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Serum vitamin D deficiency and its association with inflammatory markers in patients with chronic kidney disease: A hospital-based cross-sectional study.

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Abstract

Background:
Vitamin D deficiency is common in chronic kidney disease (CKD) and may contribute to systemic inflammation; Indian hospital-based data remain limited.

Objectives:
To estimate the prevalence of serum Vitamin D deficiency in CKD and assess its association with C-reactive protein (CRP), interleukin-6 (IL-6), and erythrocyte sedimentation rate (ESR).

Methods:
This hospital-based cross-sectional study enrolled 100 adults (≥18 years) with CKD. Serum 25(OH)D was classified as deficient (<20 ng/mL), insufficient (20–30 ng/mL), or sufficient (>30 ng/mL). CRP, IL-6, and ESR were measured. Group comparisons used one-way ANOVA, and associations were tested using Pearson's correlation; $p < 0.05$ was considered significant.

Results:
Mean age was 52.6 ± 12.4 years; 62% were men, and 68% had CKD stages 4–5. Vitamin D deficiency, insufficiency, and sufficiency were seen in 64%, 22%, and 14%, respectively. CRP and IL-6 were higher in the deficient group (9.4 ± 2.6 mg/L; 12.1 ± 4.2 pg/mL) than the insufficient (6.8 ± 2.1 mg/L; 8.6 ± 3.1 pg/mL) and sufficient groups (5.2 ± 1.8 mg/L; 6.9 ± 2.4 pg/mL) ($p < 0.001$ and $p < 0.01$). ESR was higher in deficient patients but not significantly so ($p = 0.12$). Vitamin D correlated negatively with CRP ($r = –0.48$, $p < 0.001$) and IL-6 ($r = –0.42$, $p = 0.002$); correlation with ESR was non-significant ($p = 0.12$).

Conclusion:
Vitamin D deficiency was highly prevalent and was associated with elevated CRP and IL-6, suggesting a link between hypovitaminosis D and systemic inflammation in CKD.

Recommendations:
Routine Vitamin D screening and correction may be considered in CKD care; longitudinal and interventional studies are needed to clarify causality and optimal supplementation strategies.

Keywords: Vitamin D deficiency, chronic kidney disease, C-reactive protein, interleukin-6, inflammation
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Introduction
Chronic kidney disease (CKD) is a major global health burden, affecting 10–15% of the adult population, and is associated with progressive renal dysfunction, heightened morbidity, and premature mortality [1]. Beyond the gradual decline in kidney function, CKD is characterized by a persistent pro-inflammatory state that contributes to accelerated atherosclerosis, cardiovascular disease, malnutrition, and reduced quality of life [2,3]. Identification of modifiable factors influencing this inflammatory milieu is therefore critical.

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Vitamin D, long recognized for its role in calcium–phosphate metabolism and skeletal health, has emerged as a pleiotropic hormone with wide-ranging effects on the immune and cardiovascular systems [4]. In CKD, Vitamin D deficiency is highly prevalent due to reduced renal 1-α hydroxylase activity, dietary restrictions, inadequate sunlight exposure, and urinary loss of Vitamin D–D-binding protein [1,4]. Hypovitaminosis D has been linked to elevated inflammatory biomarkers, including C-reactive protein (CRP) and interleukin-6 (IL-6), both of which are key mediators of endothelial dysfunction and cardiovascular risk [2,5].

Several observational studies have consistently shown a high prevalence of Vitamin D deficiency among CKD patients across different populations [3,5,6]. However, hospital-based data from India remain limited, particularly regarding the interplay between Vitamin D status and systemic inflammatory markers. Clarifying this relationship may provide valuable insights into the pathophysiological mechanisms driving inflammation in CKD and help develop preventive and therapeutic strategies targeting micronutrient deficiency.

The present study was undertaken to evaluate the prevalence of Vitamin D deficiency among patients with CKD and to assess its association with inflammatory markers, including CRP, IL-6, and erythrocyte sedimentation rate (ESR), in a hospital-based setting.

Materials and Methods

Study Design and Setting

This was a hospital-based, cross-sectional observational study conducted in the Department of General Medicine at Government Medical College and General Hospital, Suryapet, Telangana. The study was carried out over a period of six months, from January 2025 to June 2025.

