

Evaluation of the Effects of 25(OH)D Supplementation on Biochemical and Anthropometric Parameters of Patients Undergoing Hemodialysis

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ABSTRACT

Introduction: Deficiency of Vitamin D is prevalent in patients with chronic kidney disease (CKD). Studies have demonstrated that deficiency of this vitamin may cause insulin resistance. This work aimed to assess and compare the biochemical and anthropometric parameters of 25(OH)D supplemented and non-supplemented patients from a hemodialysis clinic in natal/RN. **Materials and Methods:** A quantitative, cross-sectional study was conducted with 32 CKD patients under hemodialysis therapy. **Results:** It was found that in patients with deficient 25(OH)D levels, glucose and glycated hemoglobin (HbA1c) levels were high. In patients with optimal levels, who take 25(OH)D supplements, it was observed that the serum levels of glucose, HbA1c, total cholesterol, and parathyroid hormone tend to diminish, reducing the probability of triggering progression of the chronic kidney disease. **Conclusion:** Supplementation of 25(OH)D can be an alternative to improve biochemical and anthropometric markers of these patients.

Key words: Chronic kidney disease, insulin resistance, Vitamin D deficiency

INTRODUCTION

Chronic kidney disease (CKD) has been associated with the development and/or progression of hypertension, diabetes, obesity, and cardiovascular diseases and can advance to end-stage renal disease (ESRD). It is classified into five stages according to the glomerular filtration rate (GFR) (Hill *et al.*, 2016).^[1]

When a patient is at CKD Stage 5, his GFR approaches the level of 15 ml/min/1.73 m². At this point, renal replacement therapy (RRT) is needed. There are some kinds of RRT, namely, peritoneal dialysis hemodialysis (HD) and kidney transplantation (Pacilio *et al.*, 2016).^[2]

Malnutrition is common in end-stage renal disease patients undergoing hemodialysis. A deficient nutritional status is common in these patients and is due to protein-energy loss, a complication that is associated with increased mortality in CKD patients. This disease has an influence on the nutritional markers, and it is common to observe in these individuals higher body mass index (BMI) levels and serum lipids, which are associated with negative outcomes in this population. It is known that 25(OH)D deficiency is prevalent in patients with this disease.^[3]

Deficiency of 25(OH)D triggers high levels of parathyroid hormone (PTH), which, in turn, causes high bone turnover, exacerbates osteopenia, resulting in cortical bone loss

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and osteoporosis pathogenesis. In addition to having been associated with increased levels of serum glucose and glycated hemoglobin (HbA1c), studies have demonstrated that Vitamin D deficiency can play a key role in the development of diabetes mellitus, caused by insulin resistance and increased glucose levels.

The correlation of 25(OH)D supplementation with decreased levels of total cholesterol and lipid fractions has been studied. It is believed that optimal levels of 25(OH)D improve insulin resistance and prevent vascular calcification.^[4]

MATERIALS AND METHODS

Type of the research study

This is a quantitative, observational, cross-sectional study and non-probability sampling with patients with chronic renal disease undergoing hemodialysis in a private clinic and accepted by the ethics committee.

Universe and sample

The study considered eligible for inclusion CKD patients aged 18 years and over under hemodialysis therapy, with no clinical interurrences and fully capable of communicating and responding to the questionnaires administered by the interviewer. The patients who accepted to participate in the survey signed the free and informed consent form. The patients with visual or neurological problems, incapable of responding to the examiner, wheelchair users, and patients on stretchers were excluded from the study.

Instrument and technique for data collection

Data were collected in a private hemodialysis clinic at the same room where the hemodialysis sessions STR carried out.

Anthropometric assessment

Anthropometric assessment was carried out after the hemodialysis session. The patients were weighted on a digital scale (Toledo) with a maximum capacity of 200 kg, wearing the least amount of clothes as possible and bare feet. The stature was measured with a stadiometer with the patient standing upright, the arms along the body, and the back against the wall in Frankfurt plane.

Based on the weight and stature measures, BMI was calculated (kg/m^2), which was classified according to the World Health Organization^[5] for adults, and Lipschitz (1997) for the elderly.

Biochemical testing

It is part of the clinic's practice to perform biochemical tests to assess and control kidney disease, so the results of semiannual tests for 25(OH)D, total cholesterol, PTH,

ferritin, transferrin, glucose, and HbA1c were collected from the patient's medical records. The reference values used for HbA1c and glucose were those proposed by the American Diabetes Association (2017), HbA1c being (VR = 4.5–5.6%), and glucose (VR \leq 100 mg/dL).

