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Original Research Article

Effect of Vitamin D Supplementation on Albuminuria in Chronic Kidney Disease: A Randomized Controlled Trial

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Abstract: ***Background*:** Chronic kidney disease (CKD) is a major public health concern worldwide, with a prevalence of 22.5% in Bangladesh. Vitamin D deficiency is common among CKD patients and is associated with increased albuminuria and renal function decline. Vitamin D metabolites may exert reno-protective and anti-proteinuric effects. This study aimed to evaluate the effect of vitamin D supplementation on albuminuria in CKD patients through a randomized clinical trial. ***Materials and Methods:*** This single-blind, randomized controlled trial was conducted at the National Institute of Kidney Diseases and Urology, Dhaka, from January 2021 to February 2022. Adult CKD patients (stages 3–5, non-dialysis) with vitamin D deficiency (<30 ng/ml) were randomized to receive weekly oral cholecalciferol 50,000 IU (n=56) or placebo (n=59) for 12 weeks. Primary outcomes were changes in serum 25-hydroxyvitamin D [25(OH)D] and urinary albumin-to-creatinine ratio (uACR). ***Results:*** Baseline 25(OH)D levels were lower in the intervention group than controls (14.1 vs. 19.6 ng/ml, p<0.05). After 12 weeks, 25(OH)D increased by 12 ng/ml (85%) in the intervention group versus 0.7 ng/ml (3%) in controls (p<0.05). uACR decreased by 882 mg/g (41%) in the intervention group versus 445 mg/g (22%) in controls (p<0.05). No significant changes were observed in serum calcium, phosphate, or eGFR between groups. ***Conclusion:*** Weekly cholecalciferol supplementation significantly improved vitamin D status and reduced albuminuria in CKD patients over 12 weeks.

Keywords: Vitamin D, Albuminuria, Chronic Kidney Disease, Cholecalciferol.

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Introduction

Chronic kidney disease (CKD) is a major global public health concern, affecting 9.1–15.1% of the world's population.[1, 2] In Bangladesh, the prevalence is estimated at 22.5%, considerably higher than the global average.[3, 4] Proteinuria and albuminuria are key prognostic markers of CKD progression and are routinely targeted in disease management.[5–7] Current therapeutic strategies, including inhibition of the renin–angiotensin–aldosterone system (RAAS) with angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARBs) and strict blood pressure control, are standard of care for CKD patient.[8, 9] Vitamin D deficiency is common in CKD, where vitamin D metabolites help inhibit the renin–angiotensin system, prevent glomerulosclerosis, and reduce albuminuria, thus slowing disease progression.[7, 10–12] Reduced synthesis of active vitamin D occurs with declining renal function, resulting in deficiency rates exceeding 50% in CKD patients

compared with those with normal kidney function.[13–15] Evidence suggests that vitamin D metabolites exert reno-protective and anti-proteinuric effects through mechanisms such as RAAS inhibition and prevention of glomerulosclerosis. Randomized clinical trials have reported variable outcomes regarding vitamin D supplementation in reducing proteinuria.[16, 17] While some studies, such as Molina *et al*., demonstrated significant reductions in albuminuria with cholecalciferol, findings remain inconsistent, and most evidence is derived from populations outside South Asia.[16, 17] Given the higher CKD burden and potential differences in baseline vitamin D status in Bangladeshi patients, there is a need for locally generated evidence to guide practice. This study aimed to evaluate the effect of vitamin D supplementation on albuminuria among Bangladeshi patients with CKD through a randomized clinical trial.

Materials and Methods

Study Design

This single-blind, randomized controlled clinical trial was conducted in the Department of Nephrology, National Institute of Kidney Diseases and Urology (NIKDU), Dhaka, Bangladesh, from January 2021 to February 2022.

Study Participants and Sample Size

Eligible participants were adults (≥18 years) with CKD stages 3–5 (non-dialysis) and serum vitamin D levels <30 ng/ml. Additional inclusion criteria were urinary albumin-to-creatinine ratio (uACR) 500–3000 mg/g, blood pressure <140/80 mmHg for ≥3 months, and controlled diabetes mellitus (HbA1C 6.5%–7.5%). Patients already on vitamin D supplementation, with serum calcium >10.0 mg/dl or phosphate >5.0 mg/dl, were excluded. A total of 120 patients meeting the selection criteria were enrolled. Sample size was determined based on feasibility rather than a priori power calculation.

