Ukrainian Journal of Nephrology and Dialysis

Scientific and Practical, Medical Journal

Founder:

- National Kidney Foundation of Ukraine

ISSN 2304-0238;
eISSN 2616-7352

Journal homepage: https://ukrjnd.com.ua

Research article

doi: 10.31450/ukrjnd.1(89).2026.05

Omar Mohammed Ramadhan, Basma A. Al-Mshhdani, Qadoori Zedan Khalaf

Mineral-axis heterogeneity in early acute kidney injury: A cross-sectional clustering analysis

Department of Chemistry, College of Education, Al-Iraqia University, Baghdad, Iraq

Citation: Ramadhan OM, Al-Mshhdani BA, Khalaf QZ. Mineral-axis heterogeneity in early acute kidney injury: A cross-sectional clustering analysis. Ukr J Nephrol Dialys. 2026;1(89):35-41. doi: 10.31450/ukrjnd.1(89).2026.05.

Abstract. *The kidney plays an essential role in maintaining mineral homeostasis through regulation of calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism. Although mineral disturbances are well established in chronic kidney disease, their early presentation in acute kidney injury (AKI) and their relationship to KDIGO-defined severity remain incompletely understood. This study aimed to evaluate early alterations in mineral metabolism within 24 hours of AKI diagnosis and to explore potential biochemical heterogeneity using clustering analysis.*

Methods. This cross-sectional study included 100 participants: 50 patients with AKI defined according to KDIGO criteria and 50 age- and sex-matched controls without AKI. Serum calcium, phosphorus, PTH, and 25-hydroxyvitamin D [25(OH)D] were measured within 24 hours of AKI diagnosis. Comparisons were performed between AKI patients and controls and across KDIGO stages. Unsupervised k-means clustering was applied to identify distinct mineral-axis profiles within the AKI group.

Results. Compared with controls, patients with AKI had significantly lower calcium and 25(OH)D levels and higher phosphorus and PTH concentrations (all $p < 0.0001$). These disturbances were observed across AKI stages, without a consistent stepwise trend according to creatinine-defined severity. Clustering analysis identified two distinct mineral-axis profiles among AKI patients. The two-cluster solution explained 23.6% of total variability ($R^2 = 0.236$) and showed clear separation. Cluster membership was not associated with creatinine levels, AKI stage, urea concentration, or dialysis requirement, indicating biochemical heterogeneity independent of conventional severity classification.

Conclusions. Early AKI is accompanied by significant disturbances in mineral metabolism that do not parallel creatinine-based staging. Distinct mineral-axis profiles can be identified within 24 hours of diagnosis, suggesting underlying biochemical heterogeneity. Further prospective studies are needed to determine the clinical and prognostic significance of these early mineral alterations.

Keywords: *acute kidney injury, mineral metabolism, calcium, phosphorus, parathyroid hormone, vitamin D, cluster analysis.*

Article history:

Received December 06, 2025
Received in revised form February 13, 2026
Accepted February 15, 2026

Conflict of interest. The authors declare no conflict of interest.

Correspondence should be addressed to Omar Mohammed Ramadhan: omar.mramadhan73@gmail.com

УДК: 616.61-008.6-036.11-071:519.237.8

Омар Мохаммед Рамадхан, Басма А. Аль-Мшхдані, Кадурі Зедан Халаф

Гетерогенність мінерального обміну в ранньому періоді гострого пошкодження нирок: поперечний кластерний аналіз

Кафедра хімії, Коледж освіти, Університет Аль-Іракія, Багдад, Ірак

Резюме. *Нирки відіграють ключову роль у підтримці мінерального гомеостазу шляхом регуляції обміну кальцію, фосфору, паратиреоїдного гормону (ПТГ) та вітаміну D. Хоча порушення мінерального обміну добре описані у хворих на хронічній хворобі нирок, їх ранні прояви у разі гострого пошкодження нирок (ГПН) та зв'язок із тяжкістю за класифікацією KDIGO залишаються недостатньо вивченими. Метою дослідження було оцінити ранні зміни мінерального обміну протягом перших 24 годин після встановлення діагнозу ГПН та дослідити можливу біохімічну гетерогенність за допомогою кластерного аналізу.*

Методи. У поперечне дослідження включено 100 учасників: 50 пацієнтів з ГПН, визначеним згідно критеріїв KDIGO, та 50 осіб контрольної групи, зіставних за віком і статтю, без ознак ГПН. Рівні кальцію, фосфору, ПТГ та 25-гідроксивітаміну D [25(OH)D] у сироватці крові визначали протягом 24 годин після встановлення діагнозу. Порівняння проводили між групами та між стадіями KDIGO. Для виявлення різних профілів мінерального обміну в групі ГПН застосовано некерований кластерний аналіз методом k-середніх.

