior to admission, n (%)	56 (21.5)	17 (17.5)	39 (23.8)	.259
Triglyceride-altering medication[‡] during admission, n (%)	42 (16.1)	14 (14.4)	28 (17.1)	.575

[*]Triglyceride levels > 500 mg/dl.
[†]During PN study period.
[‡]Fenofibrate, gemfibrozil, niacin, ω-3 fatty acids/fish oil, or statins.
Abbreviations: IQR, interquartile range; PN, parenteral nutrition.

triglyceride levels > 500 mg/dL[3], was also similar between groups (10.3% vs 7.3%, $P = .400$). No patients received four-oil (soybean/medium chain triglycerides/olive/fish oils) lipid injectable emulsion (Smoflipid; Fresenius Kabi USA, Lake Zurich, IL).

Characterization of lipid injectable emulsion reduction is outlined in Table 2. Practices for reducing lipid injectable emulsion in PN based on dose, frequency, or both were similar between groups. Patients in the levocarnitine group had a smaller reduction in the average daily dose of lipid injectable emulsion before and after the triglyceride-altering intervention compared with control group patients (–7 vs –12 g per day, $P = .019$).

Patients receiving levocarnitine in PN

Forty-four (45.4%) of the 97 patients who received levocarnitine in PN had a reduction in lipid injectable emulsion prior to levocarnitine initiation in PN, with a median (IQR) difference of 5 (3–8.5) days. Thirty-seven (38.1%) patients had levocarnitine added to PN prior to a reduction in lipid injectable emulsion, with a median (IQR) difference of 2 (1–7) days. Sixteen (16.5%) patients had levocarnitine added to PN the same day lipid injectable emulsion in PN was reduced.

Patients received levocarnitine in PN for a median (IQR) duration of 14 (8–33) days, which accounted for a median (IQR) of 68.4% (43.3%–86.0%) of the total number of days receiving PN. Patients received levocarnitine in PN for a median (IQR) of 83.3% (56.0%–100.0%) of the days receiving PN after the first triglyceride level ≥ 175 mg/dl.

Ten (45.5%) of 22 patients with severe hypertriglyceridemia, defined as triglyceride levels > 500 mg/dL[3], received levocarnitine in PN. Seventeen (58.6%) of 29 patients on renal replacement therapy while receiving PN received levocarnitine in PN.

TABLE 2 Lipid injectable emulsion reduction characterization

Characteristic	Overall (n = 261)	Levocarnitine group (n = 97)	Control group (n = 164)	*P*-value
Change in lipid injectable emulsion in PN, n (%)				.293
Decrease in dose only	91 (34.9)	28 (28.9)	63 (38.4)	
Decrease in frequency only	88 (33.7)	37 (38.1)	52 (31.7)	
Decrease in dose and frequency	82 (31.4)	32 (33.0)	49 (29.9)	
Lipid injectable emulsion in PN, g/day, median (IQR)				
Before triglyceride-altering intervention*	48 (36–51)	44 (29–50)	50 (42–54)	<.001
After triglyceride-altering intervention*	35 (26–44)	36 (23–44)	35 (27–44)	.625
Lipid injectable emulsion in PN, g/kg/day, median (IQR)				
Before triglyceride-altering intervention*	0.61 (0.45–0.75)	0.56 (0.42–0.70)	0.64 (0.49–0.77)	.046
After triglyceride-altering intervention*	0.47 (0.33–0.69)	0.49 (0.30–0.58)	0.46 (0.34–0.57)	.743
Lipid injectable emulsion in PN before vs after triglyceride-altering intervention*, g/day, median (IQR)	−10 (−24 to −1)	−7 (−17 to −5)	−12 (−25 to −3)	.019

*Triglyceride-altering intervention: initiation of levocarnitine in PN for the levocarnitine group, or reduction in fat in PN for the control group.
Abbreviations: IQR, interquartile range; PN, parenteral nutrition.

Dosing of levocarnitine in PN

The mean (± SD) initial dose of levocarnitine in PN was 524 (± 203.6) mg. The median (IQR) weight-based dose of levocarnitine in PN was 8.0 (5.7–9.9) mg/kg based on ideal body weight, or adjusted body weight in obese patients with BMI ≥30 kg/m^2. Eighty-three (85.6%) patients did not have a change in levocarnitine dose while receiving levocarnitine in PN.