Sample Size

The sample size of 100 participants was determined based on feasibility and evidence from previous hospital-based studies reporting a high prevalence of Vitamin D deficiency among patients with chronic kidney disease. Considering the cross-sectional design, limited study duration, and average patient inflow at the study centre, this sample size was considered adequate to assess associations between serum Vitamin D levels and inflammatory markers with acceptable statistical precision.

Study Population

A total of 100 patients with a confirmed diagnosis of chronic kidney disease (CKD) attending the nephrology and general medicine outpatient and inpatient services were enrolled in the study. Eligible participants were adults aged ≥18 years with CKD stages 3–5, diagnosed based on estimated glomerular filtration rate (eGFR) calculated using the CKD-EPI formula.

Inclusion Criteria

Adults (≥18 years) with clinically and biochemically confirmed CKD stages 3–5.

Patients are willing to provide informed consent.

Exclusion Criteria

Patients with acute kidney injury, active infections, chronic liver disease, or autoimmune disorders.

Those on Vitamin D supplementation, calcium analogues, or immunosuppressive drugs within the past 3 months.

Pregnant and lactating women.

Data Collection

Demographic details (age, sex), clinical history, and CKD staging were recorded. Venous blood samples were collected for estimation of serum 25-hydroxy Vitamin D [25(OH)D], C-reactive protein (CRP), interleukin-6 (IL-6), and erythrocyte sedimentation rate (ESR).

Vitamin D measurement: Serum 25(OH)D levels were estimated using chemiluminescent immunoassay.

Classification of Vitamin D status: Deficient (<20 ng/mL), Insufficient (20–30 ng/mL), and Sufficient (>30 ng/mL).

Inflammatory markers: CRP was measured by high-sensitivity immunoturbidimetry, IL-6 by enzyme-linked immunosorbent assay (ELISA), and ESR by the Westergren method.

Bias and Confounding

Efforts were made to minimise potential sources of bias in this study. Selection bias was reduced by consecutively enrolling all eligible CKD patients attending the hospital during the study period. Measurement bias was minimised by using standardised and validated laboratory assays for serum 25-hydroxyvitamin D, C-reactive protein, interleukin-6, and erythrocyte sedimentation rate. Data were collected using a structured proforma to limit information bias. To reduce confounding, patients with acute inflammatory conditions, autoimmune diseases, malignancy, or recent Vitamin D supplementation were excluded.

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Ethical Considerations

Ethical clearance was obtained from the Institutional Ethics Committee of Government Medical College, Suryapet. Written informed consent was obtained from all participants before enrollment.

Statistical Analysis

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparison of inflammatory marker levels across Vitamin D categories was performed using one-way ANOVA with post-hoc Tukey test. Pearson's correlation coefficient was used to assess the relationship between serum Vitamin D levels and inflammatory markers. A p-value <0.05 was considered statistically significant.

Results

Participant Flow

During the study period, 128 patients with a provisional diagnosis of chronic kidney disease were screened for eligibility. Of these, 18 patients did not meet the inclusion criteria (CKD stage <3 or age <18 years), and 10 were excluded due to the presence of acute infection, autoimmune disease, or recent Vitamin D supplementation. The remaining 100 patients fulfilled the eligibility criteria and were enrolled in the study. As this was a cross-sectional study with a single-time-point assessment, all enrolled participants completed the evaluation and were included in the final analysis(Figure 1).

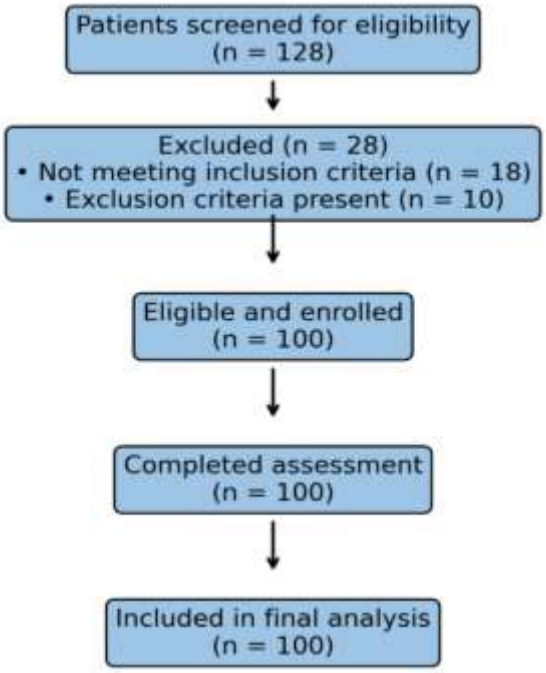

Figure 1: Participant Flow Diagram

A total of 100 patients with chronic kidney disease were included in the study. The mean age of the cohort was 52.6 ± 12.4 years, with a predominance of males (62%) compared to females (38%). Most patients (68%) were in advanced stages of CKD (stage 4–5), while 32% were in stage 3 (Table 1).

