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Marine Saour, Norddine Zeroual, Jérôme Ridolfo, Erika Nogué, Marie-Christine Picot, et al.. Serum Phosphate Kinetics in Acute Kidney Injury After Cardiac Surgery: An Observational Study. Journal of Cardiothoracic and Vascular Anesthesia, inPress, 10.1053/j.jvca.2020.05.023 . hal-02908030

HAL Id: hal-02908030

<https://hal.science/hal-02908030>

Submitted on 29 Jul 2020

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Serum Phosphate Kinetics in Acute Kidney Injury After Cardiac Surgery: An Observational Study

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Objective: Acute kidney injury (AKI) is a common complication after cardiac surgery and may affect prognosis. Serum phosphate (SPh) elevation is well-known to occur after AKI but not well-documented. The aim of the present study was to describe SPh changes during AKI after cardiac surgery and to assess the accuracy for the diagnosis of AKI severity and recovery.

Design: Prospective, single center, observational study.

Setting: Intensive care unit of a tertiary university hospital.

Participants: All patients admitted consecutively to the intensive care unit between February 2015 and March 2016.

Measurements and Main Results: AKI was defined according to Kidney Disease Improving Global Outcomes criteria and classified as nonsevere (stage 1) and severe (stages 2 and 3). Receiver operating characteristic curve analysis was conducted to test reliability of SPh for AKI severity and recovery. AKI occurred in 86 of the 260 patients included (33%) in the study; 58 (67%) experienced nonsevere AKI, and 28 (33%) experienced severe AKI. A significant elevation of SPh values was observed in AKI patients, which peaked at 48 hours. At this time, an SPh of 1.33 mmol/L demonstrated a good accuracy for AKI severity, with an area under the curve of 0.91 (95% confidence interval 0.82-1.00). For kidney recovery, a 25% SPh decrease 24 hours after the peak had a positive predictive value of 100%, and a 2.5% decrease allowed for the reclassification of patients when the serum creatinine had not decreased enough.

Conclusions: The results showed that SPh changes closely follow AKI severity and kidney recovery after cardiac surgery. In addition to serum creatinine, this simple biological marker may help predict early favorable outcome.

Key Words: Acute kidney injury; Phosphate; Cardiac surgery

ACUTE kidney injury (AKI) is a common complication after cardiac surgery, affecting up to 20% to 40% of patients, with 3% requiring renal replacement therapy (RRT).¹⁻³ AKI is a major concern because postoperative

AKI jeopardizes short- and long-term prognosis, mostly when AKI is severe.³ In addition to predictive scores,^{4,5} various new biomarkers, such as neutrophil gelatinase-associated lipocalin have been tested to improve AKI prediction with good success, but they are not able to predict AKI recovery nor are they helpful for monitoring AKI evolution.⁶⁻¹¹ Monitoring AKI evolution is challenging because it may significantly influence patient management, including the decision for RRT and intensive care unit (ICU) length of stay.¹²

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In addition to renal filtration impairment, AKI is associated with a variety of biochemical abnormalities as a result of impairment of tubular function that leads to reabsorption/excretion failure of various ions and acid-base imbalance.^{13,14} Serum creatinine (SCr) elevation has been included in the criteria to define and classify AKI.¹⁵ However, SCr elevation reflects a delayed impairment of renal filtration and may not be an accurate marker of tubular dysfunction.^{14,16} Serum phosphate (SPh) is a common anion, and its regulation depends on kidney function.¹⁷ In chronic kidney disease (CKD), SPh increase has been observed at an even relatively modest reduction of renal function¹⁸ and is considered as a major prognosis factor of CKD aggravation, dialysis requirement, and mortality.¹⁸⁻²⁰

Postoperative management after cardiac surgery requires regular biologic control, including SPh, because hypophosphatemia may be associated with a complicated postoperative outcome.²¹ During routine clinical practice, the authors of the present study had observed that SPh changes during AKI followed renal function decline and recovery; however, to their knowledge, these variations have never been studied thoroughly.¹³ Because SPh depends on minute-to-minute regulation by the kidney, a close correlation between SPh changes and AKI evolution might be expected.

Therefore, the authors designed a prospective, observational study on SPh concentration after cardiac surgery to assess its changes during AKI. The second objective of the study was to assess whether SPh is reliable for assessing AKI severity and recovery.

Methods

Trial Design

The present clinical study was a prospective, observational, single-center study (NCT 0290031) and was approved by the Institutional Review Board (Comité de Protection des Personnes régional Sud-Méditerranée I [number 2015-A00781-48]). Because of the observational nature of the study, the need for patient informed consent was waived.

Participants

All adult patients (≥ 18 years old) admitted consecutively to the ICU after cardiac surgery with cardiopulmonary bypass (CPB) were included. Patients with the following characteristics were not included: (1) severe chronic renal disease (defined as glomerular filtration rate (GFR) ≤ 15 mL/min/1.73m²) or preoperative AKI; (2) kidney transplantation or nephrectomy or single kidney disease; (3) perioperative hemofiltration; (4) left ventricular assist device; or (5) surgical revision within the first 48 hours after surgery.

