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VITAMIN D DEFICIENCY IS ASSOCIATED WITH SEVERE ACUTE KIDNEY INJURY: A CROSS-SECTIONAL STUDY FROM PESHAWAR, PAKISTAN

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Abstract

Background: *Acute Kidney Injury (AKI) is a critical medical condition associated with significant morbidity and mortality. Understanding its relationship with Vitamin D may provide insights into potential biomarkers and therapeutic targets.*

Objective: *To evaluate the association between AKI severity and vitamin D levels in patients from Peshawar, Pakistan.*

Methods: *This cross-sectional study was conducted at Lady Reading Hospital, Peshawar. Serum 25-hydroxyvitamin D was measured by electrochemiluminescence immunoassay, and serum creatinine by spectrophotometric techniques. Demographic and clinical data were collected through a structured questionnaire. Statistical analysis included one-way ANOVA, Pearson correlation, and binary logistic regression. A p-value <0.05 was considered statistically significant.*

Results: *The mean age was 45.67 ± 16.45 years with male predominance (61.4%). Mean vitamin D level was 24.78 ± 8.23 ng/mL, and mean creatinine was 3.89 ± 1.45 mg/dL. Vitamin D levels decreased progressively with AKI severity: mild AKI (31.42 ± 7.84 ng/mL), moderate AKI (25.67 ± 6.91 ng/mL), severe AKI (22.34 ± 5.67 ng/mL), and very severe AKI (19.76 ± 8.23 ng/mL) ($F = 12.456$, $p < 0.001$). Pearson correlation showed a significant negative correlation between vitamin D and creatinine ($r = -0.445$, $p < 0.001$). Vitamin D deficiency (<20 ng/mL) was the strongest independent risk factor for severe AKI (creatinine >5 mg/dL) (OR = 7.07, 95% CI: 2.83-17.65, $p < 0.001$).*

Conclusion: *There is a strong inverse association between vitamin D levels and AKI severity. Vitamin D deficiency confers a 7-fold increased risk of severe AKI, highlighting its potential as a prognostic marker and therapeutic target.*

INTRODUCTION

The kidneys are essential organs that maintain fluid and electrolyte balance, eliminate metabolic waste products, regulate blood pressure, and activate vitamin D.[1] Acute Kidney Injury (AKI), defined by KDIGO criteria as an increase in serum creatinine ≥0.3 mg/dL within 48 hours, an increase to ≥1.5 times baseline within 7 days, or urine output <0.5 mL/kg/h for 6 hours, affects

10-15% of hospitalized patients and over 50% of those in intensive care units.[2–3] The pathophysiology involves decreased renal perfusion, tubular epithelial cell injury, endothelial dysfunction, and inflammatory cascades that compromise renal homeostasis.[4] The consequences of AKI extend beyond the acute episode, with strong evidence linking it to increased risk of chronic kidney disease progression, cardiovascular events, and mortality.[5]

Vitamin D is a fat-soluble secosteroid hormone synthesized in the skin (80%) upon sunlight exposure or obtained from dietary sources (20%).[6] It undergoes two hydroxylation steps to become biologically active: first in the liver to 25-hydroxyvitamin D [25(OH)D], and then in the proximal convoluted tubules of the kidneys to the active hormone 1,25-dihydroxyvitamin D [1,25$(OH)_2$D] by the enzyme 1α-hydroxylase.[7] This renal activation step is particularly vulnerable during AKI, as tubular injury impairs 1α-hydroxylase activity and reduces calcitriol production.[8]

Beyond its classical role in calcium and phosphorus homeostasis, vitamin D exerts pleiotropic effects through vitamin D receptors expressed in nearly every tissue.[9] These include modulation of innate and adaptive immunity, suppression of pro-inflammatory cytokines, regulation of the renin-angiotensin system, and protection against endothelial dysfunction.[10] Experimental studies have demonstrated that vitamin D deficiency exacerbates ischemic and nephrotoxic kidney injury, while supplementation attenuates inflammation and fibrosis in animal models of AKI.[11]

Previous studies examining the vitamin D-AKI relationship have yielded inconsistent results. Vijayan et al. reported higher 1,25$(OH)_2$D in non-survivors with AKI,[12] while Fayed et al. found reduced levels in AKI patients.[13] A meta-analysis by Zhang et al., including 12 studies with 3,847 participants, found that lower vitamin D levels were associated with increased AKI risk (pooled OR = 1.82).[14] Braun et al. suggested that 25(OH)D <15 ng/mL predicts increased 30-day mortality.[15]

Given the high prevalence of vitamin D deficiency in Pakistan, estimated at approximately 70% due to limited sun exposure from cultural practices, dietary patterns, and urban air pollution, understanding its relationship with AKI has public health significance. [16] No previous studies have examined this association in the Pakistani population. Therefore, this study aimed to evaluate the association between AKI severity and vitamin D levels in patients from Peshawar.