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Table 1. Baseline Characteristics of Study Population (N = 100)

Characteristic	Values
Mean age (years)	52.6 ± 12.4
Male (%)	62
Female (%)	38
CKD Stage 3 (%)	32
CKD Stage 4–5 (%)	68

Vitamin D assessment revealed that deficiency (<20 ng/mL) was highly prevalent, affecting 64% of patients. Insufficiency (20–30 ng/mL) was observed in 22%, and only 14% had sufficient levels (>30 ng/mL). The mean Vitamin D concentration among the deficient group was 15.4 ± 3.8 ng/mL, compared to 24.1 ± 2.7 ng/mL and 34.2 ± 3.1 ng/mL in the insufficient and sufficient groups, respectively (Table 2).

Table 2. Distribution of Vitamin D Status in CKD Patients

Vitamin D Status	n (%)	Mean Vitamin D (ng/mL)
Deficient (<20 ng/mL)	64 (64%)	15.4 ± 3.8
Insufficient (20–30 ng/mL)	22 (22%)	24.1 ± 2.7
Sufficient (>30 ng/mL)	14 (14%)	34.2 ± 3.1

Analysis of inflammatory markers demonstrated that patients with Vitamin D deficiency had significantly elevated CRP (9.4 ± 2.6 mg/L) and IL-6 (12.1 ± 4.2 pg/mL) compared to those with insufficient (6.8 ± 2.1 mg/L; 8.6 ± 3.1 pg/mL) and sufficient Vitamin D levels (5.2 ± 1.8 mg/L; 6.9 ± 2.4 pg/mL). The differences were statistically significant for both CRP ($p < 0.001$) and IL-6 ($p < 0.01$). ESR values were higher in the deficient group but did not reach statistical significance ($p = 0.12$) (Table 3).

Table 3. Inflammatory Markers by Vitamin D Status

Parameter	Deficient (n=64)	Insufficient (n=22)	Sufficient (n=14)	p-value
CRP (mg/L)	9.4 ± 2.6	6.8 ± 2.1	5.2 ± 1.8	<0.001
IL-6 (pg/mL)	12.1 ± 4.2	8.6 ± 3.1	6.9 ± 2.4	<0.01
ESR (mm/hr)	39.5 ± 10.3	35.2 ± 9.8	33.8 ± 8.9	0.12

Correlation analysis revealed a strong negative correlation between serum Vitamin D levels and CRP ($r = –0.48$, $p < 0.001$) as well as IL-6 ($r = –0.42$, $p = 0.002$). The association between Vitamin D and ESR was weaker and statistically non-significant ($r = –0.16$, $p = 0.12$) (Table 4).

Table 4. Correlation of Vitamin D Levels with Inflammatory Markers

Parameter	Correlation Coefficient (r)	p-value
CRP	–0.48	<0.001
IL-6	–0.42	0.002
ESR	–0.16	0.12

Discussion

The present study demonstrated a high prevalence of Vitamin D deficiency (64%) among patients with chronic kidney disease, while only a small proportion (14%) had sufficient serum levels. This pattern is consistent with previously published evidence indicating that Vitamin D

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deficiency is highly prevalent in CKD populations. Prior randomized and observational studies have shown that activation of Vitamin D receptors may reduce pro-inflammatory cytokine expression and improve immune regulation in CKD, supporting the biological plausibility of these findings [7]. In addition, hypovitaminosis D has been reported to be almost universal in CKD due to impaired renal hydroxylation, altered metabolism, reduced sunlight exposure, and disease-related lifestyle factors [8].

A key finding of the study was the significant association between Vitamin D deficiency and elevated inflammatory markers, particularly C-reactive protein and interleukin-6. This observation aligns with recent clinical and review evidence suggesting that Vitamin D plays a modulatory role in systemic inflammation among CKD patients, although results from interventional trials remain variable [9]. Lower Vitamin D concentrations have also been associated with advanced CKD stages and increased inflammatory activity, further supporting a link between declining renal function and inflammatory burden [10].

