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Review

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Stratification of Acute Kidney Injury Risk, Disease Severity, and Outcomes by Electrolyte Disturbances

Stefan Erfurt[a], Rebecca Lehmann[a], Igor Matyukhin[a], Benedikt Marahrens[a], Susann Patschan[a], Daniel Patschan[a, b]

Abstract

Acute kidney injury (AKI) affects up to 30% of all hospitalized patients in Central Europe and the USA. New biomarker molecules have been identified in recent years; most studies performed so far however aimed to identify markers for diagnostic purposes. Serum electrolytes such as sodium and potassium are quantified in more or less all hospitalized patients. Aim of the article is to review the literature on the AKI predictive role of four distinct serum electrolytes in evolving/progressing AKI. The following databases were searched for references: PubMed, Web of Science, Cochrane Library, and Scopus. The period lasted from 2010 until 2022. The following terms were utilized: "AKI" AND "sodium" OR "potassium" OR "calcium" OR "phosphate" AND "risk" OR "dialysis" OR "recovery of kidney function" OR "renal recovery" OR "kidney recovery" OR "outcome". Finally, 17 references were selected. The included studies were mostly retrospective in nature. Particularly, hyponatremia has been shown to be associated with an overall poor clinical outcome. The association between dysnatremia and AKI is anything but consistent. Hyperkalemia and potassium variability are most likely AKI predictive. Serum calcium and AKI risk are associated in a U-shaped manner. Higher phosphate levels potentially predict AKI in non-coronavirus disease 2019 (COVID-19) patients. The literature suggests that admission electrolytes can offer valuable information about AKI onset during follow-up. Limited data are however available on follow-up characteristics such as the need for dialysis or the chance of renal recovery. These aspects are of particular interest from the nephrologist's perspective.

Keywords: AKI risk; Outcome; Disease severity; Mortality; Electrolytes

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[a]Department of Internal Medicine I - Cardiology, Nephrology and Internal Intensive Medicine, Brandenburg University Hospital, Brandenburg Medical School (Theodor Fontane), Brandenburg an der Havel, Germany
[b]Corresponding Author: Daniel Patschan, Department of Internal Medicine I - Cardiology, Nephrology and Internal Intensive Medicine, Brandenburg University Hospital, Brandenburg Medical School (Theodor Fontane), Brandenburg an der Havel, Germany. Email: d.patschan@klinikum-brandenburg.de

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Introduction

Acute kidney injury (AKI) affects up to 30% of all hospitalized patients in Central Europe and the USA [1]. Mortality rates are high, up to 25% of all affected individuals die in the short-term (during the hospital stay) [2, 3]. Under intensive care conditions however, mortality may reach or even surpass 50% [1]. A particularly poor prognosis has been reported in patients with non-solid tumors, sepsis and AKI combined. Under these circumstances, *de facto* 100% of all subjects die during in-hospital treatment [4]. Surviving individuals are at higher risk for chronic kidney disease (CKD) in the long term [5]. Any individual AKI episode increases the CKD risk, more severe AKI episodes put patients at higher risk, respectively [6]. In some individuals, persistent AKI, termed as "acute kidney disease" (AKD) if excretory dysfunction is impaired for longer than 7 days after an acute event [7], may transition to CKD in a continuous manner. The diagnosis of AKI is still made according to the 2012 revised "KDIGO clinical practice guidelines for acute kidney injury". Presumably, future definition criteria of the syndrome will also incorporate so-called damage biomarkers [5] for both, a more sophisticated diagnosis and staging.