Study Settings

The study was conducted in a 14-bed cardiothoracic ICU at a university hospital.

Interventions

The usual cardiac treatment was maintained until the day before surgery. Perioperative management (anesthesia, CPB, and cardioplegia) was standardized for all the patients. CPB was instituted with a membrane oxygenator primed with 1.5 L of crystalloid (Isfundin; B. Braun, Melsungen, Germany), and body temperature was maintained at 34°C to 36°C. After aortic clamping, a hyperkalemic blood cardioplegic solution was infused into the aortic root of the aorta until myocardial arrest. A nonpulsatile pump flow rate was maintained between 2.2 and 2.6 L/min/m², provided that the SvO₂ monitored on the pump exceeded 60%. Crystalloid solution (balanced crystalloid solution) could be added to maintain pump flow, and blood products were used to maintain a hemoglobin level >7 g/dL during CPB and >8 g/dL after CPB. Hypotension (mean arterial pressure [MAP] ≤ 60 mmHg) was treated with the continuous intravenous administration of norepinephrine to maintain MAP >60 mmHg. After completion of the surgical procedure, patients were weaned from CPB when a rectal temperature of 36°C had been reached.

In the ICU, inotropic support (dobutamine) was used to achieve a cardiac index ≥ 2.2 L/min/m², as assessed by thermodilution (pulmonary catheter) or echocardiography (aortic flow measured by pulsed Doppler time-velocity integral), with normal left ventricular filling pressure. Hypotension (MAP ≤ 60 mmHg) without cardiogenic shock features (cardiac index >2.2 L/min/m²) was treated with continuous intravenous administration of norepinephrine to restore MAP to >60 mmHg, and repeated boluses of morphine were used to keep patients pain free. Weaning from the ventilator was started during the emergence of anesthesia, and extubation was performed when hemodynamics were stable and normothermia was maintained for ≥ 1 hour.

Kidney function assessment included SCr and SPh measurements the day before surgery (baseline value), at ICU admission, every 12 hours during ICU stay (every morning and evening), every morning up to postoperative day 5, and then at the discretion of the physician in charge of the patient as per the authors' routine practice. EuroSCORE II²² was used to assess the cardiac surgical risk, and the Simplified Acute Physiology Score²³ was used to assess the mortality risk in the ICU.

Study Outcomes

The primary outcome was to measure SPh changes during AKI after cardiac surgery. SPh was measured during the first 5 days after surgery to detect the peak value, which then was considered as the AKI reference value. AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, except that urine output was not used to classify patients into the 3 following KDIGO stages¹⁵: stage 1, SCr at 1.5- to 1.9 times baseline or an SCr increase ≥ 26.5 μ mol/L; stage 2, SCr at 2.0- to 2.9 times baseline; and stage 3, SCr at 3.0 times the baseline or an SCr increase ≥ 353.6 μ mol/L. A subgroup analysis also was performed in patients with preoperative CKD defined by an <60 mL/min/1.73 m².

The secondary outcomes included SPh accuracy for diagnosis of AKI severity and recovery. AKI severity was graded as nonsevere (stage 1) and severe AKI (stages 2 and 3). RRT was initiated according to guidelines, independent of SPh values. Because of the absence of a consensus definition, renal recovery was defined arbitrarily as an SCr equal to or less than the preoperative SCr + 26.5 $\mu\text{mol/L}$ at hospital discharge. Recovery was studied only in AKI patients without RRT.

Statistical Analysis

The sample size was estimated from an expected AKI incidence of 30% according to a study in which neutrophil gelatinase-associated lipocalin was used to diagnose AKI, with a sensitivity (Se) of 0.7 and false positive rate at 0.1.¹⁵ For a study power of 90% and a risk α at 5%, 88 patients with AKI and 176 patients without AKI were needed (total 264). Quantitative data are expressed as mean \pm standard deviation or median [Q25-Q75] and categorical variables as the number of patients and percentage.

Patient characteristics were described and were compared between groups using the chi-square or Fisher exact test for categorical variables and Student or Wilcoxon Mann-Whitney test for continuous variables. Holm's correction was used for multiple comparisons. Receiver operating characteristic (ROC) curves were used to determine the SPh and SCr cutoff of severe AKI (KDIGO stages 2 and 3) at their peak value after surgery. The area under the ROC curve (AUC) was calculated, along with its 95% confidence interval (CI). The cutoff was determined using

the Youden index to optimize both Se and specificity (Sp), and the performances associated were reported (Se, Sp, positive predictive value [PPV], and negative predictive value [NPV]). ROC analyses also were used to study the performance of the relative decreases of SCr and SPh after the peak (value at 24 h after the peak – peak value/peak value) to assess predictability of renal recovery. The AUCs were compared using a nonparametric approach. The statistical significance threshold was set at 5%. Statistical analysis was performed using SAS Enterprise Guide, Version 7.3 (SAS Institute, Cary, NC).

