

The clinical impact of L-carnitine deficiency on secondary hyperparathyroidism and phosphocalcium levels in hemodialysis women

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SUMMARY

Background: Secondary Hyperparathyroidism (sHPT) is a common complication in patients with chronic kidney failure, resulting from an imbalance in calcium and phosphate levels due to renal dysfunction. L-Carnitine deficiency, a key compound in energy metabolism and mitochondrial function, is believed to contribute to these metabolic disturbances.

Objectives: This study aims to evaluate the relationship between L-Carnitine deficiency and secondary hyperparathyroidism, focusing on its impact on calcium and phosphate levels in patients with kidney failure undergoing hemodialysis.

Material and methods: The study included forty women with chronic kidney failure undergoing dialysis, aged between 21 and 62 years. The study was conducted on the same group and compared before and after treatment. Samples were collected for each group in August 2024 and March 2025. PTH, calcium, and phosphate levels were analyzed for the group of patients before treatment and 3-4 months after treatment.

Results: The findings revealed a significant association between L-Carnitine deficiency and elevated PTH levels, suggesting an exacerbation of secondary hyperparathyroidism. Additionally, women with L-Carnitine deficiency exhibited lower calcium levels and significantly higher phosphate levels, potentially increasing the risk of mineral and bone disorders related to kidney disease.

Conclusion: This study suggests that L-Carnitine deficiency may contribute to calcium and phosphate imbalances, leading to the progression of sHPT in patients with kidney failure. Correcting L-Carnitine deficiency through supplementation could be a potential strategy for improving mineral and bone disorder management in these patients. Further research is needed to determine the clinical benefits of L-Carnitine therapy in kidney failure women.

Keywords: L-Carnitine; Hyperparathyroidism; Calcium; Phosphate; Kidney disease

INTRODUCTION

Secondary Hyperparathyroidism (sHPT) is a common complication in patients with chronic kidney disease (CKD) [1], resulting from dysregulated mineral metabolism, particularly involving calcium (Ca^{2+}) and phosphate (PO_4^{3-}) due to impair renal function. The kidneys play a crucial role in maintaining mineral homeostasis and activating 1,25-dihydroxyvitamin D (calcitriol) [2,3], which is essential for calcium absorption. As kidney function declines the production of active vitamin D decreases, leading to hypocalcemia. In response, the parathyroid glands increase the secretion of Parathyroid Hormone (PTH) in an attempt to restore calcium balance, which can result in parathyroid hyperplasia and excessive PTH secretion, a hallmark of sHPT [4].

Furthermore, hyperphosphatemia, a direct consequence of reduced renal phosphate excretion, serves as an additional stimulus for excessive PTH secretion [5]. This dysregulated phosphate-calcium balance contributes to Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD), increasing the risk of bone demineralization, vascular calcification, and cardiovascular complications in affected patients [6].

On the other hand, L-Carnitine, a vital compound involved in fatty acid metabolism and mitochondrial bioenergetics, plays a key role in energy production by facilitating the transport of long-chain fatty acids into the mitochondria for β -oxidation [7]. Several studies have shown that L-Carnitine deficiency is prevalent among CKD patients, particularly those undergoing Maintenance Hemodialysis (MHD). This deficiency is thought to exacerbate oxidative stress, chronic inflammation, and metabolic dysregulation, potentially influencing parathyroid function and disrupting calcium-phosphate homeostasis [8,9].

Emerging evidence suggests a correlation between L-Carnitine deficiency and altered parathyroid hormone regulation, along with imbalances in calcium and phosphate levels in CKD patients. For instance, research has indicated that patients with CKD experience endocrine disturbances [10], including abnormalities in both thyroid and parathyroid function, with concomitant hypocalcemia and hyperphosphatemia, which may

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further drive the progression of sHPT. Based on these findings, L-Carnitine deficiency may contribute to the pathophysiology of secondary hyperparathyroidism by exacerbating calcium and phosphate dysregulation [11].

AIMS OF THE STUDY

L-Carnitine levels in hemodialysis patients and determine their correlation with the severity of secondary hyperparathyroidism (sHPT).

MATERIALS AND METHODS

The Imam Al-Sadiq Teaching Hospital in the Babil Governorate, Division of the Industrial College (dialysis), was the site of a case-control study. The period of data collection was August 2024–March 2025. The study population consisted of 40 female patients with chronic kidney disease who were receiving renal dialysis for three to four months to a year. The patients were at the age of 21 to 62 and were spread over different periods of time. Most women had diabetes, high blood pressure and kidney stones. Handling and finishing stages, which took place three months after the end of the therapy, were two studies. The daily dose of 1600 mg was taken oral as a capsule as part of the treatment. Each dialysis patient received a medical examination and a quick questionnaire, which asked about his height, weight, age, gender, length of the disease, the number of weekly dialysis treatment and the presence of further diseases. Data on the existence of chronic or hereditary diseases,

using SPSS software, were also collected and stored in computerized systems.

Statistical analysis

Statistical analysis was carried out using SPSS version 27. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (Means $\pm$ SD). Paired t-test was used to compare means for two paired readings. McNemar test was used to compare paired categorical variables. A p-value of ≤ 0.05 was considered as significant.