In recent years, several new AKI biomarkers have been identified [8]. Most studies performed so far aimed to identify markers for diagnostic purposes. Basically, so-called damage markers must be distinguished from markers of impaired kidney function and from stress markers [9]. The second group is represented by cystatin C and proenkephalin A. Member of the third group for instance is the protein dickkopf-3. The damage biomarker group however encompasses numerous molecules including well-known proteins such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), liver-fatty acid binding protein (L-FABP), and others. In 2020, Ostermann et al [9] published "Recommendations on Acute Kidney Injury Biomarkers From the Acute Disease Quality Initiative Consensus Conference". In this manuscript, the clinical usability of stress/damage/functional biomarkers was assessed in relation to five distinct outcome categories: risk assessment, AKI prediction, AKI diagnosis, AKI severity, and kidney recovery. From the nephrologist's perspective however, two aspects are of particular interest: which AKI subjects will most likely require kidney replacement therapy (KRT) and which patients are at higher risk of death? The endpoint death is not only restricted to individual hospital treat-

ment periods but also to longer periods after discharge (e.g., 90 days, 1 year).

Serum creatinine is quantified in hospitals on a regular basis, despite the molecule is far from being an optimal biomarker: it increases only if the glomerular filtration rate is diminished by 50% or more, it has no prognostic value at all and allows no discrimination between different AKI entities [10]. Newer biomarker candidates however have not been established for routine diagnostics in many hospitals yet. Serum electrolytes however, namely sodium and potassium, are measured in the vast majority of all patients that receive in-hospital treatment. In most cases, electrolytes are measured repeatedly during an individual hospital stay, the costs are neglectable. AKI is potentially accompanied by various electrolyte disturbances during the course of the disease. Particularly hyperkalemia puts patients at higher risk of death [11, 12]. Nevertheless, there are also several studies on the predictive value of electrolyte disorders before AKI has been evolved/progressed to higher stages (e.g., Acute Kidney Injury Network (AKIN) stage 1 to 3 [13]). Thus, electrolyte disorders are potentially useful for the prediction of outcome variables such as the need of KRT and in-hospital death, beside their diagnostic value in established AKI.

Aim of the article is therefore to review the literature on the predictive role of four distinct serum electrolytes in evolving/progressing AKI: sodium, potassium, calcium, and phosphate. We will omit to review physiological functions and metabolism, respectively. Also, we do not intend to discuss data on blood gas variables such as pH or actual bicarbonate in AKI risk prediction in this article, despite blood gas variables potentially provide prognostic information in AKI as well [14, 15].

With few exceptions, the studies will be presented according to the date of publishing (older to more recent).

Methods

The article is a systematic review. Four distinct databases were searched for references: PubMed, Web of Science, Cochrane Library, and Scopus. The period lasted from 2010 until 2022. The following terms were utilized: 'AKI' AND "sodium" OR "potassium" OR "calcium" OR "phosphate" AND "risk" OR "dialysis" OR "recovery of kidney function" OR "renal recovery" OR "kidney recovery" OR "outcome". Additional search terms were "renin" OR "angiotensin" AND "AKI" OR "acute kidney injury" OR "acute renal failure" with and without "pathophysiology". The final selection only contained those references that provided data on AKI recognition and risk prediction. The following risk categories were defined: AKI onset, in-hospital death and the need of dialysis (KRT), and recovery of kidney function. Not included were those studies that reported outcome variables in patients with established AKI and subsequent electrolyte disorders due to AKI.

Serum sodium

It must be prepended that both, serum sodium and potassium levels (and to some extent serum calcium levels also) change in response to the intake of diuretics and/or angiotensin-converting enzyme (ACE)/angiotensin II type 2 (AT2) inhibitors. This aspect always needs to be considered if electrolyte disturbances are diagnosed prior to AKI. A distinct electrolyte disorder may reflect drug-induced effects (e.g., potassium and volume depletion), ultimately responsible for AKI. The same applies for AKI-associated alterations in the renin-angiotensin axis [16], which modify electrolyte serum levels also.

The first study was published in 2015 [17]. Designed as retrospective investigation, it included 152 individuals, admitted to a single intensive care unit (ICU). The principal aim was to analyze AKI incidence and outcome variables. The baseline serum creatinine (measures at ICU admission) was significantly higher in subjects that acquired AKI during follow-up (1.68 (1.06 - 2.93) vs. 0.82 (0.63 - 0.99) mg/dL, $P < 0.001$). With regard to electrolyte disturbances, only admission hypernatremia was shown to be independently predictive for in-hospital mortality. There was however no association between any type of dysnatremia and the AKI incidence during the ICU stay. AKI naturally occurred in a timely variable manner during follow-up. Also, dysnatremia was not associated with the need for dialysis therapy.