Results

Study Population and AKI Incidence

The study flowchart is shown in Fig 1. During the study period between February 2015 and March 2016, 260 patients were included. Most participants were male (73.5%). Preoperative CKD was found in 33.1% (n=86) of the population (Table 1). AKI occurred in 86 patients (33.1%)—58 (22.3%) in stage 1, 11 (4.2%) in stage 2, and 17 (6.6%) in stage 3—and of all the patients, 9 (3.5%) required RRT, which was started with a median delay of 1.6 days.^{1,2} AKI patients were sicker, had longer surgery times, and needed more postoperative support, which resulted in a longer ICU stay and greater mortality rates (see Table 1). AKI occurred early, in the first 24 hours after surgery for 65 patients (75.6%), especially for AKI stages 2 and 3 (Fig 2).

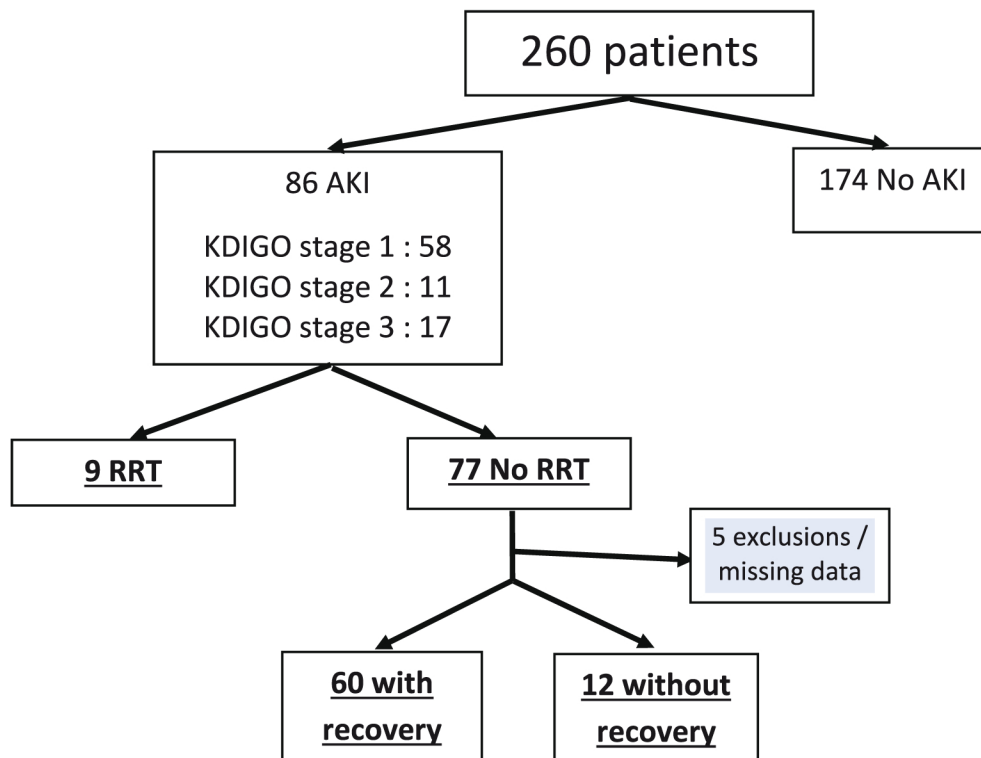


Fig 1. Study flowchart. AKI, acute kidney injury according to Kidney Disease Improving Global Outcomes (KDIGO) classification; RRT, renal replacement therapy.