Trends in triglyceride levels

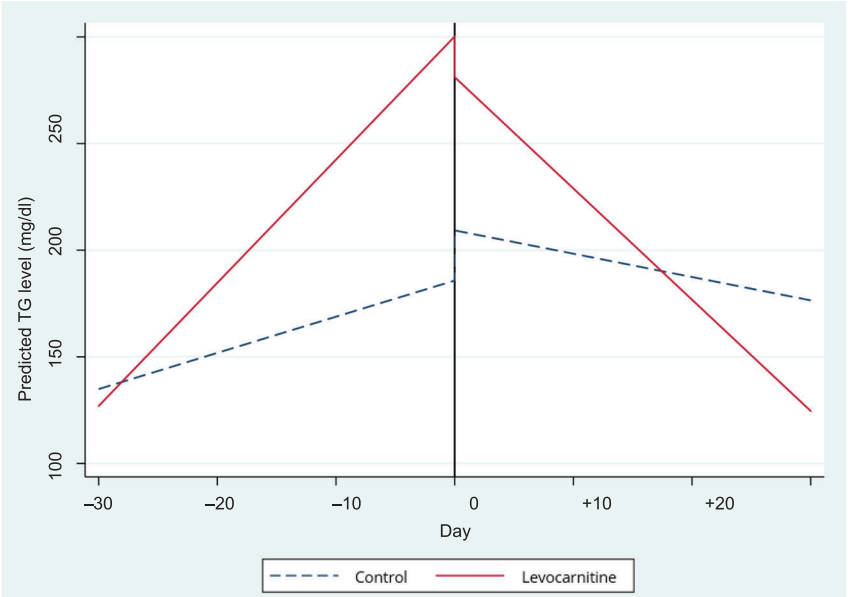
The piecewise linear regression model of trends in triglyceride levels within 30 days before and 30 days after the triglyceride-altering intervention is shown in Figure 2 (R^2, levocarnitine group, pre-intervention: 0.044; R^2, control group, pre-intervention: 0.029; R^2, levocarnitine group, post-intervention: 0.046; R^2, control group, post-intervention: 0.018). At 30 days prior to the intervention, there was no difference in triglyceride levels between the levocarnitine and control groups (127 vs 135 mg/dl; 95% CI, −106 to 90; $P = .874$). During the 30 days prior to the intervention, triglyceride levels increased more rapidly in the levocarnitine group compared with the control group (6 vs 2 mg/dl per day; 95% CI, 0.4–8; $P = .031$). Immediately prior to the triglyceride-altering intervention, triglyceride levels were significantly higher in the levocarnitine group (300 vs 186 mg/dl; 95% CI, 71–15;, $P < .001$).

After the intervention, there was no difference between the levocarnitine vs control group in the rate of decrease in triglyceride levels within 30 days (−5 vs −1 mg/dl per day; 95% CI, −9 to 1; $P = .09$) or in triglyceride levels at 30 days post-intervention (125 vs 176 mg/dl; 95% CI, −160 to 56; $P = .345$). However, the addition of levocarnitine to PN was associated with a greater pre-intervention to post-intervention rate of change in triglyceride levels compared with a reduction in lipid injectable emulsion alone (−11 vs −3 mg/dl per day; 95% CI, −15 to −2; $P = .012$).

When adjusting for renal replacement therapy during the PN study period, results were similar to the unadjusted model (Table S1). Additionally, in a subpopulation analysis of the levocarnitine group, when adjusting for initial levocarnitine dose, the results were also similar to the unadjusted model (Table S2).