Serum levels of 25(OH)D, transferrin, ferritin, and PTH were assessed according to the criteria used by Faridi *et al.*^[4] who considered 25(OH)D levels adequate when they are above 30 ng/mL. Levels of 25(OH)D between entire 20 and 30 ng/mL were considered insufficient and below 20 ng/mL were defined as a diagnosis of 25(OH)D deficiency. For the other biochemical parameters evaluated, the following values were considered adequate: PTH (VR = 11 to 65 pg / mL), Cholesterol total (VR = 190mg / dL), Transferrin (VR = 250 - 380 mg / dL) and Ferritin (VR = 11 to 306 ng / ml).

Groups division

The patients were divided into two groups. The first group comprised patients who did not use 25(OH)D supplementation (43% of the sample), and the second group consisted of patients who already used 25(OH)D supplementation, according to medical prescription (57% of the sample). These patients were then subdivided into 25(OH) deficient, 25(OH) D intermediate, and 25(OH)D optimal groups.

Statistical treatment

The results were expressed as mean and standard deviation, structured, and analyzed using descriptive analysis with the statistical package version 7.0 for Macbook. Descriptive statistics were carried out to characterize the study population. Data were shown as means and standard deviation, according to the nature of the variable.

Student's *t*-test was used for independent samples to check for difference of means between the quantitative variables of the nutritional status and biochemical data. One-way ANOVA was also performed to compare the means of the quantitative variables of nutritional status and the biochemical data.

For all hypothesis tests, statistically significant $P < 0.05$ was accepted.

RESULTS

The study was carried out with 32 individuals undergoing dialysis treatment, who were divided into two groups: One supplemented with 25(OH)D and the other non-supplemented.

Table 1 shows the anthropometric parameters of the patients who did not take Vitamin D supplements. It was found that that there was only a significant difference ($P < 0.05$) for the variable weight, which had the following results: For the 25(OH)D intermediate group, mean weight was 57.58 ± 9.66 kg, and

for the 25(OH)D optimal group, weight was higher (69.18 ± 12.19) than in the intermediate group. No individual exhibited serum levels of 25(OH)D below 20 ng/mL.

In the 25(OH)D intermediate group, the mean age was 51.71 ± 20.99 years, and for the 25(OH)D optimal group, it was 46.13 ± 14.35 years, but with no significant difference between the samples ($P = 0.192$). Regarding stature, the 25(OH)D optimal group indicated a higher mean value (1.63 ± 0.11) compared to the 25(OH)D intermediate group (1.53 ± 0.05), but no significant difference was found between the samples. For BMI, the 25(OH)D intermediate group showed a higher mean value (25.41 ± 2.10 kg/m²) compared to the 25(OH)D optimal group (24.26 ± 5.16 kg/m²), but with no significant difference between the samples ($P = 0.219$).

Table 2 describes the distribution of the mean values of the anthropometric variables of the patients who were taking 25(OH)D supplements. The results for the group of Vitamin D-supplemented patients indicated a significant difference for the variables age, weight, and BMI. The mean age of the Vitamin D-deficient group was 49.25 ± 16.56 years, in the intermediate group, it was 58.50 ± 12.01 years, and in the optimal group, it was 46.50 ± 4.95 years. The younger individuals, in the optimal group, obtained a better level of serum 25(OH)D when compared to the Vitamin D-deficient and intermediate groups.

As for the weight of individuals in the Vitamin D-supplemented group, the results found were 49.15 kg \pm 4.60 (deficient), 62.38 kg \pm 13.04 (intermediate), and 67.45 kg \pm 8.29 (optimal). It could be seen that the higher the weight, the higher the serum levels of 25(OH)D. For BMI, the following

results were obtained: 20.85 kg/m² \pm 6.75 for the Vitamin D-deficient group, 21.95 kg/m² \pm 6.75 for the intermediate group, and 25.83 kg/m² \pm 4.33 for the optimal group. We can see that the lower the BMI, the lower the 25(OH)D levels in the blood ($P = 0.002$).