Результати. Порівняно з контролем у пацієнтів із ГПН виявлено достовірно нижчі рівні кальцію та 25(OH)D і вищі концентрації фосфору та ПТГ (усі маркери $p < 0,0001$). Ці порушення спостерігалися на всіх стадіях ГПН без чіткої залежності від рівня креатиніну. Кластерний аналіз дозволив виділити два різні профілі серед пацієнтів із ГПН. Двокластерна модель пояснювала 23,6% загальної варіабельності ($R^2 = 0,236$) та демонструвала чітке розмежування груп. Належність до кластера не була пов'язана з рівнем креатиніну, стадією ГПН, концентрацією сечовини чи потребою в діалізі, що свідчить про біохімічну гетерогенність, незалежну від традиційної класифікації тяжкості.

Висновки. Ранній період ГПН супроводжується суттєвими порушеннями мінерального обміну, які не відповідають ступеню тяжкості за рівнем креатиніну. Уже в перші 24 години після встановлення діагнозу можна виділити різні профілі мінерального обміну, що вказує на наявність біохімічної гетерогенності. Необхідні подальші проспективні дослідження для визначення клінічного та прогностичного значення цих ранніх змін.

Ключові слова: *гостре пошкодження нирок, мінеральний обмін, кальцій, фосфор, паратгормон, вітамін D, кластерний аналіз*

Introduction. Acute kidney injury (AKI) is a common clinical problem and remains a major cause of short-term illness and death worldwide [1, 2]. In everyday practice, AKI is diagnosed and classified primarily by monitoring changes in serum creatinine levels and urine output, according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines [3]. Although these measures are useful for assessing changes in kidney filtration, they do not fully reflect the broader metabolic disturbances that can develop when kidney function suddenly declines. The kidneys play a central role in regulating mineral metabolism. It maintains calcium and phosphorus balance, takes part in vitamin D activation, and influences parathyroid hormone (PTH) secretion [4, 5]. Disturbances of this tightly regulated axis are well established in chronic kidney disease; however, less attention has been given to early mineral metabolic changes during the acute phase of kidney injury [6]. Although hypocalcemia and hyperphosphatemia are recognized in AKI, the timing, pattern, and variability of associated changes in PTH and 25-hydroxyvitamin D [25(OH)D] remain incompletely characterized [7].

Omar Mohammed Ramadhan
omar.mramadhan73@gmail.com

An important unresolved question is whether alterations in mineral metabolism parallel creatinine-based severity staging or whether they represent independent biochemical responses [7]. If mineral disturbances occur early and do not show a stepwise trend across KDIGO stages, this would suggest that creatinine alone does not adequately reflect the metabolic dimension of AKI [6, 7]. Besides, conventional analyses usually look at mineral markers separately, which may miss some underlying biochemical patterns within the AKI population.

Data-driven analytical approaches like unsupervised clustering offer an opportunity to explore biological heterogeneity beyond traditional classification systems. Distinct mineral-axis profiles, if identified, would improve understanding of early pathophysiological responses in AKI and reveal phenotypic variability that is not captured by standard staging criteria [8].

Therefore, the present study aimed to characterize early alterations in calcium, phosphorus, PTH, and 25(OH)D within 24 hours of AKI diagnosis and to ex-

amine their relationship to KDIGO stage. In addition, we applied unsupervised clustering analysis to investigate whether distinct mineral-axis profiles could be identified among patients with AKI.

Patients and methods. *Study design and participants*. This cross-sectional study was held at the Medical City Complex in Baghdad, Iraq, from April to September 2025.

Ethical Considerations. The study protocol was reviewed and approved by the Institutional Ethics Committee of Medical City, Baghdad, Iraq, (Ethics Committee approval number: 5501 in Feb 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment and sample collection.