Lee at al [18] retrospectively evaluated more than 19,000 patients that required in-hospital treatment during a 1-year period. Hyponatremia, defined as serum sodium below 135 mmol/L was observed in 8.2% at the time of admission. Out of all subjects, 5.1% fulfilled the diagnostic criteria of AKI. Initial hyponatremia was independently predictive for both, AKI onset and in-hospital mortality ($P = 0.004$ and $P = 0.002$).

In 2019, Gao et al [19] analyzed more than 13,000 ICU treated AKI patients, data were extracted from the "Multiparameter Intelligent Monitoring in Intensive Care Database III". The authors were interested in AKI risk prediction by serum sodium and potassium levels at the time of admission. Subjects with hypo- and hypernatremia as compared to the so-called "middle category of admission serum sodium" (136.0 - 144.9 mmol/L) were at significantly higher risk of death. Also, hyponatremic individuals with simultaneous hyperkalemia showed a higher 90-day mortality. Nevertheless, admission dysnatremia did not predict the need for dialysis therapy.

Patients with insomnia were analyzed by Bae et al [20]. The term insomnia has been well defined: it must be suspected if at least one "nocturnal sleep symptom" and a daytime or "waking symptom" occur, both attributable to impaired sleep at night [21]. The diagnosis insomnia according to these criteria is associated with a higher degree of morbidity in affected individuals: the quality of life is reduced; the cognitive function is impaired. Also, patients suffer more often from arterial hypertension [22] and coronary artery disease [23]. The cited study by Bae et al evaluated whether hyponatremia in insomnia patients possibly predicts AKI. Subjects were assigned to one out of three groups, according to the serum sodium level (< 138 mmol/L, 138 - 140.9 mmol/L, > 141 mmol/L). After 49.4 months (median), 62 out of 412 patients had developed AKI. The lowest sodium tertile was associated with all-cause mortality but not with AKI.

In 2020, Woitok et al [24] published a cross-sectional study which analyzed data from a 2-year period (January 2017

until December 2018). Subjects admitted to the emergency department were screened for AKI risk factors, particularly for dysnatremias and subsequent outcome variables. AKI was diagnosed in 8% of the patients, the prevalences of hypo- and hypernatremia were 23.16% and 1.4%, respectively. Both electrolyte disturbances were identified as independent mortality predictors.

A 2021 published, retrospective investigation [25] included septic patients, admitted to the ICU of the "Beijing Friendship Hospital" (January 2009 until December 2014). More than 590 individuals participated, who were assigned to one out of three groups, depending on the serum sodium level (normal/reduced/elevated). The study revealed hyponatremia as AKI risk predictor, hypernatremia was death-predictive in subjects with established AKI.

Basalely et al [26] published a secondary analysis of 1,979 neonates from the AWAKEN study cohort. The AWAKEN study focused neonatal AKI [27]. Included children were in the first postnatal week of age. Dysnatremia was not associated with AKI onset but both, hypo- and hypernatremia predicted mortality. Overall, more than 50% of the children presented dysnatremia.

A last year published investigation of coronavirus disease 2019 (COVID-19)-infected patients was published by Tzoulis et al [28]. Overall, 488 subjects were included, 24.3% were diagnosed with hyponatremia, 5.3% were hypernatremia (at admission, respectively). Hypernatremia was predictive for in-hospital death, while hyponatremia was not. Also, both electrolyte disturbances were not identified as risk factors for AKI onset or length of in-hospital stay.