With respect to stature, in the group of 25(OH)D-deficient individuals, mean height was 1.61 ± 0.08 m; in the 25(OH)D intermediate group, 1.55 ± 0.10 m, and in the 25(OH)D optimal group, the mean height of the patients was 1.54 ± 0.08 m. It was observed that the optimal group was made up of individuals with lower stature, but without significant difference between the samples.

Table 3 describes the biochemical parameters of patients not supplemented with Vitamin D. PTH was above the reference values in both groups, higher in the 25(OH)D intermediate group with significant difference between the samples ($P = 0.001$). By observing the biochemical parameters of 25(OH)D in patients not supplemented with this vitamin, we can see that both groups presented adequate serum values, but better in the optimal group (39.63 ± 5.48) ($P = 0.0001$).

Serum ferritin levels were high in both 25(OH)D intermediate group (367.00 ± 80.00) and in the 25(OH)D optimal group (329.80 ± 84.10), but with no significant difference between the samples ($P = 0.060$). Serum transferrin levels in the 25(OH)D intermediate group (265.00 ± 17.26) were within the recommended range, but in the 25(OH)D optimal group (134.3 ± 52.42), the result was below the normal range ($P = 0.0001$).

Total cholesterol was high in both groups, higher in the 25(OH)D optimal group (237.50 ± 11.68), with difference

Table 1: Distribution of anthropometric variables of CKD patients undergoing HD and not supplemented with 25(OH)D

Parameters of non-vitamin-D supplemented patients	Deficient 25(OH)D ng/mL ≤ 20	Intermediate 25(OH)D ng/mL $\geq 20-30$	Optimal 25(OH)D ng/mL ≥ 30	P-value
Age	***	51.71 ± 20.99	46.13 ± 14.35	0.192
Weight (kg)	***	57.58 ± 9.66	69.18 ± 12.19	<0.0001
Stature (m)	***	1.53 ± 0.05	1.63 ± 0.11	0.281
BMI (kg/m ²)	***	24.26 ± 5.16	25.41 ± 2.10	0.219

***There were not results for this group. BMI: Body mass index

Table 2: Distribution of anthropometric variables of 25(OH)D-supplemented patients with CKD under hemodialysis therapy

Parameters of supplemented patients	Deficient 25(OH)D ng/mL ≤ 20	Intermediate 25(OH)D ng/mL $\geq 20-30$	Optimal 25(OH)D ng/mL ≥ 30	P-value
Age (years)	49.25 ± 16.56	58.50 ± 12.01	46.50 ± 4.95	<0.001
Weight (kg)	$49.15 \pm 4.60^*$	62.38 ± 13.04	67.45 ± 8.29	<0.001
Stature (m)	1.61 ± 0.08	1.55 ± 0.10	1.54 ± 0.08	0.732
BMI (kg/m ²)	20.85 ± 6.75	21.95 ± 6.75	25.83 ± 4.33	<0.002

BMI: Body mass index, CKD: Chronic kidney disease

between the samples ($P = 0.0001$). Serum values of HbA1c in both groups were above the recommended values, being higher in the intermediate group ($P = 0.0032$). Serum glucose values were high in the intermediate group (219.50 ± 20.27), but in the optimal group, the mean value was lower (104.80 ± 18.85), with significant difference between the samples ($P = 0.0022$).

Table 4 describes the biochemical parameters of patients receiving hemodialysis who take Vitamin D supplements. PTH was high in the three groups, higher in the 25(OH)D-deficient group ($P = 0.001$). Vitamin D serum concentrations in the 25(OH)D-deficient group were below the recommended levels (17.50 ± 2.41), and in the 25(OH)D intermediate and optimal groups, they were within the recommended values ($P = 0.0001$).

Ferritin was high in the 25(OH)D intermediate group (235.70 ± 153.20) and in the 25 (OH)D optimal group (281.60 ± 444.90), being higher in the latter ($P = 0.0001$). Total cholesterol concentrations in the 25(OH)D-deficient group (272.70 ± 20.26) and in the 25(OH)D intermediate group (215.00 ± 17.07) were considered high, but in the 25(OH) D optimal group (189.50 ± 13.44), the result is according to recommendations ($P = 0.0001$).

The levels of HbA1c and glucose decreased significantly in the 25(OH)D supplemented patients. It can be seen that the higher the serum 25(OH)D levels, the lower the HbA1c and glucose levels.