A total of 100 participants, including 50 patients diagnosed with AKI and 50 controls matched for age and sex, were included in the study.

AKI was defined as per KDIGO: increased serum creatinine by ≥0.3 mg/dL within 48 hours; increased to ≥1.5 times baseline within the prior seven days; or urine output less than 0.5 mL/kg/h for at least six hours. Patients were categorized into KDIGO stages 1, 2, and 3 based on serum creatinine criteria at the time of diagnosis. All laboratory measurements in the AKI group were obtained within twenty-four hours after diagnosis to evaluate early biochemical alterations.

The control group consisted of individuals without clinical or laboratory evidence of AKI with estimated glomerular filtration rate (eGFR) greater than 60 mL/min/1.73 m^2 CKD-EPI.

Participants were excluded if they had:

- Previously diagnosed CKD
- Current vitamin D supplementation
- Known parathyroid disorders
- Active malignancy
- Conditions known to significantly affect calcium–phosphorus metabolism.

Sample collection and laboratory measurements. Venous blood samples were collected under aseptic conditions into serum gel separator tubes. Samples were allowed to clot at room temperature for approximately 30 minutes and were then centrifuged at 4000 rpm for 10 minutes. Serum was separated and analyzed immediately.

Serum creatinine and urea were measured using automated enzymatic colorimetric assays on chemistry analyzer (Cobas c311 /Roche Diagnostics, Basel, Switzerland).

Total serum calcium and phosphorus concentrations were determined using flame atomic absorption spectrophotometry (Cobas c111 automated analyzer / Roche Diagnostics, Basel, Switzerland) with commercially available analytical standards and reagents.

Serum 25(OH)D was measured using the VIDAS® system (VIDAS® 25 OH Vitamin D Total kit/ VIDAS® or Mini VIDAS® system (bioMérieux), Marcy-l'Étoile, France), based on enzyme-linked fluorescent assay (ELFA) technology. The assay detects total circulating 25(OH)D.

Serum PTH levels were measured using a chemiluminescent immunoassay (CLIA) method (Cobas e analyzer with Elecsys® PTH kit /Roche Diagnostics, Basel, Switzerland).

All assays were performed in accordance with manufacturer protocols and standard laboratory quality control procedures. Laboratory reference ranges were as follows: calcium: 8.6–10.2 mg/dL, phosphorus: 2.5–4.5 mg/dL, PTH: 15–65 pg/mL, 25(OH)D sufficiency: ≥30 ng/mL; insufficiency: 20–29 ng/mL; deficiency: <20 ng/mL. Internal quality control procedures were performed daily according to manufacturer recommendations. All enrolled participants had complete laboratory data for the primary study variables (calcium, phosphorus, PTH, 25(OH)D, creatinine, and urea). No imputation procedures were required.

Statistical analysis. Statistical analyses were conducted with the SAS software version 9.4 (SAS Institute, USA). Continuous variables were tested for normal distribution by appropriate normality tests. Normally distributed variables were expressed as mean and standard deviation (M ± SD) and compared between two groups with the independent t-test. Skewed variables were presented as median and interquartile range [Me (Q25-Q75)] and compared by the Mann-Whitney U test. Stage-based differences were analyzed with the Kruskal–Wallis test, while categorical variables were compared using chi-square testing.

To identify potential patterns of mineral-axis variability within the AKI population, we performed unsupervised k-means clustering based on serum calcium, phosphorus, PTH, and 25(OH)D measurements. Variables were standardized (z-score transformation) prior to clustering to allow equal weighting among them. Therefore, the optimal number of clusters was determined based on an assessment of within-cluster variance. Principal component analysis (PCA) was used for visualization of cluster separation as well as estimation of proportions of total variance explained by principal components. Clinical and biochemical characteristics were then compared between clusters to assess their association with creatinine level, AKI stage, and dialysis requirement.

Results. *Study population*. A total of 100 participants were included, comprising 50 patients with AKI and 50 non-AKI controls. There were no significant differences between groups in age, sex distribution, or prevalence of diabetes and cardiovascular disease. Hypertension was more common among AKI patients. Within the AKI cohort, patients were represented across KDIGO stages 1 through 3, with roughly 25% requiring dialysis support. As anticipated, indicators of renal function were significantly higher in the AKI group compared with controls, with both serum creatinine and urea levels markedly elevated ($p < 0.0001$).