RESULTS

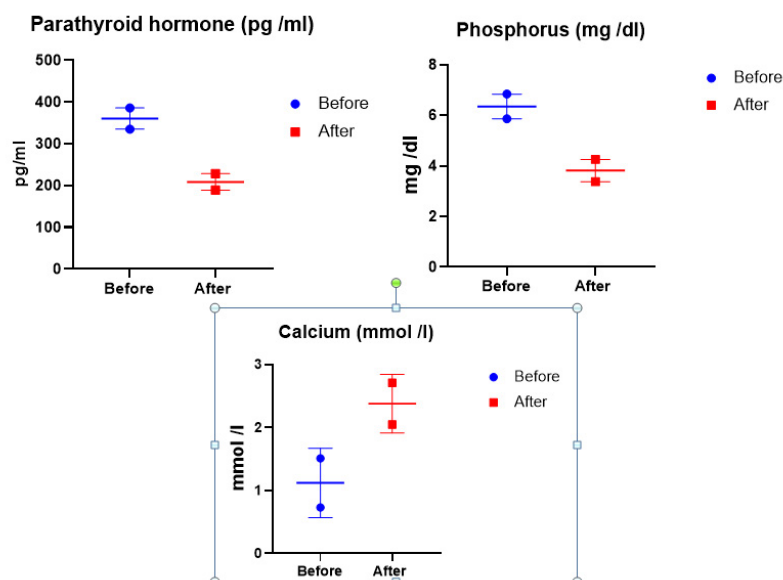
According to the findings of our study, the distribution of patients who are undergoing hemodialysis is presented in accordance with their socio-demographic characteristics, which include their current age and gender. The mean age of the patients was found to be 36.93 ± 10.99 , with the oldest patient being 21 years old and the youngest being 62 years old. The majority of patients (N=25, 62.5%) were male (Tab. 1.).

After three months of therapy with L-carnitine, a statistically significant decrease in the mean levels of phosphorus (mg/dl) and parathyroid hormone (pg/ml) was found in patients who were receiving hemodialysis. On the other hand, our findings revealed that individuals receiving hemodialysis had a considerable mean rise of calcium (measured in millimoles per liter) (Fig. 1. and Tab. 2.).

Tab. 1. Distribution of patients according to the gender.

Variables	(Mean $\pm$ SD)	Minimum	Maximum
Age (years)	(36.93 $\pm$ 10.99)	21	62
Gender	Male	Female	Total
	25 (62.5%)	15 (37.5%)	40 (100%)

Fig. 1. The mean differences of parathyroid hormone before and after treatment.



Tab. 2. The mean differences of parathyroid hormone before and after treatment.

Study variable	Periods of assessment		P-value
	Before treatment	After treatment	
Parathyroid hormone (pg/ml)	360.60 $\pm$ 25.37	208.68 $\pm$ 19.77	<0.001*
Phosphorus (mg/dl)	6.35 $\pm$ 0.49	3.82 $\pm$ 0.44	<0.001*
Calcium (mmol/l)	1.12 $\pm$ 0.39	2.38 $\pm$ 0.33	<0.001*

*P value ≤ 0.05 was significant.

DISCUSSION

Our study found that after three months of therapy with L-carnitine, a statistically significant decrease in the mean levels of phosphorus (mg/dl) and parathyroid hormone (pg/ml) was reported in patients who were receiving hemodialysis. On the other hand, our findings demonstrated that people having hemodialysis experienced a large mean elevation of calcium

Patients with chronic renal failure suffer from an increase in hospitalization and need dialysis due to the loss of kidney function [12]. They are more likely to suffer from low levels of carnitine which is one of the factors responsible for the various disorders that chronic kidney disease patients suffer from, such as anemia, muscle weakness, low pressure during dialysis, and cardiomyopathy. Maintaining the level of carnitine in the serum or tissues can reduce these disorders [13,14].

Secondary hyperparathyroidism and enlarged parathyroid glands are common complications in patients with renal failure due to the inability of the affected kidney to produce 1-alpha hydroxylase and the inability of the patient to convert vitamin D to its active form [15]. Decreased calcitriol in the blood, moderate decrease in ionized calcium, inability of the kidneys to excrete phosphate, and its rise in the blood contribute to the increase in the synthesis and secretion of PTH hormone with the progression of the disease [16]. The increase in the level of PTH is associated with

the occurrence of hyperparathyroidism in hemodialysis patients which is a physiological disease caused mainly by hyperphosphatemia and hypocalcemia [17].

L-Carnitine supplementation was used to investigate its effect on secondary hyperparathyroidism in hemodialysis patients and found that L-Carnitine had little effect and non-significant expression in patients with secondary PTH hyperactivity and this is inconsistent with the results of the current study [11,18].

Pre-treatment, patients with elevated PTH levels had hyperparathyroidism and elevated serum phosphorus levels [18], which is believed to be caused by an increase in the secretion of bone cells and not due to a decrease in kidney filtering [19]. Also, most of the dialysis patients were suffering from calcium deficiency due to irregular metabolism, as hyperparathyroidism stimulates the bone to release bone from the calcium in the blood [20].

CONCLUSION

This study suggests that L-Carnitine deficiency may contribute to calcium and phosphate imbalances, leading to the progression of sHPT in patients with kidney failure. Correcting L-Carnitine deficiency through supplementation could be a potential strategy for improving mineral and bone disorder management in these patients. Further research is needed to determine the clinical benefits of L-Carnitine therapy in kidney failure women.

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