Table 1
Perioperative Characteristics

	Total (n = 260)		AKI (n = 86)		No AKI (n = 174)		p Value
Age (y)	69	[59-76]	70	[62-77]	69	[59-76]	0.32 [*]
Male	191	(73.5%)	55	(64.0%)	136	(78.2%)	0.01 [†]
BMI	26.6	±4.7	26.6	±5.0	26.6	±4.5	0.97 [‡]
Comorbidities							
COPD	47	(18.1%)	23	(26.7%)	24	(13.8%)	0.01 [†]
Diabetes mellitus	65	(25.0%)	22	(25.6%)	43	(24.7%)	0.88 [†]
Hypertension	132	(50.8%)	46	(53.5%)	86	(49.4%)	0.54 [†]
LVEF <30%	14	(5.4%)	11	(12.8%)	3	(1.7%)	<0.01 [§]
Peripheral vascular disease	43	(16.5%)	17	(19.8%)	26	(14.9%)	0.32 [†]
Right-sided heart failure/pulmonary hypertension	8	(3.1%)	5	(5.8%)	3	(1.7%)	0.12 [§]
EuroSCORE II (%)	2.5	[1.4-5.6]	6.7	[3.1-12.6]	1.8	[1.1-3.6]	<0.01 [*]
Preoperative renal function							
GFR <60 mL/min/1.73 m ²	86	(33.1%)	42	(48.8%)	44	(25.3%)	<0.01 [†]
Serum creatinine (μmol/L)	91.5	[77-109]	95	[78-120]	88	[77-105]	0.01 [*]
Serum phosphate (mmol/L)	1.06	[0.93-1.18]	1.00	[0.91-1.15]	1.08	[0.94-1.19]	0.11 [*]
Surgical characteristics							
Surgery type							
Valve surgery	96	(36.9%)	33	(38.4%)	63	(36.2%)	0.73 [†]
CABG	122	(46.9%)	31	(36.0%)	91	(52.3%)	0.01 [†]
Combined surgery	82	(31.5%)	36	(41.9%)	46	(26.4%)	0.01 [†]
Emergency surgery	54	(20.8%)	29	(33.7%)	25	(14.4%)	<0.01 [†]
CPB (min)	107	[84-146.5]	145	[95-182]	98	[82-126]	<0.01 [*]
Aortic cross-clamping (min)	80	[62-107]	99	[72-136]	72	[59-98]	<0.01 [*]
Circulatory arrest (min) (n = 10)	46	±12	47	±13	42	±4	0.61 [‡]
Red blood cell transfusion	57	(21.9%)	34	(39.5%)	23	(13.2%)	<0.01 [†]
Vasopressors	121	(46.7%)	60	(69.8%)	61	(35.3%)	<0.01 [†]
Inotropes	38	(14.7%)	28	(32.6%)	10	(5.8%)	<0.01 [†]
Postoperative evolution							
Red blood cell transfusion	18	(6.9%)	15	(17.4%)	3	(1.7%)	<0.01 [†]
Vasopressors	63	(24.3%)	53	(61.6%)	10	(5.8%)	<0.01 [†]
Inotropes	25	(9.7%)	24	(27.9%)	1	(0.6%)	<0.01 [†]
Mechanical ventilation in ICU (h)	4	[3-6]	6	[4-23]	4	[3-5]	<0.01 [*]
ICU length of stay (d)	1	[1-2]	3	[1-6]	1	[1-1]	<0.01 [*]
Hospital length of stay (d)	9	[7-14]	14	[10-21]	8	[7-10]	<0.01 [*]
Mortality	8	(3.1%)	8	(9.3%)	0	(0.0%)	<0.01 [§]

NOTE. Quantitative data are expressed as mean ± standard deviation or median [Q25-Q75] and were compared with the Student *t* or Wilcoxon Mann–Whitney test, as appropriate. Categorical variables are expressed as the number of patients and percentage and were compared with the chi-square or Fisher exact test.

Abbreviations: BMI, body mass index; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; CPB, cardiopulmonary bypass; GFR, glomerular filtration rate; ICU, intensive care unit; LVEF, left ventricle ejection fraction.

* Compared with Wilcoxon Mann–Whitney test.

† Compared with chi-square test.

‡ Compared with Student *t* test.

§ Compared with Fisher exact test.

In patients with preoperative CKD, AKI occurred in 42 (49%) patients, with 28 (32.6%) in AKI stage 1, 8 (9.3%) in AKI stage 2, and 6 (7%) in AKI stage 3 (necessitated RRT).

Primary Outcome: SPh Increase During AKI

Preoperative SPh did not differ between AKI and no-AKI patients, whereas preoperative SCr was greater in AKI patients than in no-AKI patients (see Table 1). In AKI patients, SPh increased significantly from the preoperative value until the peak, which occurred at 48 hours (24-60) (H48 peak) and was significantly greater at each postoperative day compared with no-AKI patients (Fig 3). In CKD AKI patients, SPh and SCr changes at 48 hours were similar during AKI (Table 2).

Secondary Outcomes

SPh and AKI Severity

The H48 peak of SPh demonstrated an excellent accuracy for the diagnosis of KDIGO stages 2 or 3, with an ROC AUC of 0.91 (95% CI 0.83-1.00) and an Se of 88%, an Sp of 86%, a PPV of 45%, and an NPV of 98%, for a cutoff value of 1.33 mmol/L. In this series, the H48 peak of SCr had an ROC AUC of 0.97 (95% CI 0.92-1.00). The SCr and SPh ROC AUC tended to be different (*p* = 0.055) (Fig 4).

SPh and AKI Recovery

Among AKI patients without RRT (*n* = 72), renal recovery was observed in 60 patients (83%) and was more common in

