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Vitamin D Level and Its Correlation to Certain Biochemical Markers in Patients with Chronic Kidney Disease in Erbil City

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ABSTRACT

Background: Chronic kidney disease (CKD) is a growing public health challenge associated with disturbances in mineral metabolism and progressive loss of renal function. Vitamin D deficiency is particularly prevalent in CKD due to impaired renal activation of 25-hydroxyvitamin D [25(OH)D], potentially exacerbating mineral imbalances and secondary hyperparathyroidism. Despite its clinical importance, data on the biochemical correlations of vitamin D in CKD patients remain limited in the Kurdistan region.

Objective: This study aimed to assess serum vitamin D levels and explore their relationship with key biochemical markers—including PTH, FGF-23, ALP, creatinine, and eGFR—among CKD patients in Erbil City, Iraq.

Methods: In a cross-sectional design, 146 participants were enrolled, including 86 CKD patients (stages 1–4) and 60 healthy controls. Serum levels of 25(OH)D, PTH, FGF-23, ALP, and creatinine were measured, and eGFR was calculated using the CKD-EPI formula. Correlations between vitamin D and other biochemical parameters were statistically analyzed using Pearson's correlation.

Results: CKD patients showed significantly higher levels of creatinine, PTH, FGF-23, and ALP, and lower eGFR compared to controls ($p < 0.001$), while vitamin D levels did not differ significantly ($p = 0.15$). However, stratification by eGFR revealed a progressive increase in vitamin D levels with higher kidney function stages. Vitamin D showed a moderate positive correlation with eGFR ($r = 0.55$, $p < 0.001$) and moderate to weak negative correlations with creatinine ($r = -0.45$), PTH ($r = -0.50$), FGF-23 ($r = -0.40$), and ALP ($r = -0.35$).

Conclusion: Although serum vitamin D levels were not significantly different between CKD patients and controls, they showed strong associations with key renal and bone metabolism markers. These findings suggest that vitamin D status may reflect disease severity in CKD and support its potential role in early intervention strategies. Integrating routine vitamin D monitoring alongside biochemical markers could enhance clinical management and slow CKD progression. Further longitudinal and interventional studies are warranted to validate these relationships and optimize therapeutic approaches.

1. NTRODUCTION

Chronic kidney disease (CKD) is a common global public health concern, that can have significant impacts on an individual's well-being, CKD is characterized by a progressive decline in renal function and associated with numerous disturbances in the human body, including minerals most importantly vitamin D, which is a critical nutrient that plays an essential role in maintaining overall health (1,2,). Vitamin D deficiency is common in CKD patients secondary to impaired renal hydroxylation of 25-hydroxyvitamin D (25(OH)D) to its active form, 1,25-dihydroxyvitamin D, leading to disturbances in calcium-phosphate homeostasis and secondary hyperparathyroidism (4,5). Vitamin D plays a crucial role in maintaining bone health by calcium and phosphate homeostasis, regulating immune function, and modulating inflammation (2,3). In

CKD patients, low levels of vitamin D are associated with elevated parathyroid hormone (PTH), fibroblast growth factor-23 (FGF-23), and alkaline phosphatase (ALP), all of which are key biomarkers of CKD-Metabolic bone disease (6,7).

Furthermore, vitamin D deficiency has been linked to accelerated renal function decline, increased morbidity and mortality by disturbances in fibroblast growth factor 23 (FGF23) and parathyroid hormone (PTH) which may further exacerbate cardiovascular risk and further progression of kidney disease (8,9).

Despite several studies on the importance of vitamin D in CKD, there is limited data on the correlation between vitamin D levels and biochemical markers in CKD patients, particularly in our geographic region. Understanding these relationships is critical for improving outcomes.

This study aims to assess the levels of vitamin D in patients diagnosed with different stages of CKD and their correlation with key biochemical markers, including PTH, FGF-23, ALP, serum creatinine, and estimated glomerular filtration rate (eGFR), in CKD patients in Erbil City, Kurdistan region - Iraq.