DISCUSSION

In the non-supplemented group, the individuals with optimal 25(OH)D levels exhibited a higher weight per kilogram ($P = 0.0001$). This result was also observed in the 25(OH) D supplemented group, where the average weight of the individuals assessed in the 25 (OH) D deficient group (49.15 ± 4.60 kg) was lower than in the intermediate (62.38 ± 13.04 kg) and optimal (67.45 ± 8.29 kg) groups. In a study carried out by Costa *et al.*, 2017,^[6] the individuals with 25(OH)D levels considered deficient had an average weight per kilogram higher than in individuals with adequate levels of 25(OH)D.

The BMI of individuals who use 25(OH)D supplementation increased exponentially according to the serum 25(OH) D levels. In the optimal group, the mean BMI was rated as overweight according to the 1995 WHO's criteria. This result was contrary to that found by Stokić *et al.*^[7] who observed

Table 3: Distribution of biochemical variables in CKD patients under hemodialysis therapy and not supplemented with 25(OH)D ng/mL

Parameters	Deficient 25(OH) D ng/mL < 20	Intermediate 25 (OH) D ng/mL ≥20–30	Optimal 25 (OH) D ng/mL ≥30	P-value
PTH (pg/dL)	***	927.60±204.00	688.60±227.00	<0.0001
Vitamin D	***	25.50±3.87	39.63±5.48	<0.0001
Ferritin (ug/mL)	***	367.00±80.00	329.80±84.10	0.060
Transferrin (mg/dL)	***	265.00±17.26	134.3±52.42	<0.0001
Total cholesterol	***	212.00±8.37	237.50±11.68	<0.0001
Glycated hemoglobin	***	7.37±1.05	6.10±2.26	0.0032
Glucose	***	219.50±20.27	104.80±18.85	0.0022

PTH: Parathyroid hormone, CKD: Chronic kidney disease

Table 4: Distribution of biochemical variables in CKD patients under hemodialysis therapy receiving supplementation of 25(OH)D ng/mL

Parameters	Deficient 25 (OH) D ng/mL <20	Intermediate 25 (OH) D ng/mL >20–30	Optimal 25 (OH)D ng/mL >30	P-value
PTH (pg/dL)_	740±252.00*	519.50±223.90	512.40±232.50	<0.001
Vitamin D	17.50±2.41	23.33±2.58	36.00±2.83	<0.0001
Ferritin (ug/mL)	156.00±108.70	235.70±153.20	281.60±444.90	<0.0001
Transferrin (mg/dL)	257.00±11.26	157.30±16.20	150.90±5.45	0.0032
Total cholesterol	272.70±20.26	215.00±17.07	189.50±13.44	<0.0001
Glycated hemoglobin	7.00±1.57	6.20±1.43	5.00±1.14	<0.0001
Glucose	200.50±14.85	108.00±5.27	99.50±2.70	<0.0001

PTH: Parathyroid hormone, CKD: Chronic kidney disease

low 25(OH)D levels in individuals with BMI above the adequate limits.

In a study conducted by Alkhatatbeh *et al.*,^[8] they found a correlation between 25(OH)D deficiency and the development of non-communicable chronic diseases such as high blood pressure, cardiovascular diseases, diabetes mellitus, and metabolic syndrome. In the present study, we can see predominance of 25(OH)D-deficient individuals. Deficiency of this vitamin has been associated with diabetes mellitus in CKD patients in various studies.

A correlation was observed between low serum levels of 25(OH)D and increased PTH levels both in the 25(OH)D supplemented group and in the non-supplemented group. Similar outcome was found by Kroll *et al.*^[9] who investigated the seasonal relationship between 25(OH)D and PTH. According to Clarke *et al.*,^[10] 25(OH)D and PTH act directly on calcium homeostasis and bone metabolism by regulating them, increasing turnover and resulting in the release of calcium and phosphate into the circulation, also improving renal tubular reabsorption of calcium and stimulating the kidney to convert 25-hydroxyvitamin D to the active 1.25-di-hydroxyvitamin D.

In their study, Kramer *et al.*^[11] observed 494 women undergoing serial metabolic characterization and found that Vitamin D deficiency levels with increased PTH levels were independent predictors of β -cell dysfunction, insulin resistance, and glycemia.