One additional study needs to be mentioned, although it did not evaluate the predictive role of electrolyte disturbances in AKI [29]. It particularly aimed to test, whether hyponatremia is associated with clinical outcome parameters independently from the excretory kidney function. Both AKI and CKD are potent mortality risk factors but also induce hyponatremia through certain mechanisms, namely the retention of free water. It was hypothesized that excretory kidney dysfunction with ensuing hyponatremia rather than hyponatremia *per se* worsens the prognosis of affected individuals. The study included all patients that were hospitalized between January 2009 and December 2011 (database study). CKD and AKI were defined according to respective estimated glomerular filtration rate (eGFR) values and dynamics. Cox regression analysis was performed in order to compare mortalities between subjects with versus without community-acquired hyponatremia (hyponatremia at hospital admission). In subjects with suspected CKD and AKI, the following parameters were associated with mortality: hyponatremia, age, race, illness severity and the Charlson score [30]. The most important finding was related to hyponatremia in different kidney disease stages: the respective hazard ratios (HRs) were comparable.

Serum potassium

Hyperkalemia is a well-known complication in AKI, it should be recognized instantly in order to prevent respective patients from severe cardiac arrhythmias. The data on AKI risk and outcomes in relation to initial dyskalemia are quite limited in contrast.

The study by Gao et al [19] has already been discussed (serum sodium). It showed a higher 90-day mortality in hyponatremic AKI individuals with simultaneous hyperkalemia. Admission serum potassium was not predictive with regard to the need of dialysis during follow-up.

A 2021 published, retrospective, cross-sectional evaluation [31] included all patients admitted to the emergency department of a single Swiss hospital (January 2017 until December 2018). In total, 8% of the subjects were diagnosed with AKI, hyperkalemia occurred in 13%, hypokalemia in contrast was diagnosed in 11%. Both electrolyte disturbances were associated with prolonged in-hospital treatment periods and in-hospital death.

In 2022, Lombardi et al [32] published a retrospective, observational trial (cohort study), that included in-hospital treated patients (January 2010 until December 2014, Fondazione Policlinico Universitario A. Gemelli IRCCS). The study design was interesting in so far, that all patients finally included in the analysis had not developed AKI during an initial period of 10 days. Serum potassium had to be measured at least twice, serum creatinine at least three times during that particular period. Primary endpoint was AKI onset. Finally, 555 out of 18,836 patients (2.9%) fulfilled the diagnostic AKI criteria. Higher variability of serum potassium was independently associated with AKI onset; also, subjects with hyperkalemia developed AKI more often during follow-up. Thus, the study, although performed in a retrospective manner, helped to identify serum potassium variability and hyperkalemia as AKI risk factors.

Serum calcium

Studies on the AKI predictive role of both, serum calcium and phosphate were particularly performed by Thongprayoon et al [33-35].

In 2018, the authors reported data from a single-center, retrospective evaluation [34]. In-hospital patients treated between 2009 and 2013 were included if they serum calcium analysis had been performed at the time of hospital admission. Six groups were defined, depending on the total calcium range. Hospital-acquired AKI was the primary endpoint. Finally, almost 13,000 individuals were included, the AKI incidence was identified with 13.9%. Regression analysis led to the identification of four ranges of serum calcium to be associated with an increased AKI risk ($\leq$ 7.9, 9.0 - 9.4, 9.5 - 9.9 and $\geq$ 10 mg/dL).

Two years later, the same group published data collected during the same period (2009 - 2013) [35]. This time, the study group was even larger (n = 25,844). In contrast to the 2018 published investigation, the authors divided the six categories according to the ionized instead of the total serum calcium. The ranges were $\leq$4.39, 4.40 - 4.59, 4.60 - 4.79, 4.80 - 4.99, 5.00 - 5.19, and $\geq$ 5.20 mg/dL, respectively. The AKI incidence was 12.7%. The essential finding was a U-shaped association between serum calcium and the in-hospital onset of AKI. Particularly the following ranges of ionized calcium were AKI risk predictive: 4.40 - 4.59, $\leq$ 4.39 mg/dL, and $\geq$ 5.20 mg/dL.