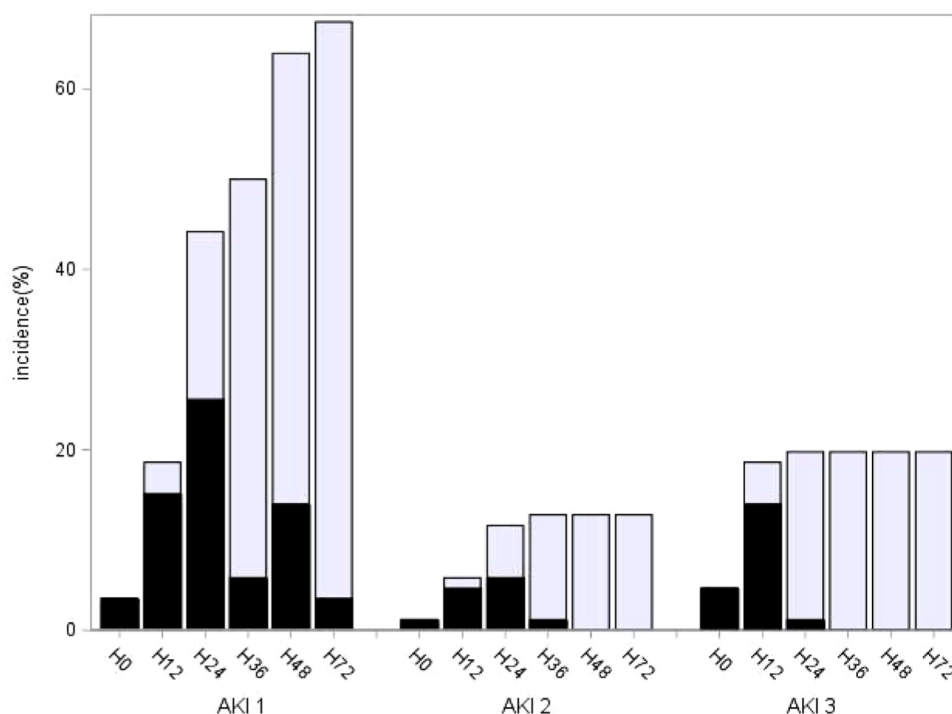


Fig 2. Acute kidney injury incidence over the first 3 postoperative days is represented by black bars and cumulative incidence by grey bars, grouped by acute kidney injury stages. Acute kidney injury, defined according to Kidney Disease Improving Global Outcomes stages, occurred early whatever its severity, with a maximal incidence at 24 hours for stages 1 and 2 and a maximal incidence at 12 hours for stage 3. AKI, acute kidney injury; H, hour.

nonsevere AKI (stage 1) than in severe AKI (stages 1 and 2)—49 (89%) patients versus 11 (65%) patients, respectively; $p=0.03$. The mortality rate was significantly greater in patients in whom renal function did not recover (33%) compared with other patients (5%) ($p=0.01$).

Evolution of SPh and SCr in AKI patients over 7 days showed significant differences between patients who experienced kidney recovery compared with patients whose kidneys did not recover (Fig 5).

In renal recovery patients, SPh and SCr decreased significantly 24 hours after the H48 peak (Table 3); the SPh decrease tended to be more substantial than the SCr decrease ($p=0.066$) (see Fig 5).

The relative decreases of SPh and SCr 24 hours after the H48 peak are reported in Table 3 along with the ROC curve analysis. Of note, a threshold of -25% SPh relative variation resulted in a 100% PPV value (Se 35.5%, Sp 100%, and NPV 35%).

Using the cutoff values of SPh and SCr decrease, the authors calculated a net reclassification improvement of 10 patients of 24 who experienced an SCr decrease lower than the SCr cutoff value (details on reclassification improvement study in the Supplemental Material).

Discussion

To the authors' knowledge, the present study was the first detailed description of SPh evolution during AKI in adult cardiac surgery patients. SPh increase at the H48 peak parallels AKI severity, and SPh early decrease after the peak may help to predict renal recovery. Indeed, the H48 peak SPh value

<1.33 mmol/L was associated with the absence of severe AKI, with an NPV of 98%. A 25% SPh decrease 1 day after the H48 peak demonstrated a PPV of 100% for renal recovery.

AKI remains one of the most common and serious clinical issues in the ICU and is associated with high morbidity and mortality.^{1,2} AKI in the context of cardiac surgery is specific because it may result from an acute but short injury related to surgery and CPB.²⁴ Identification of AKI at the early stage seems crucial for improving AKI management and AKI prognosis.^{3,25} Several investigations have been undertaken to identify biomarkers able to detect renal failure as early as possible.^{6-8,25} These biomarkers are promising for AKI diagnosis and severity but do not help to describe AKI evolution. SCr is used to diagnose and classify AKI according to KDIGO^{3,15}; however, SCr may not be a good biomarker of early AKI evolution.²⁶ SPh, which has a homeostasis strongly regulated by the kidneys, has not been studied thoroughly during AKI.¹³ In the present study, SPh showed paralleled evolution to SCr. It is noticeable that preoperative SCr was greater in AKI patients than in no-AKI patients, whereas SPh was in the same normal range in both groups. However, acute changes in SPh and SCr during AKI were similar in non-CKD patients and CKD patients. Therefore, SPh is neither an early marker of AKI nor a specific marker of AKI in CKD patients. Conversely, SPh showed good accuracy for AKI severity diagnosis, with ROC AUC values around 0.9, not much less than those for SCr (0.97), the gold standard of KDIGO definition. Similar findings on SPh correlation with disease severity have been observed in CKD patients. A significant increase in SPh has been independently associated with declines in renal function and dialysis requirement and cardiovascular events.^{20,27} Indeed, hyperphosphatemia, which develops inevitably at end-stage CKD, points out a significant