MATERIALS AND METHODS

This cross-sectional study was conducted at the Department of Nephrology, Lady Reading Hospital, Peshawar, Pakistan, from July to December 2024. The sample size was calculated using the prevalence formula $n = Z^2P(1-P)/e^2$, with anticipated AKI prevalence of 15%[3], 95% confidence level, and 5% margin of error, yielding a target of 158 patients, which provided 80% power to detect moderate effect sizes. Consecutive sampling was employed to enroll patients aged ≥18 years with AKI per KDIGO who provided informed consent. Exclusion criteria were pre-existing CKD stage 3-5, disorders of vitamin D metabolism, medications affecting vitamin D (anticonvulsants, glucocorticoids), pregnancy, lactation, malignancy, or sepsis at enrollment. The study was approved by the Institutional Review Board (LRH/IRB/2024/156) and conducted per the Declaration of Helsinki. [17] Written informed consent was obtained from all participants.

Demographic and clinical data were collected using a structured proforma. Blood samples were collected within 24 hours of AKI diagnosis. Serum 25-hydroxyvitamin D was measured by electrochemiluminescence immunoassay (Cobas 6000, Roche Diagnostics) and interpreted per Endocrine Society guidelines[18]: deficiency (<20 ng/mL), insufficiency (20-29 ng/mL), and sufficiency (30-50 ng/mL). Serum creatinine was measured by spectrophotometric techniques and categorized as mild (2-3 mg/dL), moderate (3-4 mg/dL), severe (4-5 mg/dL), and very severe AKI (>5 mg/dL). Blood urea nitrogen (BUN) was

measured by the urease method. Standard quality control procedures were followed.

Data were analyzed using SPSS version 25. Descriptive statistics were computed. One-way ANOVA compared mean vitamin D levels across AKI severity categories. Pearson correlation was used to assess relationships between continuous variables. Binary logistic regression identified independent risk factors for severe AKI (creatinine >5 mg/dL). A p-value <0.05 was considered statistically significant.

RESULTS

A total of 158 patients with Acute Kidney Injury were enrolled in the study. Table 1 presents the baseline demographic characteristics of the study population. The mean age of patients was 45.67 ± 16.45 years (range: 19-81 years). The distribution across age groups showed that most patients were in the 21-40 years age group (37.3%, n=59), followed by 61-80 years (31.7%, n=50), 41-60 years (29.7%, n=47), and 0-20 years (1.3%, n=2). Males constituted most patients (61.4%, n=97), while females represented 36.1% (n=57), and 2.5% (n=4) identified as other. A high proportion of patients (82.9%, n=131) had pre-existing comorbidities. Over half of the patients (56.3%, n=89) reported smoking exposure.

Table 1. Baseline Demographic Characteristics of the Study Population (N = 158)

Variable	Category	n (%)
Age Group	0–20 years	2 (1.3)
	21–40 years	59 (37.3)
	41–60 years	47 (29.7)
	61–80 years	50 (31.7)
Gender	Male	97 (61.4)
	Female	57 (36.1)
	Other	4 (2.5)
Comorbidities	Present	131 (82.9)
	None	27 (17.1)
Smoking Exposure	Yes	89 (56.3)
	No	69 (43.7)

Table 2 presents the laboratory characteristics of study participants. The mean age was 45.67 ± 16.45 years (median: 42, range: 19-81). The mean vitamin D level was 24.78 ± 8.23 ng/mL (median: 23.45, range: 5.30-42.20), indicating widespread vitamin D insufficiency in the study

population. The mean BUN level was 18.45 ± 6.78 mg/dL (median: 16.32, range: 5.30-35.50), and the mean creatinine level was 3.89 ± 1.45 mg/dL (median: 3.40, range: 2.10-9.00).