Serum phosphate

Thongprayoon et al [33] included more than 5,000 patients between January and December 2013. Similar to the studies published on serum calcium and AKI risk by the same leading author [34, 35], six groups were defined, depending on the serum phosphate range (2.4, 2.4 - 2.9, 2.9 - 3.4, 3.4 - 3.9, 3.9 - 4.4, and ≥ 4.4 mg/dL). An admission serum phosphate of > 4.4 mg/dL was associated with an increase AKI risk, whereas levels below 4.4 mg/dL were not.

Moon et al [36] retrospectively reviewed 20,686 adult patients, treated at the Seoul National University Bundang Hospital during a 1-year period. Subjects were assigned to one out of four groups, depending on the serum phosphate level at admission, respectively. AKI incidence was identified with 11.2%. The AKI odds ratios were higher in the third and fourth quartile as compared to the first quartile.

A prospective investigation (observational, single-center) on the predictive role of phosphate in post-cardiac surgery AKI was published in 2020 [37]. Serum phosphate increased in AKI, reaching a peak at 48 h. A phosphate decrease of at least 25% after 24 h was associated with recovery of kidney function.

A 2021 published [38], retrospective investigation enrolled 823 COVID-19 patients. All individuals underwent analysis of kidney excretory function at least twice during the hospital stay. All subjects were recruited from one out of four hospitals in Wuhan (China). The AKI incidence was high in this respective cohort (40.9%). By using Cox regression analysis, a total number of eight independent AKI risk factors were identified, including hypophosphatemia, which was diagnosed at the time of hospital admission. Affected individuals accumulated several common characteristics such as higher age, lower serum albumin, lower serum uric acid and others. Particularly, renal phosphate excretion was inversely correlated with hypophosphatemia.

Patients with tumor lysis syndrome were analyzed in a recent study [39]. In total, 120 individuals with a cumulative number of 130 episodes of the syndrome were included (retrospective design, observational period 11 years). Multivariate analysis revealed three factors to be associated with AKI onset: exposure to platinum-containing agents, elevated lactate dehydrogenase (LDH) activity, and hyperphosphatemia before AKI.

Further studies evaluated the prognostic role of hypophosphatemia during renal replacement therapy in established AKI [40-42]. However, in all cited investigations AKI had already been evolved.

Table 1 summarizes all studies discussed in the text [17-20, 24-26, 28, 31-39].

Discussion

The most important limitation of studies published so far is the lack of prospective data. Only one out of 17 selected investigations [37] was designed in a prospective manner. The problem with such an approach is often the lack of control groups and/or the lack of comparisons with other, already established AKI biomarkers. For instance, the following biomarkers have been shown to predict the mortality risk in AKI: NGAL, KIM-1 (both [43]), the fibrinogen-to-albumin ratio (FAR) [44], soluble suppression of tumorigenicity 2 (sST2) (circulating interleukin (IL)-33 receptor isoform [45]), and certain panels of metabolites [46]. A 2020 published study by Fiorentino [47] on the other hand identified urinary (TIMP-2) • (IGFBP7) (tissue metalloproteinase-2 and insulin-like growth factor-binding protein-7) of > 0.3 as dialysis predictive in septic AKI individuals. The current literature on the role of electrolytes in AKI risk prediction (regarding the endpoints AKI onset, death, KRT, and recovery of kidney function) shows several associations, partly in an independent manner: dysnatremia and AKI onset (n = 2), dyskalemia and AKI onset (n = 1), dyscalcemia and AKI onset (n = 2), dysphosphatemia and AKI onset (n = 4), dysnatremia and death (n = 9 references), dyskalemia and death (n = 2), phosphate decrease and recovery of kidney function (n = 1). No associations at all were shown with regard to the need of KRT. If only the data on serum sodium and AKI onset are considered, the findings are everything but consistent. Two studies revealed a role of hyponatremia in AKI onset prediction (18,25), four studies did not confirm the observations [17, 20, 26, 28]. The article by Peres at al [17] included ICU treated individuals (n = 152), recruited from a single ICU, dysnatremia was not AKI predictive. In contrast, Lee et al [18] identified hyponatremia as an independent risk factor of AKI onset, over 19,000 individuals were included in the analysis which was not restricted to intensive care receiving patients. This discrepancy indicates two limitations of the data available so far: group sizes differ significantly (150 vs. > 19,000), patient recruitment is not comparable at all. Nevertheless, electrolytes fulfill at least two important requirements of a so-called "optimal biomarker": the methodologies used for electrolyte quantification have reliably been established in most hospitals worldwide, the results are comparable, and the respective costs are neglectable. However, prospective and controlled data are needed. Future studies should therefore consider the following requirements: 1) prospective design; 2) inclusion of established biomarkers for AKI risk prediction (e.g., NGAL, KIM-1); and 3) the following endpoint categories: AKI onset, need of KRT, in-hospital death, and recovery of kidney function.