Randomization and Blinding

Participants were randomized into intervention and control groups in a 1:1 ratio using a computer-generated random sequence. Allocation concealment was maintained with sequentially numbered, opaque, sealed envelopes prepared by an independent investigator. The trial was single-blinded: participants were unaware of their group assignment, while investigators and outcome assessors were aware due to logistic constraints.

Intervention

The intervention group received oral cholecalciferol 50,000 IU weekly for 12 weeks, while the control group received identical placebo capsules. Both groups continued standard CKD management, including RAAS blockade as clinically indicated.

Data Collection and Measurements

Data were collected through face-to-face interviews and review of medical records using a semi-structured questionnaire. Baseline assessments included demographic data, clinical history, and laboratory tests (serum creatinine, calcium, phosphate, 25-hydroxyvitamin D [25(OH)D], fasting blood sugar, HbA1c, and uACR). The same parameters were reassessed at 12 weeks.

Outcome Measures

The primary outcomes were changes in serum 25(OH)D and uACR after 12 weeks. Secondary outcomes included changes in serum calcium, phosphate, creatinine, and estimated glomerular filtration rate (eGFR).

Ethical Considerations

Ethical approval was obtained from the Institutional Review Board of NIKDU (Approval No.: [insert number]). Written informed consent was obtained from all participants before enrollment. The study was conducted in accordance with the Declaration of Helsinki.

Statistical Analysis

Data were analyzed using SPSS version 25 (Chicago, IL, USA). Quantitative variables were summarized as mean ± standard deviation (SD) and qualitative variables as frequencies and percentages. Between-group comparisons were performed using Student's t-test or chi-square test, as appropriate. Paired t-tests assessed within-group changes. One-way ANOVA and Pearson's correlation were used for subgroup analyses. A p-value <0.05 was considered statistically significant.

Results

A total of 120 patients were enrolled; five participants (four from the intervention group and one from the

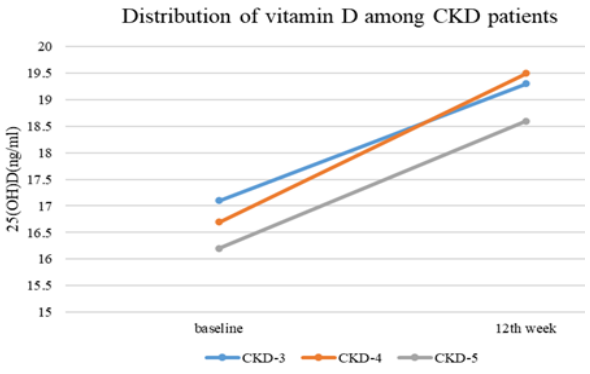
control group) withdrew voluntarily before follow-up, leaving 115 patients for analysis (56 in the intervention group and 59 in the control group).

Baseline Characteristics of Study Participants

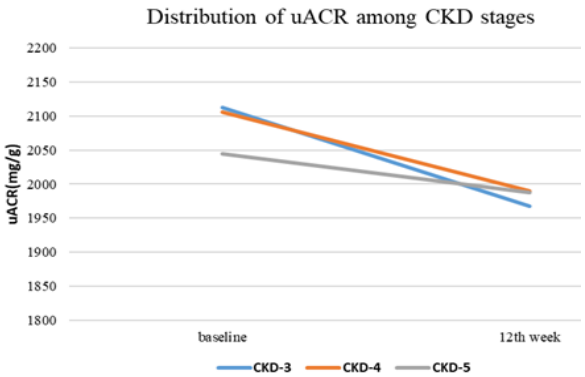
Of the 120 patients initially enrolled, 115 completed follow-up and were included in the analysis (56 intervention, 59 control). Baseline characteristics were comparable between groups (Table 1). The mean age was 43.7 ± 11.9 years in the intervention group and 42.7 ± 11.4 years in the control group (p=0.71). Females comprised slightly more than half of both cohorts (51.8% vs. 55.9%, p=0.66). Mean body mass index was similar (23.4 ± 2.3 vs. 23.8 ± 2.0 kg/m²). In the intervention group, 44.6% had stage 4 CKD and 41.2% had stage 3 CKD, while in the control group, 45.8% and 42.4% had stages 4 and 3 CKD, respectively, with stage 5 present in 14.2% and 11.8% of the intervention and control groups. Diabetes mellitus was more frequent in controls (42.4%), whereas glomerulonephritis predominated in the intervention group (42.9%). Use of RAAS inhibitors did not differ significantly between groups (p=0.83) (Table 1).