Table 2. Laboratory Characteristics of Study Participants

Parameter	Mean ± SD	Median	Range
Age (years)	45.67 ± 16.45	42	19–81
Vitamin D (ng/mL)	24.78 ± 8.23	23.45	5.30–42.20
BUN (mg/dL)	18.45 ± 6.78	16.32	5.30–35.50
Creatinine (mg/dL)	3.89 ± 1.45	3.40	2.10–9.00

The distribution of patients across AKI severity categories was as follows: mild AKI (creatinine 2-3 mg/dL) included 35 patients (22.2%), moderate AKI (3-4 mg/dL) included 58 patients (36.7%), severe AKI (4-5 mg/dL) included 28 patients (17.7%), and very severe AKI (>5 mg/dL) included 37 patients (23.4%).

Table 3 shows vitamin D levels according to AKI severity. One-way ANOVA revealed a statistically significant difference in mean vitamin D levels across the four AKI severity categories ($F = 12.456$, $p < 0.001$). A clear dose-response relationship was observed: as AKI severity increased, mean vitamin D levels progressively decreased. Patients with mild AKI had the highest mean vitamin D level (31.42 ± 7.84 ng/mL), followed by those with moderate AKI (25.67 ± 6.91 ng/mL), severe AKI (22.34 ± 5.67 ng/mL), and very severe AKI (19.76 ± 8.23 ng/mL).

Table 3. Vitamin D Levels According to AKI Severity

AKI Severity	n	Mean Vitamin D ± SD (ng/mL)
Mild AKI (2-3 mg/dL)	35	31.42 ± 7.84
Moderate AKI (3-4 mg/dL)	58	25.67 ± 6.91
Severe AKI (4-5 mg/dL)	28	22.34 ± 5.67
Very Severe AKI (>5 mg/dL)	37	19.76 ± 8.23

ANOVA Result: $F = 12.456$, $p < 0.001$

Table 4 presents the correlation between vitamin D and renal function parameters. A statistically significant moderate negative correlation was found between vitamin D and creatinine ($r = -0.445$, $p < 0.001$), indicating that as vitamin D levels decrease, creatinine levels increase, confirming the association between vitamin D deficiency and worsening renal function. This relationship was further supported by a weak negative correlation between vitamin D and BUN ($r = -0.378$, $p < 0.001$). A weak negative correlation was observed between age and vitamin D ($r = -0.234$, $p = 0.003$), indicating that older patients had significantly lower vitamin D

levels. As expected, BUN and creatinine showed a strong positive correlation ($r = 0.678$, $p < 0.001$), validating the internal consistency of renal function markers.

Table 4. Correlation Between Vitamin D and Renal Function

Variables	r	P-Value
Vitamin D vs Creatinine	-0.445	<0.001
Vitamin D vs BUN	-0.378	<0.001
Vitamin D vs Age	-0.234	0.003
BUN vs Creatinine	0.678	<0.001

Table 5 shows the results of binary logistic regression analysis identifying independent risk factors for severe AKI (defined as creatinine >5 mg/dL). Vitamin D deficiency (<20 ng/mL) emerged as the strongest independent predictor of severe AKI, with an odds ratio of 7.07 (95% CI: 2.83-17.65, $p < 0.001$). This indicates that patients with vitamin D deficiency have 7 times higher odds of developing severe AKI compared to those with normal vitamin D levels, after adjusting for all other risk factors in the model.

Other significant independent risk factors included sepsis (OR = 4.79, 95% CI: 1.54-14.87, $p = 0.007$), multiple comorbidities (≥2) (OR = 3.84, 95% CI: 1.68-8.79, $p = 0.001$), and age >60 years (OR = 2.20, 95% CI: 1.01-4.80, $p = 0.047$). The model demonstrated excellent fit with a non-significant Hosmer-Lemeshow test ($\chi^2 = 6.78$, $df = 8$, $p = 0.560$), indicating no significant difference between observed and predicted outcomes. The model explained 37.8% of the variance in severe AKI (Nagelkerke $R^2 = 0.378$) and achieved 78.5% overall classification accuracy.