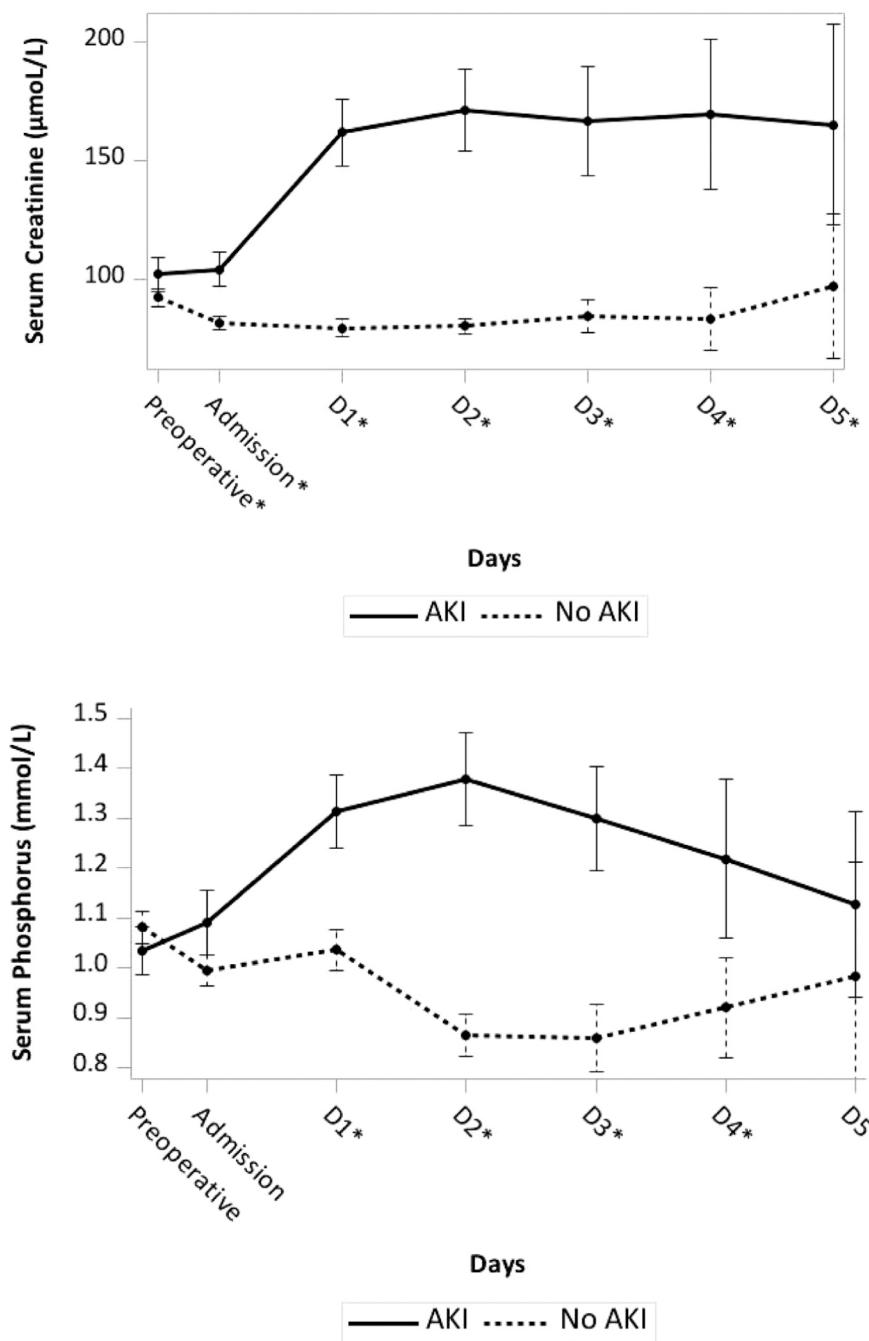


Fig 3. Serum creatinine (*upper panel*) and serum phosphate (*lower panel*) mean \pm standard deviation over the first 5 days after surgery in acute kidney injury patients and no-acute kidney injury patients. In acute kidney injury patients, serum phosphate and serum creatinine increased from the preoperative value until the 48-hour peak and appeared significantly different from that of no-acute kidney injury patients after Holm's correction (*). AKI, acute kidney injury; D, day.

change in the prognosis of the disease because it correlates with death.¹⁸⁻²⁰ The present study suggested that SPh correlates with kidney disease severity, as it already has been reported in AKI of various causes.²⁸ Conversely, the good correlation between SPh and AKI severity allows for the anticipation that AKI will not be severe if the SPh peak value at 48 hours after surgery is less than 1.33 mmol/L.