In addition, patients with AKI exhibited pronounced disturbances in mineral metabolism relative to

the control group. Serum calcium and 25(OH)D concentrations were significantly lower in the AKI group, while phosphorus and parathyroid hormone (PTH) levels were significantly higher (all $p < 0.0001$). The observed biochemical pattern was characterized by concomitant hypocalcemia, hyperphosphatemia, elevated PTH, and reduced 25(OH)D levels in AKI patients. The consistency and statistical strength of these differences suggest an early disruption of mineral homeostasis accompanying AKI (Table 1).

Table 1

Clinical characteristics and laboratory findings of AKI patients and non-AKI controls

Variable	AKI patients (n = 50)	Controls (n = 50)	p-value
Age (years)	52.6 ± 14.2	49.8 ± 13.7	0.323
Age ≥ 60 years (%)	28%	20%	0.412
Male sex (%)	60%	48%	0.184
Hypertension (%)	52%	32%	0.041
Diabetes Mellitus (%)	36%	22%	0.095
Cardiovascular Disease (%)	18%	10%	0.273
Dialysis (%)	24% (12/50)	–	–
KDIGO Stage (%)	Stage 1: 40% Stage 2: 34% Stage 3: 26%	–	–
Urea (mg/dL)	101 (92.3–107.0)	28 (27–29)	<0.0001
Creatinine (mg/dL)	2.85 (2.63–3.09)	1.25 (0.97–1.41)	<0.0001
Calcium (mg/dL)	8.81 (8.72–8.95)	9.60 (9.50–9.65)	<0.0001
Phosphorus (mg/dL)	5.18 (5.04–5.27)	3.80 (3.71–3.84)	<0.0001
PTH (pg/mL)	40 (37–43)	32 (31–33)	<0.0001
25(OH)D (ng/mL)	20.2 (19.4–21.6)	29.3 (27.6–31.2)	<0.0001

Age is presented as M ± SD. Skewed continuous variables are presented as Me (Q25–Q75). Categorical variables are expressed as percentages. Between-group comparisons were performed using independent t-test (age), Mann–Whitney U test (continuous skewed variables), and chi-square test (categorical variables). Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone.

Mineral metabolism within 24 hours across KDIGO stages. To determine whether early mineral-axis abnormalities were proportional to creatinine-based severity, AKI patients were stratified according to KDIGO stage. All biochemical measurements were obtained within 24 hours of AKI diagnosis. Disturbances in calcium, phosphorus, PTH, and 25(OH)D were observed across all stages at this early time point. However, comparison between stages did not demonstrate a progressive or graded increase in mineral abnormalities with advancing stage. Median values of these parameters were comparable among stages, and statistical testing did not support a consistent trend across severity categories (Table 2).

Table 2

Mineral metabolism within 24 hours according to KDIGO stage in AKI patients

Variable	Stage 1 (n = 20)	Stage 2 (n = 17)	Stage 3 (n = 13)	p-value
Calcium (mg/dL)	8.84 (8.74–8.96)	8.75 (8.64–8.88)	8.83 (8.75–8.97)	0.177
Creatinine (mg/dL)	2.84 (2.69–3.02)	2.82 (2.55–3.13)	2.90 (2.60–3.24)	0.899
Phosphorus (mg/dL)	5.09 (5.04–5.25)	5.22 (5.09–5.35)	5.08 (5.00–5.26)	0.435
PTH (pg/mL)	41.5 (38.5–43.5)	40.0 (36.8–45.3)	39.0 (35.0–40.0)	0.152
Urea (mg/dL)	101.0 (95.0–105.5)	107.0 (96.5–113.3)	96.0 (89.0–101.0)	0.054
25(OH)D (ng/mL)	20.35 (19.45–21.50)	19.80 (18.48–21.70)	21.00 (19.80–21.45)	0.728

Continuous variables are presented as median (Q25–Q75). Comparisons across stages were performed using the Kruskal–Wallis test. Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone.