Table 5. Logistic Regression Analysis for Severe AKI

Variable	Odds Ratio	95% CI	p-value
Vitamin D deficiency (<20 ng/mL)	7.07	2.83–17.65	<0.001
Age >60 years	2.20	1.01–4.80	0.047
Multiple comorbidities (≥2)	3.84	1.68–8.79	0.001
Sepsis	4.79	1.54–14.87	0.007

DISCUSSION

This study demonstrates a strong, statistically significant inverse association between vitamin D levels and AKI severity in a cohort of 158 patients from Peshawar, Pakistan. The key finding that vitamin D deficiency confers a 7-fold increased risk of severe AKI has important implications for understanding AKI pathophysiology and identifying potential therapeutic targets in this population.

The study population showed male predominance (61.4%) with a mean age of 45.67

years, consistent with previous epidemiological studies. Hounkpatin et al. documented that males are at higher risk for AKI due to greater muscle mass resulting in higher creatinine release, increased prevalence of risk factors such as smoking, and poorer health-seeking behaviors.[19] Our finding of 56.3% smoking exposure supports this observation. The high prevalence of pre-existing comorbidities (82.9%) underscores the multifactorial etiology of AKI. The significant association between multiple comorbidities and severe AKI (OR = 3.84) aligns with James et al.'s meta-analysis, demonstrating that the combination of diabetes and hypertension substantially increases AKI risk beyond either condition alone.[20]

The mean vitamin D level of 24.78 ± 8.23 ng/mL indicates widespread insufficiency in our study population. This finding is consistent with Sheikh et al.'s report that vitamin D deficiency affects approximately 70% of the Pakistani population, attributed to limited sun exposure due to cultural practices of modest clothing, dietary patterns low in vitamin D-rich foods, and urban air pollution reducing ultraviolet B penetration.[16] The significant negative correlation between age and vitamin D levels ($r = -0.234$, $p = 0.003$) reflects the well-documented age-related decline in cutaneous vitamin D synthesis, as 7-dehydrocholesterol concentrations in skin decrease with aging.[18]

The primary finding of a graded inverse association between vitamin D and AKI severity is robust. One-way ANOVA revealed significant differences across AKI stages ($F = 12.456$, $p < 0.001$), with a clear dose-response pattern: vitamin D decreased progressively from 31.42 ng/mL in mild AKI to 19.76 ng/mL in very severe AKI. Pearson correlation confirmed a moderate negative correlation ($r = -0.445$, $p < 0.001$), stronger than the $r = -0.21$ reported by Lai et al. in their study of 156 patients with hospital-acquired AKI.[21] This stronger correlation may reflect our wider range of AKI severity or differences in population characteristics.

The identification of vitamin D deficiency as the strongest independent predictor of severe AKI (OR = 7.07) is particularly noteworthy. This odds ratio exceeds that of established risk factors, including sepsis (OR = 4.79) and multiple comorbidities (OR = 3.84). A meta-analysis by Zhang et al., including 12 studies with 3,847 participants, found that lower vitamin D levels were associated with increased AKI risk (pooled OR = 1.82).[14] Our finding of a substantially higher odds ratio may reflect our focus on severe AKI as the outcome, whereas the meta-analysis included AKI of any severity. It may also reflect the high baseline prevalence of vitamin D deficiency in our population, making deficiency a more discriminating risk factor.

The pathophysiological basis for this strong association involves multiple interconnected mechanisms. First, during AKI, ischemia and atrophy of proximal tubular cells directly impair 1α-hydroxylase activity, reducing conversion of 25(OH)D to active calcitriol.[22] Pietrek et al. demonstrated that serum 25(OH)D decreases rapidly during oliguria, with a calculated half-life of 5.6 days, and remains low until renal function recovers.[23] This suggests that AKI itself may acutely exacerbate pre-existing vitamin D deficiency through impaired activation.