DISCUSSION

In this study, trends in triglyceride levels were compared in hypertriglyceridemic hospitalized adult patients who



Estimated mean finding	Levocarnitine Group	Control Group	Difference	95% CI	p value
TG 30 days pre-intervention, mg/dl	127	135	–8	–106 to 90	p=0.874
Rate of change in TG pre-intervention, mg/dl per day	6	2	4	0.4 to 8	p=0.031
TG on day of intervention, mg/dl	300	186	114	71 to 158	p<0.001
Rate of change in TG post-intervention, mg/dl per day	–5	–1	–4	–9 to 1	p=0.09
TG 30 days post-intervention, mg/dl	125	176	–52	–160 to 56	p=0.345
Rate of change in TG pre- to post-intervention*, mg/dl per day	–11	–3	–8	–15 to –2	p=0.012

CI, confidence interval; TG, triglyceride
Day 0=intervention (i.e., addition of levocarnitine in PN in levocarnitine group, or reduction in lipid injectable emulsion in PN in control group)
*Calculated by subtracting the rate of change in TG pre-intervention from the rate of change of TG post-intervention as a measure of the total degree of the rate of change in TG

FIGURE 2 Piecewise linear regression of triglyceride trends before and after intervention. TG, triglyceride

received levocarnitine supplementation in PN vs a reduction in lipid injectable emulsion in PN alone. Triglyceride levels at 30 days were not different between groups, nor was the rate of change in triglyceride levels post-intervention. However, the addition of levocarnitine in PN was associated with a greater rate of improvement in pre-intervention to post-intervention triglyceride levels compared with lipid injectable emulsion reduction alone. Additionally, the average daily dose of lipid injectable emulsion was reduced more significantly in control group patients than in levocarnitine group patients, suggesting that levocarnitine may have contributed to a reduction in triglyceride levels.

This is the largest study to date evaluating the effect of levocarnitine supplementation in PN on triglyceride levels. Overall, there are few existing studies assessing the use of levocarnitine in this setting. In a study of 16 adult patients who received levocarnitine in PN postoperatively, there was no difference in triglyceride levels preoperatively vs 11 days postoperatively when compared with those who did not receive levocarnitine.[14] In neonates, a meta-analysis showed no difference in triglyceride levels in patients supplemented with levocarnitine in PN vs those who were not.[15]

The effect of levocarnitine outside of PN on triglyceride levels has been studied in patients with end-stage renal disease on intermittent hemodialysis[6–11] given their risk of carnitine deficiency due to removal of carnitine through dialysis and decreased synthesis by the kidneys.[12] The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) reviewed 32 small studies with significant heterogeneity in levocarnitine dosing (ranging from 1 mg/kg to 3000 mg), frequency (ranging from three times weekly after intermittent hemodialysis to daily), route of administration (including oral, intravenous, and intradialysate), and duration (ranging from 1 week to 15 months). There was no significant change in triglyceride levels in 23 out of 32 studies. In the remaining studies, a significant reduction in triglyceride levels in patients who received levocarnitine was observed in patients who had elevated triglyceride levels at baseline. Based on this review, the KDOQI Guidelines for Nutrition in Chronic

Renal Failure state there are insufficient data to support the routine use of levocarnitine for dialysis-associated hypertriglycedemia.[16] In this study, more patients in the levocarnitine group received renal replacement therapy during the PN study period. Adjusting the triglyceride trend regression model for renal replacement therapy did not significantly impact the results. However, <15% of the study population received renal replacement therapy, which may have limited the ability to adequately assess this variable.

In addition to carnitine deficiency,[4,5] patients receiving PN may be at risk for hypertriglyceridemia because of lipid injectable emulsion exposure itself. The recommended dose for soybean oil–based lipid injectable emulsion in PN is ≤1 g/kg/day and should not exceed 2.5 g/kg/day or 0.11 g/kg/h because of increased risk of adverse effects, including hypertriglyceridemia. Lipid injectable emulsion should be initiated with caution in patients with hypertriglyceridemia and withheld in PN when triglyceride levels exceed 400 mg/dl.[17] To capture attempts at mitigating hypertriglyceridemia for patients receiving PN, all patients included in this study had a lipid injectable emulsion dose reduction, which served as the triglyceride-altering intervention for the control group in the piecewise linear regression, compared with the addition of levocarnitine in PN for the levocarnitine group.

Compared with lipid injectable emulsion reduction alone, levocarnitine supplementation in PN in addition to lipid injectable emulsion reduction was associated with a greater decrease in pre-intervention to post-intervention triglyceride levels. Triglyceride levels were increasing more rapidly prior to the intervention in the levocarnitine group, and triglyceride levels immediately prior to the intervention were higher in the levocarnitine group. Consequently, selection bias may have impacted the results such that a larger effect on triglyceride levels was able to be observed in the levocarnitine group. Additionally, there was no standard approach for reducing the lipid injectable emulsion dose, and there was variability in the timing of initiation of levocarnitine in PN relative to the lipid injectable emulsion reduction. In assessing the trajectory of triglyceride levels, caution should be used in drawing direct comparisons between these groups.