Conclusions and Perspective

In the past, prospective trials have been conducted only sporadically. Thus, reliable conclusions are possible in a limited fashion only. Most studies published so far showed an association between dysnatremia (particularly hyponatremia) and an overall poor clinical outcome. The association between dysnatremia and AKI onset however is anything but consistent. Both hyperkalemia and potassium variability are most likely AKI predictive. Serum calcium and AKI risk are apparently associated in a U-shaped manner. Higher phosphate levels potentially predict AKI in non-COVID-19 patients, opposing findings have however been reported from patients under dialysis therapy of established AKI. Limited data are available on ad-

Table 1. Summary of All Studies Discussed in the Main Text

Reference/year	Design	Outcome				Summary
		AKI onset	Death	Dialysis	Recovery of kidney function	
Serum sodium						
Peres et al, 2015 [17]	Retrospective, single ICU, 152 AKI patients	No association with dysnatremia	Hypernatremia independently predictive	No association with dysnatremia	-	Admission dysnatremia has limited value with regard to AKI-related outcome variables at the ICU
Lee et al, 2016 [18]	Retrospective, 1-year period, > 19,000 hospitalized patients	Hyponatremia independently predictive	Hyponatremia independently predictive	-	-	Hospital admission hyponatremia suitable for AKI prediction
Gao et al, 2019 [19]	Retrospective, > 13,000 ICU-treated AKI patients	-	Hyper- and hyponatremia predictive	Dysnatremia not predictive	-	ICU admission dysnatremia has limited value in dialysis risk prediction of AKI subjects
Bae at al, 2020 [20]	Retrospective, 2-year period, 412 subjects with insomnia	Hyponatremia not predictive	Hyponatremia associated with all-cause mortality	-	-	Hyponatremia not AKI predictive over 24 months
Woitok et al, 2020 [24]	Retrospective, cross-sectional, 2-year period, > 1,700 subjects, ED	-	Hyper- and hyponatremia predictive in AKI subjects	-	-	ED admission dysnatremia mortality predictive in established AKI
Zhi et al, 2021 [25]	Retrospective, 6-year period, 590 ICU-treated patients	Hyponatremia predictive	Hypernatremia death predictive in AKI	-	-	Admission hyponatremia potentially useful in AKI risk prediction under ICU conditions
Basalely et al, 2021 [26]	Secondary analysis of 1,979 neonates included in the AWAKEN trial [28]	Dysnatremia not predictive	Hyper- and hyponatremia predicted mortality	-	-	Dysnatremia not suitable for no AKI prediction in neonates
Tzoulis et al, 2021 [28]	Retrospective, longitudinal cohort study, 488 patients, 8-week period, COVID-19 patients	Dysnatremia not predictive	Hypernatremia predictive for in-hospital death	-	-	Admission dysnatremia not suitable for AKI prediction
Serum potassium						
Gao et al, 2019 [19] - see above	Retrospective, > 13,000 ICU-treated AKI patients	-	Higher 90-day mortality in hyponatremic AKI individuals with simultaneous hyperkalemia	Admission serum potassium not predictive	-	Additional potassium analysis in hyponatremic AKI individuals helpful in mortality prediction
Ravioli et al, 2021 [31]	Retrospective, cross-sectional, 2-year period, > 1,700 patients, ED, AKI subjects	-	Dyskalemia predictive in established AKI	-	-	NA