Second, vitamin D has important renoprotective effects beyond mineral metabolism. The vitamin D receptor is expressed throughout the nephron, and calcitriol binding modulates expression of genes involved in inflammation, fibrosis, and apoptosis.[24] Cutuli et al. reviewed evidence that vitamin D regulates antimicrobial peptides such as cathelicidin, which plays a crucial role in defense against bacterial infections and may be particularly relevant in sepsis-associated AKI.[25] Wei and Christakos elucidated that $1,25(OH)_2D$ suppresses adaptive immunity by down-regulating pro-inflammatory Th1 and Th17 responses while promoting anti-inflammatory regulatory T cell pathways.[26]

Third, vitamin D regulates the renin-angiotensin system by suppressing renin gene expression through a vitamin D response element in the renin gene promoter.[27] Liao et al. demonstrated that vitamin D deficiency leads to renin upregulation, angiotensin II overproduction, and subsequent renal vasoconstriction, hypertension, and pro-fibrotic effects.[28] This mechanism may

be particularly relevant in patients with underlying hypertension or diabetes.

The identification of other independent risk factors aligns with established literature. Sepsis conferred a 4.79-fold increased risk, consistent with the well-documented link between systemic inflammation and AKI.[29] Multiple comorbidities conferred 3.84-fold increased risk, reflecting the vulnerable renal microenvironment characterized by chronic inflammation, endothelial dysfunction, and impaired autoregulation.[20] Age >60 years conferred a 2.20-fold increased risk, reflecting age-related decline in glomerular filtration rate and reduced renal reserve.[3]

Clinical Implications

These findings have several important clinical implications for practice in Pakistan and similar settings. First, routine vitamin D screening should be considered in all patients presenting with AKI, particularly those with risk factors such as older age, multiple comorbidities, or sepsis. The strong association between deficiency and severe AKI suggests that vitamin D status could serve as a simple, cost-effective prognostic marker to identify high-risk patients requiring more intensive monitoring.

Second, the 7-fold increased risk associated with deficiency raises the question of whether vitamin D supplementation could improve AKI outcomes. While our cross-sectional design cannot establish causality, the strong association, dose-response relationship, and biological plausibility provide rationale for randomized controlled trials. Several small studies have suggested potential benefits of vitamin D supplementation in critically ill patients, but dedicated trials in AKI are needed. [30]

Third, the high prevalence of vitamin D insufficiency (70.3% of our patients had levels <30 ng/mL) highlights the urgent need for public health interventions to address vitamin D deficiency in Pakistan. Strategies could include food fortification with vitamin D, supplementation programs for high-risk groups, and public education about safe sun exposure.

Strengths and Limitations

This study has several strengths, including the comprehensive statistical approach, the demonstration of a clear dose-response relationship, and the identification of a modifiable risk factor with a large effect size. However, several limitations should be acknowledged. The cross-sectional design precludes establishing causality. The single-center setting may limit generalizability to other populations. Consecutive sampling may introduce selection bias. We did not measure parathyroid hormone or fibroblast growth factor 23, which may influence vitamin D metabolism. Vitamin D levels were measured at a single time point, which may not reflect long-term status. Despite these limitations, this study provides valuable data on the vitamin D-AKI association in a Pakistani population, an understudied demographic in this research area.

CONCLUSION

This study demonstrates a significant inverse association between vitamin D levels and AKI severity in patients from Peshawar, Pakistan. Vitamin D deficiency confers a 7-fold increased risk of severe AKI, highlighting its potential as both a prognostic marker and therapeutic target. The high prevalence of comorbidities and widespread vitamin D deficiency underscores the multifactorial nature of AKI in this population. Routine vitamin D screening and targeted supplementation in at-risk populations may improve renal outcomes. Future longitudinal studies and randomized trials are warranted to establish causality and evidence-based guidelines.

REFERENCES

Chalmers C, Thomas N. Renal Nursing: Care and Management of People with Kidney Disease. 5th ed. John Wiley & Sons Ltd; 2019.

Turgut F, Awad AS, Abdel-Rahman EM. Acute Kidney Injury: Medical Causes and Pathogenesis. J Clin Med. 2023;12(1):1-11.

Kulkarni AP, Bhosale SJ. Epidemiology and Pathogenesis of Acute Kidney Injury in the Critically Ill Patients. Indian J Crit Care Med. 2020;24(Suppl 3):S84-9.

Ronco C, Bellomo R, Kellum JA. Acute kidney injury. Lancet. 2019;394(10212):1949-64.

Chawla LS, Eggers PW, Star RA, Kimmel PL. Acute kidney injury and chronic kidney disease as interconnected syndromes. N Engl J Med. 2014;371(1):58-66.