Taking for granted that an SPh increase correlates with AKI severity, an SPh decrease after its H48 peak could provide important information on renal recovery. The authors are aware that the present study's arbitrary definition of

renal recovery had at least 2 restrictions. First it depended on SCr, and SCr may not be accurate to assess renal function recovery after AKI.²⁶ Second, renal recovery was assessed at hospital discharge, meaning within 1- to- 2 weeks after AKI. This short-term evaluation may have missed late recovery because various patterns of renal recovery are possible.²⁹⁻³¹ Nevertheless, it is noticeable that renal recovery started early, within 24 hours after the SCr peak. However, SCr declined slowly, and its early decrease was not excellent for predicting renal recovery, as assessed by the ROC curve analysis, although it is the

Table 2
Serum Phosphate and Serum Creatinine Comparisons in AKI Versus Non-AKI Among CKD Status

	CKD (n = 86) No-AKI (n = 44)	AKI (n = 42)	p	Non-CKD (n = 174) No-AKI (n = 130)	AKI (n = 44)	p
Serum creatinine						
Serum phosphatePreoperative value ($\mu\text{mol/L}$)	(n = 44) 116.5 [105.0-125.5]	(n = 42) 120.0 [103.0-139.0]	0.531*	(n = 129) 81.0 [74.0-93.0]	(n = 43) 85.0 [70.0-93.0]	0.607*
Serum phosphateRelative variation (%)	(n = 23) -15.7 [-22.3 to -6.7]	(n = 32) 33.8 [18.5-87.0]	<0.001	(n = 126) -11.8 [-19.5 to -3.9]	(n = 40) 48.3 [23.5-125.0]	<0.001†
Serum phosphate						
Preoperative value ($\mu\text{mol/L}$)	(n = 29) 1.1 [1.0-1.2]	n = 34 1.0 [0.9-1.1]	0.038*	(n = 107) 1.1 [0.9-1.2]	(n = 29) 1.0 [0.9-1.2]	0.704*
Serum phosphateRelative variation (%)	(n = 23) -7.5 (\pm 17.4)	(n = 32) 40.6 (\pm 33.6)	<0.001‡	(n = 81) -26.1 [-40.6 to -11.2]	(n = 25) 37.6 [13.2-66.0]	<0.001*

NOTE. Quantitative data are expressed as mean (\pm standard deviation) or median [Q25-Q75] and were compared with the Wilcoxon Mann–Whitney (*) or Student *t* test (‡) or the test, as appropriate. †p values are comparisons between AKI and no-AKI.

Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease, defined as preoperative EPI-eGFR (EPIdemiology-estimated GFR) <60 mL/m/1.73 m² (variation at 48 hours: [value at hour 48 – preoperative value]/preoperative value); IRA, xxx.

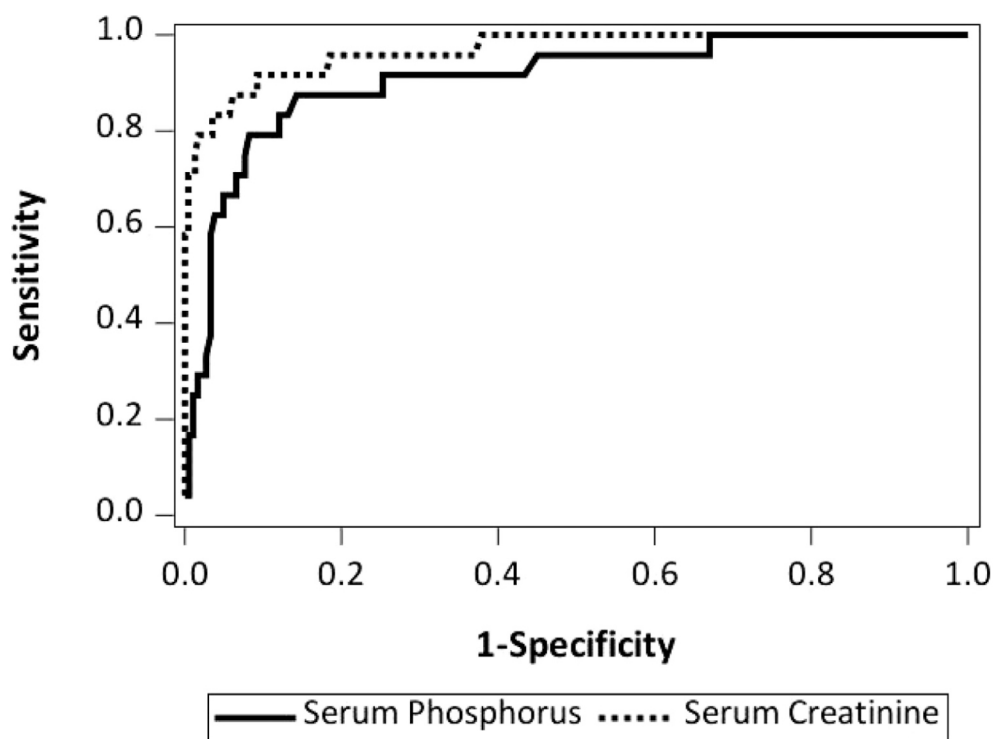


Fig 4. The receiver operating characteristic curve of serum phosphate and serum creatinine at 48 hours for Kidney Disease Improving Global Outcomes stages 2 or 3 diagnosis.

reference criterion. The percent decrease of SPh 24 hours after the H48 peak was 2.5 times greater than the percent decrease of SCr. The ROC AUC of the SPh decrease was not better than that of SCr, but 2 points recommended SPh monitoring during renal recovery. First, the SPh decrease helped to reclassify 42% of AKI patients for whom the SCr decrease was less than the SCr cutoff value predictive of renal recovery. Second, a probability of 100% recovery had been observed if the SPh decrease 24 hours after the H48 peak exceeded 25%. Taken together, these results suggested that SPh could be considered as a useful biomarker of AKI severity and evolution after cardiac surgery.