Mineral-axis heterogeneity identified by unsupervised clustering. Given the absence of a clear stage-dependent gradient in mineral parameters, unsupervised k-means clustering was performed to explore potential biochemical heterogeneity within the AKI cohort. Clustering was conducted using calcium, phosphorus, PTH, and 25(OH)D. Two distinct mineral-axis profiles were identified. Principal component analysis demonstrated separation of patients into two clusters along mineral-related dimensions (Fig. 1A).

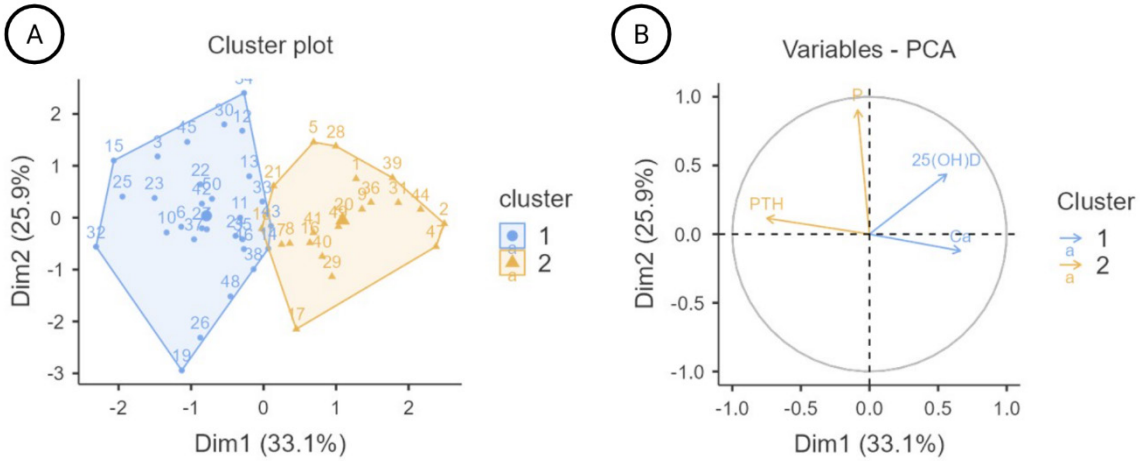

Fig. 1. Mineral-axis heterogeneity identified by unsupervised clustering in AKI.

Panel A: Principal component analysis scatter plot showing separation of AKI patients into two clusters based on calcium, phosphorus, PTH, and 25(OH)D.

Panel B: Standardized cluster profile plot illustrating opposing mineral-axis patterns between clusters.

The two-cluster solution included 29 patients in Cluster 1 and 21 patients in Cluster 2. The total sum of squares was 196.0, of which 46.3 represented between-cluster variation and 149.7 represented within-cluster variation. Thus, the two-cluster model explained 23.6% of the total variability ($R^2 = 0.236$). The corresponding pseudo-F statistic was 14.85, indicating meaningful separation between clusters.

The first principal component explained 33.1% of the variance and the second component explained 25.9%, together accounting for approximately 59% of total variability. Cluster profiling revealed divergent biochemical patterns (Fig. 1B). One cluster was characterized by lower calcium and 25(OH)D levels accompanied by higher PTH concentrations, whereas the second cluster demonstrated the opposite pattern. Phosphorus contributed to cluster separation but showed less pronounced divergence compared with PTH and 25(OH)D.

Importantly, the clusters were not associated with differences in age, AKI stage distribution, serum creatinine, urea, or dialysis requirement (Table 3), indicating that the observed biochemical profiles were not explained by creatinine-based severity classification.

Table 3

Clinical and biochemical characteristics according to mineral-axis cluster

Variable	Cluster 1 (n = 29)	Cluster 2 (n = 21)	p-value
Age (years)	56.0 (40.5–64.8)	50.0 (43.5–54.3)	0.497
Creatinine (mg/dL)	2.84 (2.59–3.08)	2.86 (2.69–3.11)	0.658
Urea (mg/dL)	101.0 (91.8–107.5)	100.0 (95.5–104.0)	0.693
Dialysis (n, %)	6 (20.7%)	6 (28.6%)	0.523
Calcium (mg/dL)	8.75 (8.70–8.85)	8.92 (8.77–8.99)	0.001
Phosphorus (mg/dL)	5.22 (5.04–5.39)	5.11 (5.04–5.24)	0.140
PTH (pg/mL)	42.0 (39.0–46.3)	37.0 (34.8–41.0)	0.0005
25(OH)D (ng/mL)	19.5 (18.5–20.1)	21.6 (20.7–22.4)	<0.0001

Continuous variables are presented as median (Q25–Q75). Comparisons across stages were performed using the Kruskal–Wallis test. Abbreviations: 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone.