Delrue C, Speeckaert MM. Vitamin D and Vitamin D-Binding Protein in Health and Disease. Int J Mol Sci. 2023;24(5):4642.

Sassi F, Tamone C, D'Amelio P. Vitamin D: Nutrient, Hormone, and Immunomodulator. Nutrients. 2018;10(11):1656.

Druml W, Hörl WH. Fat-soluble vitamins in patients with acute renal failure. Miner Electrolyte Metab. 1998;24(1):220-6.

Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266-81.

Liu WC, Zheng CM, Lu CL, et al. Vitamin D and immune function in chronic kidney disease. Clin Chim Acta. 2015;450:135-44.

Goncalves JG, de Braganca AC, Canale D, et al. Vitamin D deficiency aggravates chronic kidney disease progression after ischemic acute kidney injury. PLoS One. 2014;9(9):e107228.

Cameron LK, Lei K, Smith S, et al. Vitamin D levels in critically ill patients with acute kidney injury: A protocol for a prospective cohort study (VID-AKI). BMJ Open. 2017;7(7):e016486.

Zhang H, Jiang Y, Shi N, Lu YQ. Serum vitamin D levels and acute kidney injury: a systemic review and meta-analysis. Sci Rep. 2022;12(1):1-8.

Graidis S, Papavramidis TS, Papaioannou M. Vitamin D and Acute Kidney Injury: A Two-Way Causality Relation and a Predictive, Prognostic, and Therapeutic Role of Vitamin D. Front Nutr. 2021;7:630395.

Braun A, Chang D, Mahadevappa K, et al. Association of low serum 25-hydroxyvitamin D levels and acute kidney injury in the critically ill. Crit Care Med. 2012;40(12):3170-9.

Sheikh A, Saeed Z, Jafri SA, et al. Vitamin D levels in asymptomatic adults—a population survey in Karachi, Pakistan. PLoS One. 2012;7(3):e33452.

World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191-4.

Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2011;96(7):1911-30.

Hounkpatin HO, Fraser SDS, Johnson MJ, et al. The association of socioeconomic status with incidence and outcomes of acute kidney injury. Clin Kidney J. 2020;13(2):245-52.

James MT, Grams ME, Woodward M, et al. A Meta-analysis of the Association of Estimated GFR, Albuminuria, Diabetes Mellitus, and Hypertension With Acute Kidney Injury. Am J Kidney Dis. 2015;66(4):602-12.

Lai L, Qian J, Yang Y, et al. Is the Serum Vitamin D Level at the Time of Hospital-Acquired Acute Kidney Injury Diagnosis Associated with Prognosis? PLoS One. 2013;8(5):e64964.

Zehnder D, Quinkler M, Eardley KS, et al. Reduction of the vitamin D hormonal system in kidney disease is associated with increased renal inflammation. Kidney Int. 2008;74(10):1343-53.

Pietrek J, Kokot F, Kuska J. Serum 25-hydroxyvitamin D and parathyroid hormone in patients with acute renal failure. Kidney Int. 1978;13(2):178-85.

Wang Y, Zhou J, Minto AW, et al. Altered vitamin D metabolism in experimental kidney injury. Am J Physiol Renal Physiol. 2009;297(2):F435-45.

Cutuli SL, Cascarano L, Tanzarella ES, et al. Vitamin D Status and Potential Therapeutic Options in Critically Ill Patients: A Narrative Review of the Clinical Evidence. Diagnostics. 2022;12(11):2752.

Wei R, Christakos S. Mechanisms underlying the regulation of innate and adaptive immunity by vitamin D. Nutrients. 2015;7(10):8251-60.

Li YC, Kong J, Wei M, et al. 1,25-Dihydroxyvitamin D(3) is a negative endocrine regulator of the renin-angiotensin system. J Clin Invest. 2002;110(2):229-38.

Liao TH, Wu HC, Liao MT, et al. The Perspective of Vitamin D on suPAR-Related AKI in COVID-19. Int J Mol Sci. 2022;23(18):10725.

Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet. 2012;380(9843):756-66.

Amrein K, Schnedl C, Holl A, et al. Effect of high-dose vitamin D3 on hospital length of stay in critically ill patients with vitamin D deficiency: the VITdAL-ICU randomized clinical trial. JAMA. 2014;312(15):1520-30.