Table 1: Baseline Characteristics of The Study Population (n=115)

Characteristics	Case group (n=56)	Control group (n=59)	p-value
Age (years)	43.7 ± 11.9	42.7 ± 11.4	0.71[a]
Gender			
Male	27 (48.2%)	26 (44.1%)	0.66[b]
Female	29 (51.8%)	33 (55.9%)	
BMI (kg/m²)	23.4 ± 2.3	23.8 ± 2.0	0.24[a]
Primary disease			0.31[b]
Hypertension	10 (17.9%)	16 (27.1%)	
Diabetes mellitus	22 (39.3%)	25 (42.4%)	
Glomerulonephritis	24 (42.9%)	18 (30.5%)	
CKD Stage			0.93[b]
Stage 3	23 (41.2%)	25 (42.4%)	
Stage 4	25 (44.6%)	27 (45.8%)	
Stage 5	8 (14.2%)	7 (11.8%)	
Use of RAAS agents			0.83[b]
With ACEi/ARB	38 (69.6%)	40 (67.8%)	
Without ACEi/ARB	17 (30.3%)	19 (32.2%)	



(a): Changes in 25(OH)D among CKD stages from baseline to 12 weeks.
*p = 0.287 at baseline & 0.897 at 12 weeks.



(b): Changes in uACR among CKD stages from baseline to 12 weeks.
*p = 0.050 at baseline & 0.284 at 12 weeks.

Figure 1: Change in 25(OH)D and uACR Across CKD Stages from Baseline to Week 12.

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Figure 1(a) demonstrates that 25(OH)D levels did not differ significantly across CKD stages at either baseline (16.2–17.1 ng/mL) (p = 0.287) or week 12 (19.5–23.0 ng/mL) (p = 0.897), although a consistent upward trend was observed in all stages. Figure 1(b) shows a gradual decline in mean uACR from baseline to week 12 across CKD stages 3, 4, and 5; however, this reduction did not reach statistical significance (p = 0.050 and p = 0.284, respectively).

Comparison of Serum 25(OH)D, uACR by Primary Disease, and Other Laboratory Parameters between Groups at Baseline and Week 12

Across primary disease groups (diabetes, hypertension, and glomerulonephritis), mean 25(OH)D and uACR values showed no statistically significant differences at either baseline or week 12. All groups exhibited improved 25(OH)D levels and reduced uACR following intervention, but the magnitude of change did not vary significantly by underlying disease (Table 2). In contrast, when analyzed by treatment allocation (Table III), baseline 25(OH)D levels were significantly lower in the cholecalciferol group compared with controls ($p<0.05$). After 12 weeks, 25(OH)D increased to 26.1 ± 4.3 ng/mL in the intervention group versus 20.3 ± 2.7 ng/mL in controls ($p<0.05$). uACR declined more markedly in the intervention group (1269.7 ± 391.3 mg/g) compared with controls, while serum calcium, phosphate, creatinine, and eGFR remained comparable between groups.

Table 2: Serum 25(OH)D and uACR by Primary Disease at Baseline and Week 12 (n=115)

Parameter	Visit	Diabetes Mellitus	Hypertension	Glomerulonephritis	*p*-value[c]
25(OH)D (ng/mL)	Baseline	16.8 ± 4.61	16.2 ± 3.90	16.6 ± 4.34	0.444
	Week 12	24.3 ± 4.13	22.4 ± 4.18	24.5 ± 4.01	0.904
uACR (mg/g)	Baseline	2239 ± 494.3	1998 ± 764.1	1968 ± 729.2	0.397
	Week 12	1200.7 ± 499	1385 ± 615	1261 ± 564	0.119

Data are presented as mean ± SD. [a]Student's t-test; [c]One-way ANOVA. A p-value <0.05 was considered statistically significant.