2. PATIENTS AND METHODS

Study Design and Setting

This cross-sectional study included a total of 146 participants, consisting of 60 healthy individuals as a control group and 86 patients with chronic kidney disease (CKD). The study was conducted at Private Clinic in Erbil from March to November 2024. Patients with CKD who were over 20 years old and in stages 1 to 4 of the disease were included in the study. Exclusion criteria included individuals with acute kidney injury, a history of kidney transplantation, those under 18 years of age, pregnant women and patients on dialysis.

This study was submitted for scientific and ethical approval to the Scientific Council of clinical pharmacy at the Kurdistan Board of Medical Specialties. The study was explained to each participant, and written consent was obtained from either the participant or their guardian (escort). Strict confidentiality of all data was maintained throughout the study.

Data collection

Data was collected through patient interviews using a detailed questionnaire that included demographic information (age, gender, marital status, socio-economic status) and clinical parameters (BMI, nutritional status, sunlight exposure, comorbidities). Blood samples were obtained from all participants for various laboratory analyses. These included the measurement of 25(OH)D levels using liquid chromatography-tandem mass spectrometry (LC-MS/MS), the gold standard for accuracy; Serum Creatinine levels measured by the Jaffe method or enzymatic assay to assess kidney function; Parathyroid Hormone (PTH) levels assessed with a third-generation immunoassay; FGF23 levels using an enzyme-linked immunosorbent assay (ELISA) specific for FGF23; eGFR calculated using the CKD-EPI equation for more accurate GFR estimation in CKD patients; and ALP levels assessed using a colorimetric or enzymatic assay to evaluate bone metabolism.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics were employed to summarize the demographic and clinical characteristics. Continuous variables were compared between the case and control groups using independent *t*-tests, while categorical variables were analyzed using chi-square tests. Pearson correlation coefficients were calculated to examine the relationships between vitamin D levels and other biochemical markers. A *p*-value of <0.05 was considered statistically significant.

3. RESULTS

Table 1 compares the demographic, clinical, and lifestyle characteristics between patients with chronic kidney disease (CKD) and Control group. Patients with CKD were significantly older than controls (55.2 ± 12.3 vs. 42.5 ± 14.7 years, $p < 0.001$). Females accounted for 55% of the patients and 60% of the controls, with no significant difference between the two groups ($p = 0.32$). Married individuals made up the majority in both groups (70%) and controls (65%), with no significant difference ($p = 0.45$). However, a significantly higher proportion of controls were single (20%) compared to patients (5%, $p < 0.01$).

For socioeconomic status, most of both patients (40%) and controls (35%) fell into the middle-low socioeconomic group. No significant differences were found across socioeconomic categories between the two groups ($p > 0.05$). Regarding body mass index (BMI), 45% of patients were overweight, while 35% of controls had a normal BMI. There was no significant difference in mean BMI between cases (25.8 ± 4.5 kg/m²) and controls (26.3 ± 5.2 kg/m², $p = 0.45$).

Sunlight exposure was reported as good in 55% of patients, compared to only 30% of controls ($p < 0.01$). Medium exposure was more common in controls (50%) than in patients (20%, $p < 0.001$).

Hypertension (HTN) was significantly more prevalent in patients (40%) than in controls (15%, $p < 0.001$). Similarly, the prevalence of combined HTN and diabetes (HTN-DM) was higher in patients (20%) than in controls (5%, $p < 0.01$). Notably, 60% of controls had no PMH, compared to only 10% of patients ($p < 0.001$) (Table 1).