Although the rate of decrease in triglyceride levels after the triglyceride-altering intervention in the levocarnitine group did not differ from that in the control group, an important trend favoring the levocarnitine group was observed that should be evaluated in a larger sample size. Although this study does not confirm a cause-and-effect relationship between levocarnitine supplementation in PN and a reduction in triglyceride levels, it is hypothesis-generating, and prospective studies are needed to evaluate this comparison. Given levocarnitine supplementation in PN was associated with a significant reduction in pre-intervention to post-intervention triglyceride levels compared with the control group despite a lesser reduction in lipid provision in the levocarnitine group, and with its favorable adverse effect profile, it may be reasonable to consider supplementing levocarnitine in PN for hospitalized adult patients with elevated triglyceride levels.

A median levocarnitine dose of 8 mg/kg/day was utilized in our study. At the time of this study at our institution, there was not a standardized dosing protocol for levocarnitine supplementation in PN. Evidence for dosing recommendations is limited, with suggested doses ranging from 2 to 50 mg/kg/day.[1] The decision to report the weight-based dose of levocarnitine in PN using ideal body weight or adjusted body weight for obese patients with BMI ≥30 kg/m^2 was twofold: the volume of distribution of levocarnitine is small at 0.2–0.3 L/kg,[18] and ideal body weight is independent of factors that can affect actual body weight during acute illness, such as volume status. A change in levocarnitine dose in PN occurred infrequently. Given the lack of evidence related to levocarnitine dosing in PN, the findings of this study may serve as a general framework for weight-based levocarnitine dosing.

There were several limitations to this study. First, the primary outcome of trends in triglyceride levels was evaluated using piecewise linear regression, which uses estimated means, assumes linearity, and is sensitive to outliers. In this regression model, the strength of the relationship between triglyceride levels and time in the levocarnitine and control groups was low, and prospective evaluation with systematic capture of triglyceride levels is warranted. The retrospective, observational study design limits the ability to establish cause-and-effect relationships between the addition of levocarnitine in PN and triglyceride levels as well as draw direct comparisons between the levocarnitine and control groups. Additionally, the single-center design limits the generalizability of these results, particularly given variable lipid injection emulsion adjustment practices in hospitalized adult patients at other institutions. Although an elevated triglyceride level in this study was defined as ≥175 mg/dl in accordance with AHA/ACC criteria, that cutoff may not prompt a triglyceride-altering intervention in clinical practice. PN history and whether patients were receiving levocarnitine in PN prior to admission was not assessed. Notably, liver function tests were not evaluated and, independent of triglyceride levels, may have been a driver both for reducing lipid injectable emulsion in PN and adding levocarnitine to PN. Finally, we did not assess levocarnitine addition to PN without any changes in lipid injectable emulsion, which, if effective, may be a more desirable approach because it would not affect caloric intake.

CONCLUSION

In this study of hospitalized adult patients with hypertriglyceridemia who had a reduction in lipid injectable emulsion dose in PN, the addition of levocarnitine in PN was associated with a greater rate of improvement in preintervention to postintervention triglyceride levels, though there was no significant difference in triglyceride values at 30 days' postintervention. The median weight-based dose was 8.0 mg/kg based on ideal body weight, or adjusted body weight if BMI $\geq$ 30 kg/m^2.

AUTHOR CONTRIBUTIONS

Andrew S. Jarrell contributed to the conception of the research. All authors contributed to the design of the research. Traci M. Grucz and Andrew S. Jarrell contributed to the acquisition of the data. Traci M. Grucz, Kenneth M. Shermock, Andrew S. Jarrell, and Jessica Crow contributed to the analysis of the data. All authors contributed to the interpretation of the data. Traci M. Grucz drafted the manuscript. All authors critically revised the manuscript, read and approved the final manuscript, and agree to be fully accountable for ensuring the integrity and accuracy of the work.

CONFLICT OF INTEREST

None declared.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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