Discussion. The present study examined early alterations in mineral metabolism among patients with acute kidney injury and explored whether these changes exhibit internal biochemical heterogeneity. Several observations merit attention.

Within the first 24 hours of diagnosis, patients with AKI demonstrated clear disturbances in calcium–phosphorus homeostasis. Compared with matched controls, serum calcium and 25(OH)D concentrations were reduced, whereas phosphorus and PTH levels were increased. This pattern suggests that disruption of mineral balance occurs early in the course of AKI rather than developing only after prolonged impairment. Previous reports have described hyperphosphatemia and hypocalcemia in critically ill patients with AKI [9, 10]. However, data on the concurrent changes in PTH and 25(OH)D within the first 24 hours are still scarce [6]. Our results demonstrate that aspects of mineral-axis dysregulation appear early after AKI.

The lack of a graded trend across KDIGO stages is particularly interesting. Several studies have shown that mineral parameters did not always follow a stepwise increase or decrease pattern [7]. This observation suggests that creatinine-based staging may not completely reflect the metabolic dimension of AKI. Serum creatinine primarily reflects filtration capacity; mineral metabolism is affected by tubular handling, endocrine signaling, and systemic regulatory pathways [11]. In other words, impairment of endocrine and tubular functions may occur even when the rise in creatinine is modest. Biochemical disruption may therefore occur regardless of how much creatinine has increased.

Another important finding from this study is the identification of two different mineral-axis profiles through unsupervised clustering. These profiles were defined by different patterns in calcium, PTH, and 25(OH)D concentrations. Importantly, membership in clusters did not differ in levels of creatinine, KDIGO stage, urea concentration, or need for dialysis. This observation supports the growing concept that AKI is not a uniform entity but a syndrome with biological variability. Similar clustering approaches in critical care populations have revealed distinct inflammatory and metabolic phenotypes despite comparable traditional severity markers [8, 12]. Our results extend this concept to mineral metabolism, suggesting that patients with comparable creatinine-defined AKI may still differ in their endocrine–metabolic response.

The early clinical significance of mineral-axis disturbance remains to be clarified. Hypocalcemia and hyperphosphatemia can affect cardiac function, neuromuscular stability, and inflammatory pathways [7, 13]. High PTH levels in AKI may indicate compensatory mechanisms that try to keep calcium in balance [6]. Reduced 25(OH)D concentrations have been linked in previous studies to immune dysfunction and poorer outcomes in hospitalized patients [14]. Although we did not assess mortality or recovery in this study, our findings raise the possibility that early mineral profiling could help identify subgroups of patients at different biological risk.

Some limitations in our study need to be recognized. The researchstudy was conducted at a single tertiary center, with a relatively small sample size. Therefore, the results might not completely reflect other patient populations or healthcare settings.

The cross-sectional design is another important limitation. All measurements were obtained within 24 hours of AKI diagnosis, which allowed us to focus on very early changes. However, this approach does not show how mineral parameters evolve during recovery or progression. We cannot determine whether the identified patterns are transient responses or persistent disturbances. Longitudinal assessment would be necessary to clarify these dynamics.

In addition, total serum calcium was used rather than ionized calcium and fibroblast growth factor 23 was not measured. Because vitamin D deficiency is very common in many populations, including ours, it is hard to say if lower 25(OH)D levels are showing recent changes or an old deficiency.

Finally, while clustering analysis revealed distinct biochemical profiles, the clinical implications of these profiles remain uncertain. We did not examine associations with mortality, renal recovery, or hospital stay, and thus, the prognostic value of the identified mineral-axis patterns cannot be established from this dataset.

Despite these limitations, our findings showed that early disturbances in mineral metabolism are consistently present in AKI and that biochemical heterogeneity exists beyond traditional creatinine-based staging. Future prospective studies with longitudinal follow-up are needed to determine if mineral-axis phenotypes have any association with recovery, progression, or mortality and if they may have therapeutic implications.