Table 3: Comparison of Laboratory Parameters between Cholecalciferol and Control Groups at Baseline and Week 12 (n = 115)

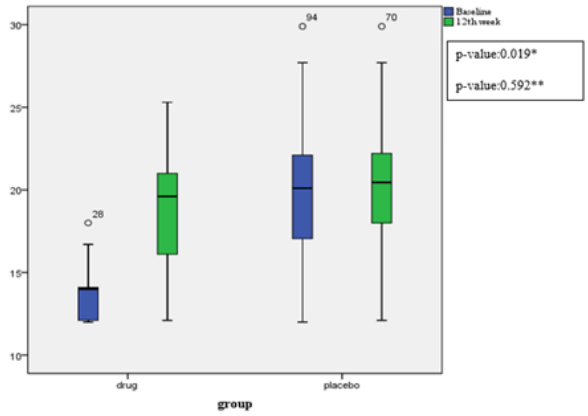
Parameter	Timepoint	Cholecalciferol Group (n=56)	Control Group (n=59)	*p*-value[a]
25(OH)D (ng/mL)	Baseline	14.1 ± 1.9	19.6 ± 4.1	<0.05
	Week 12	26.1 ± 4.3	20.3 ± 2.7	<0.05
Serum Calcium (mg/dL)	Baseline	9.2 ± 0.9	9.2 ± 0.8	0.131
	Week 12	9.3 ± 0.9	9.2 ± 0.8	0.635
Serum Phosphate (mg/dL)	Baseline	4.6 ± 0.8	4.3 ± 0.8	0.945
	Week 12	4.3 ± 0.5	4.1 ± 0.6	0.060
Serum Creatinine (mg/dL)	Baseline	4.0 ± 1.9	4.1 ± 2.1	0.188
	Week 12	3.6 ± 1.3	3.7 ± 1.9	0.479
uACR (mg/g)	Baseline	2152.1 ± 753.5	2010.8 ± 588.3	0.362
	Week 12	1269.7 ± 391.3	1565.8 ± 655.8	<0.05
eGFR (mL/min/1.73 m²)	Baseline	40.9 ± 6.9	40.6 ± 7.6	0.210
	Week 12	41.5 ± 7.7	40.5 ± 8.4	0.712
HbA1c (%)	Baseline	6.6 ± 1.1	6.8 ± 1.2	0.566
	Week 12	6.7 ± 1.1	6.8 ± 1.2	0.566

Parameter	Timepoint	Cholecalciferol Group (n=56)	Control Group (n=59)	*p*-value[a]
FBS (mmol/L)	Baseline	5.5 ± 0.8	5.7 ± 0.9	0.060
	Week 12	5.7 ± 0.8	5.8 ± 0.9	0.060

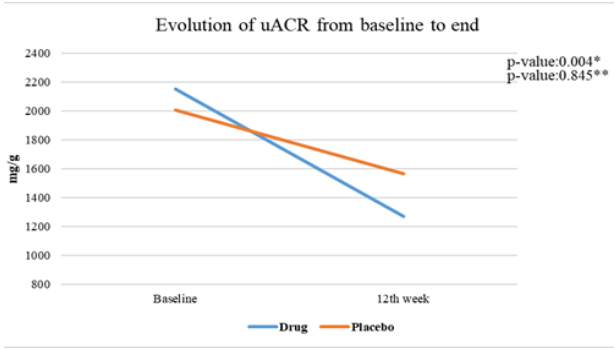
Data are presented as mean ± SD. [a]Student's t-test was used; p<0.05 was considered statistically significant.

Changes in Serum 25(OH)D and uACR Levels in Case and Control Groups Over 12 Weeks

The box plot illustrates a significant increase in mean 25(OH)D levels in the case group, while a decrease was observed in the control group (Figure 2a). In the case group, the mean 25(OH)D level increased significantly (p = 0.019) by 12 ng/ml. In contrast, the control group experienced a mean increase of only 0.7 ng/ml, which was not statistically significant (p = 0.592). A significant reduction in mean uACR level was observed exclusively in the case group (Figure 2b). Cholecalciferol administration significantly (p = 0.004) reduced the mean uACR level by 882.3 mg/g in the case group, whereas the reduction in the control group was 445 mg/g, which was not significant (p = 0.845).



(a): Change in Serum 25(OH)D level in Case and Control Groups Over 12 Weeks

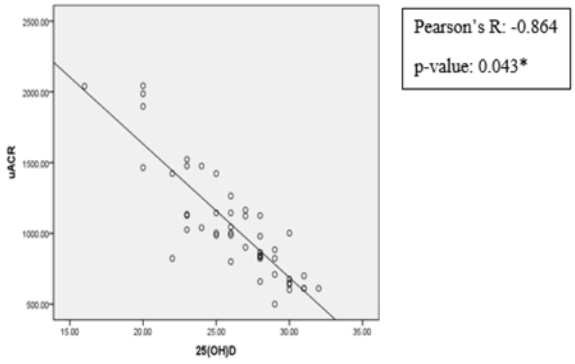


(b): Change in uACR in Case and Control Groups Over 12 Weeks.