The inverse correlation observed between Vitamin D levels and CRP and IL-6 reinforces the hypothesis that Vitamin D may exert anti-inflammatory effects. Similar associations have been documented across different age groups and clinical settings, indicating that the relationship between Vitamin D insufficiency and inflammation is not limited to CKD alone but extends to other chronic conditions as well [11]. Nevertheless, the literature also reflects ongoing debate, as some interventional studies have failed to demonstrate consistent anti-inflammatory benefits following Vitamin D supplementation, highlighting heterogeneity in study designs, dosing regimens, and patient characteristics [12].

In contrast, erythrocyte sedimentation rate did not differ significantly across Vitamin D categories. This finding may reflect the nonspecific nature of ESR in CKD, where factors such as anemia, altered plasma proteins, and multiple comorbidities limit its reliability as an inflammatory marker. Similar patterns have been reported in other CKD and pediatric populations, where ESR did not consistently correlate with inflammatory severity despite prevalent Vitamin D deficiency [13]. Mechanistic evidence further suggests that Vitamin D influences immune homeostasis through pathways involving dendritic cell regulation and immune precursor modulation, processes that are closely linked to systemic inflammation in CKD [14]. Moreover, low Vitamin D levels in advanced CKD and dialysis populations have been associated not only with inflammation but also with anemia and oxidative stress, underscoring the broader clinical implications of Vitamin D deficiency in this population [15].

Generalizability

The findings of this single-center, hospital-based study conducted at Government Medical College, Suryapet, provide valuable insights into the prevalence of Vitamin D deficiency and its association with inflammatory markers in patients with CKD. However, the results should be interpreted with caution when extrapolated to the general CKD population, as the sample was limited to 100 patients over six months and may not capture seasonal or regional variations in Vitamin D status. Furthermore, lifestyle, nutritional, and genetic factors specific to this cohort may differ from other populations. While the observed associations between Vitamin D deficiency and elevated CRP and IL-6 are consistent with international evidence, multicentric studies with larger and more diverse patient populations are necessary to enhance external validity and confirm the broader applicability of these results.

Conclusion

This hospital-based study highlights the high prevalence of Vitamin D deficiency among patients with chronic kidney disease and its significant association with elevated inflammatory markers, particularly CRP and IL-6. The findings support the growing evidence that hypovitaminosis D contributes to the pro-inflammatory state in CKD, potentially influencing disease progression and cardiovascular risk. Although ESR showed no significant association, the consistent negative correlation between Vitamin D and CRP/IL-6 underscores its role as a modifiable factor. Routine assessment and timely correction of Vitamin D deficiency should be considered in CKD management. Future multicentric longitudinal studies are needed to validate these observations.

Limitations

The study has certain limitations. Being a single-center, cross-sectional design, it cannot establish causality between Vitamin D deficiency and inflammation in CKD. The relatively small sample size and short study duration may limit statistical power and preclude subgroup analysis across CKD stages. Seasonal variation in Vitamin D status was not assessed, and important modulators such as parathyroid hormone and fibroblast growth factor-23 were not measured. Hence, findings may not be fully generalizable to broader CKD populations.

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Recommendations

Routine screening of Vitamin D status should be integrated into the clinical management of chronic kidney disease patients, particularly those in advanced stages, given the strong association with systemic inflammation. Early correction of deficiency through tailored supplementation, dietary fortification, and lifestyle modification may help attenuate inflammatory burden and potentially improve renal and cardiovascular outcomes. Clinicians should individualize Vitamin D therapy based on the severity of deficiency, CKD stage, and comorbid conditions. Future multicentric, longitudinal, and randomized controlled trials in diverse populations are warranted to establish causality, determine optimal dosing regimens, and validate the long-term benefits of Vitamin D supplementation.

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Abbreviations

CKD – Chronic Kidney Disease
CRP – C-Reactive Protein
IL-6 – Interleukin-6
ESR – Erythrocyte Sedimentation Rate
PTH – Parathyroid Hormone
FGF-23 – Fibroblast Growth Factor-23
eGFR – Estimated Glomerular Filtration Rate
ELISA – Enzyme-Linked Immunosorbent Assay
SPSS – Statistical Package for the Social Sciences

Source of funding

The study had no funding.

Conflicts of interest

The Aauthor declares no conflict of interest.

Data Availability

Data available on request

Author's contribution

PHK-Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript. **ST** -Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, **SK**-revision of manuscript.results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript.

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