Conclusions. AKI is accompanied by early disturbances in mineral metabolism that are not proportional to creatinine-based staging. Distinct mineral-axis profiles can be identified within the first 24 hours of diagnosis, indicating biochemical heterogeneity beyond traditional severity classification. Larger longitudinal studies are warranted to clarify their clinical relevance.

Conflict of interest. The authors declare no competing interests.

Findings. The authors received no financial support for the research.

Authors' contributions. All authors contributed equally to the conceptualization, data curation, investigation, methodology, project administration, resources, visualization, software, writing of the original draft, and writing – review & editing of the manuscript. All authors read and approved the final version of the manuscript.

References:

1. *Ostermann M, Lumlertgul N, Jeong R, See E, Joannidis M, James M.* Acute kidney injury. The Lancet. 2025;405:241-56. doi: 10.1016/S0140-6736(24)02385-7.
2. *Esposito P, Cappadona F, Prenna S, Marengo M, Fiorentino M, Fabbrini P, et al.* Acute kidney injury in hospitalized patients with real-life analysis of incidence and clinical impact in Italian hospitals (the SIN-AKI study). Sci Rep. 2025;15:14261. doi: 10.1038/s41598-025-96236-8.
3. *Khwaja A.* KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract. 2012;120(4):c179-84. doi: 10.1159/000339789.
4. *Al-Tameemi HK, Ibrahim N, Abdullah DAA, Oddah NN.* Assessment of parathyroid hormone, vitamin D3, calcium, phosphate, and RFT in patients with chronic renal failure: A three-year follow-up study. Polski Merkuriusz Lekarski. 2025;53:40-6. doi: 10.36740/Merkur202501106.
5. *Yu J, Li Y, Zhu B, Shen J, Miao L.* Vitamin D: an important treatment for secondary hyperparathyroidism in chronic kidney disease? International Urology and Nephrology. 2024;57:1853-63. doi: 10.1007/s11255-024-04334-9.
6. *Singh NP, Panwar V, Aggarwal NP, Chhabra SK, Gupta AK, Ganguli A.* Regulation of Calcium Homeostasis in Acute Kidney Injury: A Prospective Observational Study. Indian J Crit Care Med. 2022;26(3):302-306. doi: 10.5005/jp-journals-10071-24124.
7. *Leaf DE, Christov M.* Dysregulated Mineral Metabolism in AKI. Semin Nephrol. 2019;39:41-56. doi: 10.1016/j.semnephrol.2018.10.004.
8. *Liu W, Shi T, Xu H, Zhao H, Hao J, Kong G.* Identifying acute kidney injury subtypes based on serum electrolyte data in ICU via K-medoids clustering. AMIA Annu Symp Proc. [Internet]. 2025;2024:733 -737. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC12099402/.
9. *Lim C, Tan HK, Kaushik M.* Hypophosphatemia in critically ill patients with acute kidney injury treated with hemodialysis is associated with adverse events. Clin Kidney J. 2017;10(3):341-347. doi: 10.1093/ckj/sfw120.
10. *Jetanapirom R, Boonsrirat U, Geater SL, Leelawattana R, Phongphithakchai A.* Impact of Calcium Phosphate Product on Acute Kidney Injury and Mortality: A Retrospective Cohort Study. Cureus. 2024;16(7):e64861. doi: 10.7759/cureus.64861.
11. *Girling BJ, Channon SW, Haines RW, Prowle JR.* Acute kidney injury and adverse outcomes of critical illness: correlation or causation? Clin Kidney J. 2019;13(2):133-141. doi: 10.1093/ckj/sfz158.
12. *Fathi M, Markazi Moghaddam N, Markazi Moghadam H, Fathi M, Hajiesmaeili M, Nooraei N, et al.* Data-driven phenotypes across the full AKI severity spectrum in patients admitted to the ICU. Nefrologia. 2025;:501469. doi: 10.1016/j.nefro.2025.501469.
13. *Hegde A, Denburg MR, Glenn DA.* Acute Kidney Injury and Pediatric Bone Health. Front Pediatr. 2021;8:635628. doi: 10.3389/fped.2020.635628.
14. *Lai L, Qian J, Yang Y, Xie Q, You H, Zhou 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. doi: 10.1371/journal.pone.0064964.