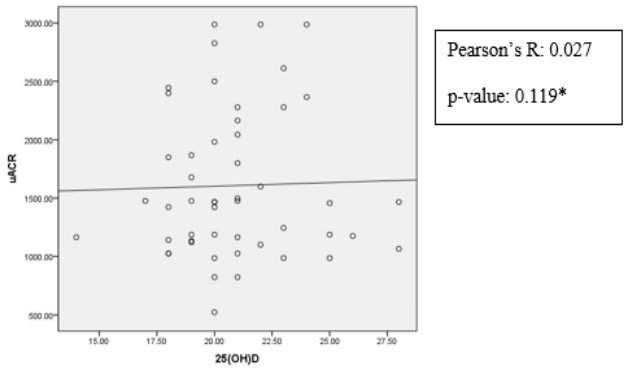
Figure 2: Changes in Serum 25(OH)D and uACR Levels in Case and Control Groups Over 12 Weeks. *p-Value Obtained by paired t-test

Correlation Between Serum 25(OH)D and uACR Levels from Baseline to Week 12 in Case and Control Groups

Correlation analyses of serum 25(OH)D with uACR from baseline to the 12th week follow-up, as shown in Figures 3a and 3b for the Case and Control groups, indicate that in the case group, 25(OH)D levels were significantly and inversely associated with uACR; that is, as the 25(OH)D level increases, the urinary albumin-to-creatinine ratio decreases (Pearson's R = -0.864, p = 0.043). A significant correlation was observed in the case group. In contrast, a positive but not statistically significant correlation was found in the control group (Pearson's R = 0.027, p = 0.119).



(a) Case Group

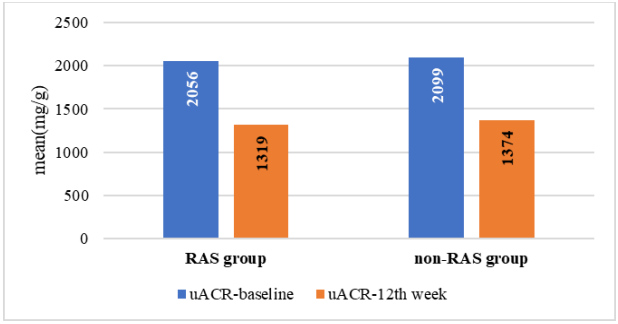


(b) Control Group

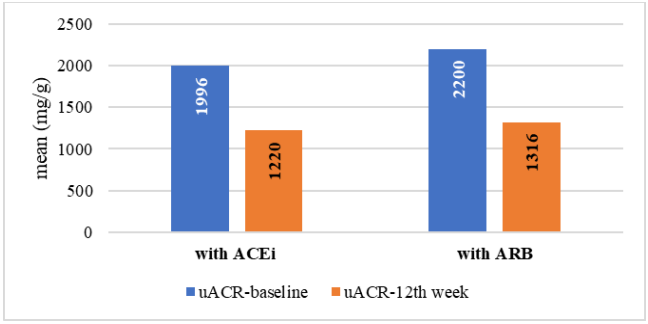
Figure 3: Correlation analysis of changes from baseline to 12 weeks in 25(OH)D and uACR in case and control groups.

Effect of RAS Agents on Urinary Albumin-to-Creatinine Ratio in CKD Patients Over 12 Weeks

The use of RAS agents did not significantly affect the spot urine albumin-creatinine ratio among CKD patients from baseline to the 12th week, although it was reported that the uACR was lower at the 12th week in both the RAS and non-RAS groups (Figure 4a). The use of ACE inhibitors (ACEi) reduced the uACR from 2056 mg/g to 1319 mg/g, while angiotensin receptor blockers (ARB) reduced the uACR from 2099 mg/g to 1374 mg/g. However, ACEi and ARB did not significantly affect the spot urine albumin-creatinine ratio among CKD patients from baseline to the 12th week (Figure 4b).



(a) Distribution of uACR with RAS Agents
*p-value at baseline:0.792
*p-value at 12th week:0.672



(b) Distribution of uACR with ACEi/ARB Agents
*p-value at baseline:0.883
*p-value at 12th week:0.246

Figure 4: Distribution of uACR with Antihypertensive Drugs at Baseline and Week 12.