In the Vitamin D-supplemented group, it was found that the higher the serum 25(OH)D levels, the higher the ferritin levels ($P = 0.0001$). According to Kang *et al.*,^[12] ferritin is an acute-phase protein that enhances the inflammatory state. In patients on HD treatment, the levels of ferritin tend to increase due to oxidative stress, aggravating the kidney injury. In this study, the individuals of the supplemented group who presented higher ferritin levels (281.60 ± 444.90 ng/mL) also presented higher BMI (25.83 ± 4.33 kg/m²).

The previous studies have reported that high levels of serum ferritin are associated with insulin resistance syndrome, hypertension, dyslipidemia, obesity, and metabolic syndrome as risk factors for CKD patients.

Concerning transferrin, it can be shown in Tables 3 and 4 that increased serum 25(OH)D levels occur when transferrin levels are lower. The lowest mean value of transferrin was found in the non-supplemented group (134.3 ± 52.42 ng/mL). Low levels of this transport protein can be found in patients with kidney injury in line with their acute or chronic inflammatory state. In the present study, high ferritin levels and low transferrin levels were similar to the ones found by Giachini *et al.*,^[13] which explains that high ferritin levels

present in the inflammatory state results in low transferrin saturation.

Total serum levels of cholesterol were lower in the 25(OH)D supplemented group, showing a relation with serum 25(OH)D levels. In the 25(OH)D supplemented group, we can see that the higher the serum 25(OH)D levels, the lower the total cholesterol levels. However, in the non-supplemented group, the result was the opposite, that is, the higher the 25(OH)D level, the higher the serum level of total cholesterol (p -value 0.0001). Hattori *et al.*^[14] in a study conducted with animals, showed that higher levels of total cholesterol can increase the rate of progression of renal disease, and a diet rich in cholesterol which may cause macrophage infiltration and formation of foam cells.

Concentrations of glucose and HbA1c were significantly higher in individuals with 25(OH)D <20 ng/mL and 25(OH)D 20–30 ng/mL compared with individuals with 25(OH)D >30 ng/mL supplemented and non-supplemented. Optimal 25(OH)D levels were associated with a significant improvement of the serum values of HbA1c and glucose. 25(OH)D deficiency is related with insulin secretion, insulin resistance, and dysfunction of β -cells in pancreas.

Kramer *et al.*^[11] mentioned that administration of this vitamin may restore glucose-stimulated insulin secretion and contributed to the survival of β -cells by modulating the cytokines generation and effects. 25(OH)D has the function of regulating calbindin, a systolic calcium-binding protein found in pancreas β -cells, where it acts as a modulator of depolarization-stimulated insulin secretion through the regulation of intracellular calcium.

In the study of Nakashima *et al.*,^[15] 25(OH)D deficiency was associated with modulation of the immune system and cytokines secretion responsible for inhibiting NF- κ B activity. NF- κ B is a transition factor responsible for the expression of multiple pro-inflammatory and pro-atherogenic cytokines. 25(OH)D promotes a decrease of NF- κ B, consequently diminishing the inflammatory cytokines, thus regulating insulin and improving the glucose levels.

A prospective cohort study carried out in the United Kingdom showed that 25(OH)D concentrations in non-diabetic patients were inversely related to the risk of hyperglycemia and insulin resistance. There are few evidences available on the relationship between HbA1c levels and clinical outcomes in patients with advanced CKD. Reported in their study that HbA1c predicts increased kidney injury in patients at CKD Stages 3 and 4, but further studies are necessary to elucidate the effects of HbA1c on patients at Stage 5.

Therefore, the use of 25(OH)D supplementation suggests reduced levels of glucose and HbA1c in CKD patients on

hemodialysis. We could observe that low levels of 25(OH)D may be a factor of risk for DM progression.

CONCLUSION

The anthropometric profile of patients on hemodialysis showed that the BMI of individuals who presented optimal 25(OH)D levels tends to increase, when compared with individuals who had deficient 25(OH)D levels. The mean BMI of individuals with optimal 25(OH)D levels was scored as overweight.

In this study, it was concluded that patients with deficient 25(OH)D presented high levels of glucose and HbA1c. Patients with optimal 25(OH)D levels who use supplementation of this vitamin tend to exhibit decreased serum levels of glucose, HbA1c, total cholesterol, and PTH.

Supplementation of 25(OH)D can be an alternative to improve biochemical and anthropometric markers of chronic diseases to prevent more severe complications of the renal disease. However, due to the complexity and importance of this subject, further studies are necessary to elucidate the therapeutic benefits of 25(OH)D in the control of kidney disease.

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