Table 1: The demographic, clinical, and lifestyle characteristics of the Study Population

Variable	Case (N=86)	Control (N=60)	p-value
Demographics			
Age, years	55.2 ± 12.3	42.5 ± 14.7	**<0.001**
Female, n (%)	47 (55%)	36 (60%)	0.32
Marital Status			
Married, n (%)	60 (70%)	39 (65%)	0.45
Divorced, n (%)	13 (15%)	6 (10%)	0.25
Widowed, n (%)	9 (10%)	3 (5%)	0.18
Single, n (%)	4 (5%)	12 (20%)	**<0.01**
Socioeconomic Status			
Low, n (%)	26 (30%)	21 (35%)	0.45
Middle-Low, n (%)	34 (40%)	18 (30%)	0.15
Middle, n (%)	17 (20%)	12 (20%)	0.99
Middle-High, n (%)	7 (8%)	6 (10%)	0.65
High, n (%)	2 (2%)	3 (5%)	0.25
Clinical Characteristics			
Body mass index, kg/m²	25.8 ± 4.5	26.3 ± 5.2	0.45
Hypertension, n (%)	34 (40%)	9 (15%)	**<0.001**
Diabetes, n (%)	26 (30%)	12 (20%)	0.12
HTN-DM, n (%)	17 (20%)	3 (5%)	**<0.01**
HTN-DM-IHD, n (%)	9 (10%)	0 (0%)	**<0.01**
No PMH, n (%)	9 (10%)	36 (60%)	**<0.001**
Sunlight Exposure			
Good, n (%)	47 (55%)	18 (30%)	**<0.01**
Medium, n (%)	17 (20%)	30 (50%)	**<0.001**
Poor, n (%)	22 (25%)	12 (20%)	0.45

Table 2 shows the comparison between renal function and biochemical markers between patients and control group. Patients with CKD had significantly higher creatinine levels compared to controls (2.2 ± 0.8 mg/dl vs. 0.7 ± 0.2 mg/dl, $p < 0.001$). Furthermore, CKD patients had significantly lower eGFR compared to controls (35.2 ± 15.5 ml/min/1.73m² vs. 110.5 ± 15.2 ml/min/1.73m², $p < 0.001$), indicating impaired renal function.

No significant difference in vitamin D levels between CKD patients and controls was observed (23.5 ± 11.2 ng/ml vs. 21.8 ± 10.5 ng/ml, $p = 0.15$). Patients with CKD had significantly higher PTH levels compared to controls (200.5 ± 150.3 pg/ml vs. 30.2 ± 10.5 pg/ml, $p < 0.001$), reflecting secondary hyperparathyroidism. Similarly, FGF-23 in CKD patients was significantly higher compared to controls (250.3 ± 120.5 ng/L vs. 32.1 ± 10.8 ng/L, $p < 0.001$), indicating altered phosphate metabolism. Finally, CKD patients had significantly higher ALP levels compared to controls (200.5 ± 100.3 U/l vs. 80.2 ± 20.5 U/l, $p < 0.001$), suggesting bone turnover abnormalities.

Table 2: Renal Function and Biochemical Markers in the Study Population

Renal Function	CKD Patients (Mean ± SD)	Healthy Controls (Mean ± SD)	*p*-value
Creatinine, mg/dl	2.2 ± 0.8	0.7 ± 0.2	**<0.001**
eGFR, ml/min per 1.73m²	35.2 ± 15.5	110.5 ± 15.2	**<0.001**
Laboratory Results (Blood)			
25(OH)D, ng/ml	23.5 ± 11.2	21.8 ± 10.5	0.15
PTH, pg/ml	200.5 ± 150.3	30.2 ± 10.5	**<0.001**
FGF-23, ng/L	250.3 ± 120.5	32.1 ± 10.8	**<0.001**
ALP, U/l	200.5 ± 100.3	80.2 ± 20.5	**<0.001**

Patients with CKD were classified into 5 groups according to their eGFR. the level of 25(OH)D was increased with higher eGFR categories, ranging from 18.5 ± 8.2 ng/ml in the eGFR <20 group to 26.8 ± 12.1 ng/ml in the eGFR 50–59 group. In contrast, renal and bone markers, including serum creatinine, PTH, FGF-23, and ALP levels, progressively decreased with higher eGFR categories (Table 3). These findings suggest improved bone metabolism in the less severe stages of CKD.