Discussions

The present study was conducted with the view to evaluating the renoprotective effects, Vitamin D status, and functional alteration of the kidney with weekly oral cholecalciferol supplementation in CKD patients of Bangladesh. In the present study, the mean age of the patients was 43.7 ± 11.9 and 42.7 ± 11.4 years for case and control, respectively. More than half of the CKD patients were females in both groups, 51.8% and 55.9% respectively. There was no statistically significant difference in age and gender between case and control. Similar findings were also observed in Molina *et al.*, (2013, a prospective, controlled, intervention study among CKD patients with albuminuria, where gender was not significantly associated among the CKD patients ($p = 0.470$), but age of the patients was ($p < 0.05$).[18]

In the present study, it was observed that CKD patients with stages 3, 4, and 5 had 25(OH)D levels below the normal range (20 – 40 ng/ml), with stage 5 having the lowest level of 25(OH)D at baseline.[19] CKD patients are more prone to having vitamin D deficiency, which is closely associated with albuminuria and lower glomerular filtration rate (GFR).[20] Levin *et al.*, (2007 reported that in CKD stage 3, 20% of CKD patients were found to have low 25(OH)D concentrations (<20 ng/mL), whereas in CKD stages 4 and 5, >30% of patients were deficient.[21] These findings support the present study showing patients with CKD stage 3, 4, and 5 to have lower levels of 25(OH)D. At the end of the follow-up (12th week), 25(OH)D levels significantly ($p < 0.05$) increased (26.1 ± 4.26 ng/ml) in patients taking cholecalciferol (vitamin D supplement) compared to the placebo group (20.3 ± 2.7 ng/ml). A randomized double-blinded placebo-controlled study among 175 respondents reported that patient receiving higher dose of vitamin–D supplements had improved 25(OH)D levels compared to placebo group, which supports present study findings.[22] Another 6-month prospective, controlled, intervention study among non-dialysis CKD patients with albuminuria showed similar findings where Cholecalciferol administration directed to an increase in mean 25(OH)D levels by 53.0 ± 41.6% ($p < 0.05$). At the end of the follow-up (12th week), uACR significantly ($p < 0.05$) lowered in

patients taking cholecalciferol (1269.7 ± 391.3 mg/g) compared to the control group.

Cholecalciferol administration reduced the uACR by 882.3 mg/g, whereas in the placebo group, the reduction was 445mg/g, suggesting that the use of vitamin D supplements like cholecalciferol may have a positive impact on reducing proteinuria in CKD patients. Molina *et al*., (2013 showed urinary albumin-to-creatinine ratio (uACR) to decrease from 284 mg/g to 167 mg/g at 6 months follow-up ($p < 0.05$) in the cholecalciferol group, and no change in the control group, which is consistent with the findings of the present study.[18, 23] The present study outcome has shown that the patient taking the cholecalciferol drug had significantly improved 25(OH)D levels compared to the placebo group at the end of the study; moreover, this change in 25(OH)D level was inversely associated with the uACR level among the patient's taking cholecalciferol. Kim *et al*., (2011 observed a significant reduction in the uACR among CKD patients after treatment with oral cholecalciferol, which supports the present study findings.[24] Alborzi *et al*., and association data from the Third National Health and Nutrition Examination Survey (NHANES III) demonstrated an inverse relationship between the level of vitamin D and the degree of albuminuria.[25, 26] Boer *et al*., 2007 also showed similar findings about the anti-proteinuric effect of Vitamin D receptor (VDR) activation.[20] The present study showed that ACEi reduced the uACR from 2056mg/g to 1319 mg/g, and ARB reduced the uACR from 2099mg/g to 1374 mg/g. However, the use of ACEi and ARB agents did not significantly affect the spot urine albumin creatinine ratio among the CKD patients from baseline to the 12th week. Hence, making it evident that it was cholecalciferol which reduced the uACR rather than the ACEi and ARBs. Molina *et al*., (2013 showed similar findings where patients taking cholecalciferol had a significant reduction in uACR, with no significant reduction in uACR for both ACEi and ARBs.[18]

Conclusion

The findings of the present study suggest that vitamin D supplementation may effectively reduce albuminuria in patients with chronic kidney disease (CKD), thereby improving prognosis. It should be considered as the first therapeutic approach for CKD patients with low vitamin D status. However, a large-scale, multi-center prospective study is needed to assess the long-term benefits and effectiveness of vitamin D supplementation among CKD patients.

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