Table 1. Summary of All Studies Discussed in the Main Text - *(continued)*

Reference/year	Design	Outcome				Summary
		AKI onset	Death	Dialysis	Recovery of kidney function	
Lombardi et al, 2022 [32]	Retrospective, cohort study, 5-year period, no AKI from day 1 to 10 of the hospital stay, 21,830 hospital admissions	Hyperkalemia and potassium variability associated with AKI onset	-	-	-	Increased serum potassium useful in AKI prediction several days in advance
Serum calcium						
Thongprayoon et al, 2018 [34]	Retrospective, 5-year period, 25,844 hospitalized patients, six groups defined, depending on the total serum calcium level	4 out of 6 distinct calcium ranges AKI predictive	-	-	-	Total calcium useful with regard to the prediction of AKI onset
Thongprayoon et al, 2020 [35]	Retrospective, 5-year period, 12,784 patients, six groups defined, depending on the serum level of ionized calcium	U-shaped association between ionized calcium and AKI onset	-	-	-	Ionized calcium useful with regard to the prediction of AKI onset
Serum phosphate						
Moon et al, 2019 [36]	Retrospective, 1-year period, 20,686 patients, four groups defined, depending on the serum phosphate level	Higher AKI odds ratios in quartiles 3 and 4 as compared to quartile 1	-	-	-	Certain serum phosphate ranges are associated with higher AKI risk
Saour et al, 2020 [37]	Prospective, observational, single center, 260 individuals, post-cardiac surgery AKI	-	-	-	Phosphate decreases of 25% or more after 24 h predictive	Limited value with regard to AKI-related outcome variables
Thongprayoon et al, 2018 [33]	Retrospective, 5-year period, 5,036 patients, six groups defined, depending on the total serum phosphate level	Serum phosphate > 4.4 mg/dL associated with increased AKI risk	-	-	-	Severe hyperphosphatemia predicts AKI onset
Chen et al, 2021 [38]	Retrospective investigation in 823 COVID-19 patients	Hypophosphatemia predictive	-	-	-	Hyperphosphatemia independently AKI predictive
Lemerle et al, 2022 [39]	Retrospective, 11-year period, 120 patients, tumor lysis syndrome	Hyperphosphatemia predictive	-	-	-	Hyperphosphatemia AKI predictive

The outcome variables of interest are AKI onset during in-hospital treatment, in-hospital death, the need for dialysis due to AKI and recovery of kidney function until demission. Fields remain empty if no particular information have been provided. AKI: acute kidney injury; ICU: intensive care unit; COVID-19: coronavirus disease 2019; ED: emergency department; NA: not available.

mission electrolytes and follow-up characteristics such as the need for dialysis or the chance of renal recovery. These aspects are of particular interest from the nephrologist's perspective.

In summary, the literature suggests that admission electrolytes can offer valuable information about AKI onset during follow-up. Future studies should therefore be performed, but they must be designed prospectively and should regularly include follow-up characteristics also (dialysis/recovery of kidney function). The advantage of electrolytes is their general availability.

Learning points

Admission electrolytes can offer valuable information about AKI onset during follow-up.

Prospective trials have nevertheless been conducted only sporadically.

Future studies should therefore be performed, but they must be designed prospectively and should regularly include follow-up characteristics also (dialysis/recovery of kidney function). The advantage of electrolytes is their general availability.

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Conflict of Interest

The authors declare that they do not have any conflict of interest.

Author Contributions

SE and DP wrote the article. RL searched for references. IM searched for references and designed the table. BM and SP searched for references and assisted in writing. DP designed and wrote the article. All authors finally approved the final version of the manuscript.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

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