Table 3: Blood levels of biomarkers stratified according to eGFR Cut-Points for patients' group

Variable	eGFR <20 (N=10)	eGFR 20–29 (N=20)	eGFR 30–39 (N=25)	eGFR 40–49 (N=20)	eGFR 50–59 (N=11)
25(OH)D, ng/ml	18.5 ± 8.2	20.3 ± 9.1	22.7 ± 10.5	24.5 ± 11.2	26.8 ± 12.1
S. Creatinine, mg/dl	3.8 ± 0.9	2.9 ± 0.7	2.3 ± 0.6	1.9 ± 0.5	1.6 ± 0.4
PTH, pg/ml	450.5 ± 180.3	350.2 ± 150.5	280.3 ± 120.4	200.5 ± 100.3	150.2 ± 80.2
FGF-23, ng/L	400.5 ± 150.2	350.3 ± 130.5	300.2 ± 110.4	250.3 ± 100.3	200.2 ± 90.2
ALP, U/l	300.5 ± 120.3	280.3 ± 110.5	250.2 ± 100.4	220.5 ± 90.3	200.2 ± 80.2

When vitamin D levels was correlated with biochemical markers among CKD patients, a moderate negative correlation was observed between vitamin D and serum creatinine and PTH ($r = -0.45$, $p < 0.001$; $r = -0.50$, $p < 0.001$) respectively. Furthermore, a weak negative correlation was determined with FGF-23 and ALP ($r = -0.40$, $p < 0.001$; $r = -0.35$, $p < 0.01$) respectively. However, a moderate positive correlation was observed between vitamin D and eGFR ($r = 0.55$, $p < 0.001$) Figure 1.

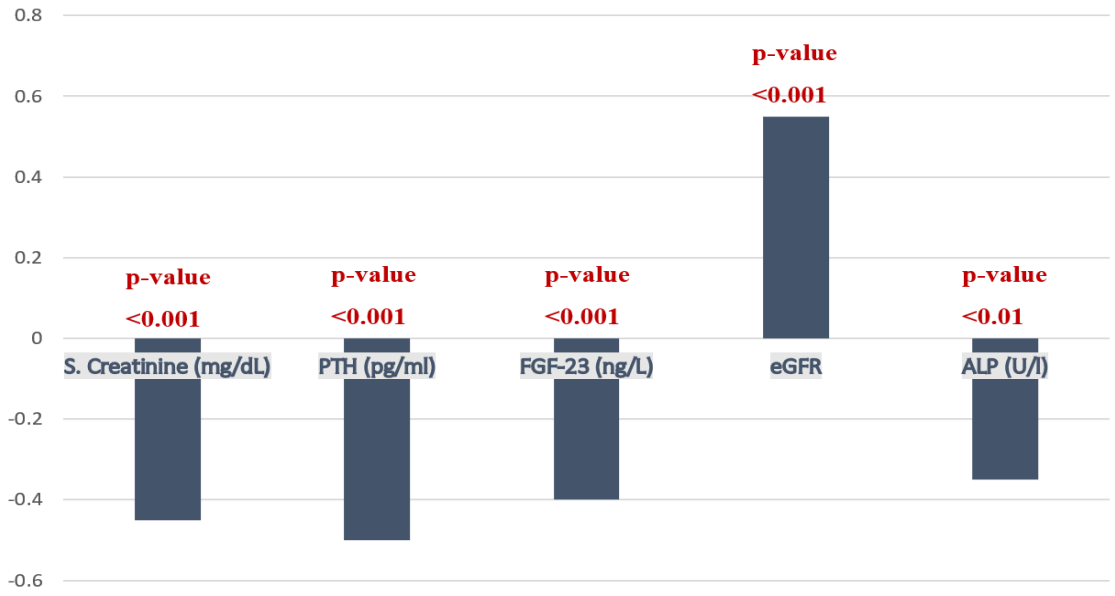

Figure 1: Correlation between levels of 25(OH)D with Biochemical Markers Among Patients' group

4. DISCUSSION

The findings of our study provide significant data on the prevalence of vitamin D deficiency and its correlation with biochemical markers in patients with chronic kidney disease in Erbil City.

The findings demonstrate a trend of deteriorating renal function, increasing biochemical derangement, and decreasing vitamin D levels as CKD progresses from stage 1 to 4. Although the mean serum 25(OH)D levels were not significantly different among CKD patients and healthy controls, vitamin D levels were significantly correlated with key markers of renal and bone metabolism in the CKD group.

The inverse relationship between vitamin D and serum creatinine ($r = -0.45$) supports the widely recognized concept that renal dysfunction leads to impaired activation of vitamin D due to reduced 1-alpha hydroxylase activity in the kidneys, limiting the conversion of 25(OH)D to its active form, calcitriol (10,11). This deficiency contributes to further progression of CKD, as vitamin D is not only essential for mineral metabolism but also for modulating inflammation and immune response (12,13).

The significant negative correlation between vitamin D and PTH ($r = -0.50$) reflects the pathophysiology of secondary hyperparathyroidism in CKD patients. Low vitamin D levels fail to suppress PTH production, leading to elevated PTH, which disrupts calcium and phosphate balance, aggravating bone demineralization and vascular calcification (14,15). Similarly, increased FGF-23 levels ($r = -0.40$ with vitamin D) have been recognized as an early marker of disturbed phosphate metabolism and a contributor to left ventricular hypertrophy and cardiovascular events in CKD patients (16,17).

Although vitamin D levels were not significantly different between the CKD and control groups, stratified data by eGFR revealed a gradual increase in 25(OH)D levels with higher eGFR categories. This trend underscores the importance of early-stage intervention before renal function declines substantially (18). The progressive decline in ALP levels with increasing eGFR also suggests improvement in bone turnover status, which is tightly regulated by vitamin D and PTH (19).

Notably, moderate positive correlation between vitamin D and eGFR ($r = 0.55$) aligns with existing evidence showing that higher vitamin D status is associated with preserved kidney function and reduced risk of ESRD (20,21). While some studies argue whether vitamin D deficiency is a cause or consequence of CKD progression, the mutual reinforcement of these findings supports a bidirectional influence (22).

These biochemical trends are clinically significant. They support the rationale for routine vitamin D monitoring and supplementation in CKD patients—not merely to prevent bone complications but also to modulate cardiovascular and renal outcomes. Nonetheless, the cross-sectional nature of this study limits causal interpretations. Additionally, while this research provides valuable data for the Kurdistan region, further multicenter longitudinal studies are needed to generalize findings across larger populations.

5. CONCLUSION

The findings of our study emphasize the significant associations between serum vitamin D levels and selected biochemical markers in CKD patients in Erbil City. Decreased vitamin D levels were moderately associated with declining renal function (lower eGFR and higher creatinine), elevated PTH, FGF-23, and ALP levels, reflecting bone and mineral disturbances typical in CKD.

Despite no significant difference in vitamin D levels between CKD patients and controls, stratification by eGFR unveiled a compelling relationship—suggesting that earlier stages of CKD may benefit from vitamin D optimization. These relationships support the integration of vitamin D screening and supplementation as a preventative strategy in CKD management.

Given the metabolic complexity of CKD, a multifactorial approach, including monitoring of PTH, FGF-23, ALP, and vitamin D, may help delay disease progression and improve patient quality of life. Future longitudinal studies are essential to better understand the causal mechanisms and evaluate the long-term benefits of vitamin D-based interventions in this population.

6. FUTURE PERSPECTIVES

“Future studies should focus on longitudinal evaluation of vitamin D supplementation in early CKD stages, ideally through randomized controlled trials with larger sample sizes and diverse populations.”

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