This study had several limitations. First, it was a monocentric, observational study. Second, the authors chose not to report other electrolytic disorders during AKI such as serum pH, calcemia, and bicarbonate. The study was not designed to address the issue of AKI's effect on all electrolytes but to focus on SPh kinetics as was done previously in CKD. In addition, SPh perioperative changes were independent of dietary or external intake because there were no potential medical interventions except the use of RRT when appropriate. Third, results on renal recovery were based on a small number of patients, which may have limited the statistical power of this specific analysis. Fourth, 260 patients were included

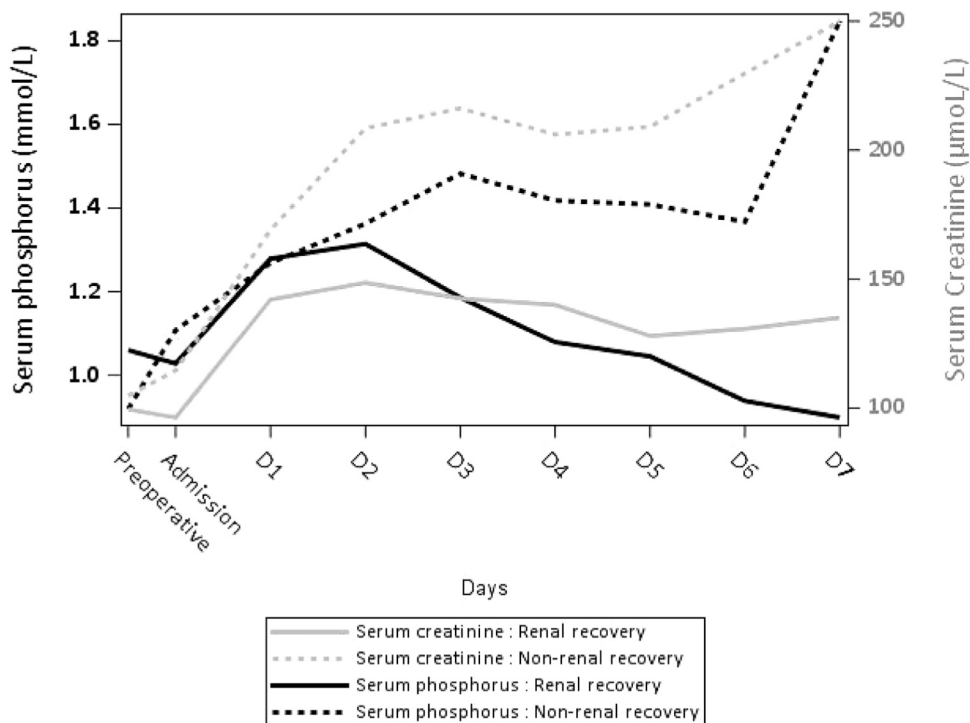


Fig 5. Follow-up over 7 postoperative days of serum phosphate and serum creatinine levels in acute kidney injury patients. Serum creatinine and serum phosphate are expressed as the mean in acute kidney injury patients over the first 7 days after surgery in kidney recovery and non-kidney recovery patients. The serum phosphate changes 24 hours after the 48-hour peak were -12.4% (± 23.9) in renal recovery patients, whereas it increased by 12.6% (± 25.7) in non-renal recovery patients ($p = 0.007$). Kinetic of serum creatinine showed similar significant variations between groups but with a slope less steep than for serum phosphate. D, day.

Table 3
Relative Variation of Serum Phosphate and Serum Creatinine 24 Hours After the 48-Hour Peak in AKI Patients

Relative Variation Between 48 h and 72 h (%)	SPh	SCr
Renal recovery patients	-12.4% (± 23.9)	-7.8% [-18.9 to 1.7]
No renal recovery patients	12.6% (± 25.7)	4.5% [-5.0 to 16.0]
p value	0.007*	0.016†
ROC analysis		
AUC [95% CI]	0.76% [0.62-0.91]	0.74% [0.60-0.89]
Threshold	-2.5%	-6.4%
Se	69.8%	54.7%
Sp	77.8%	90.0%
PPV	93.8%	96.7%
NPV	35.0%	27.3%

NOTE. Quantitative data are expressed as mean (\pm standard deviation) or median [Q25-Q75] and were compared between the 2 groups (renal v no renal recovery) with the Student t (*) or Wilcoxon Mann–Whitney (†) test, as appropriate.

Abbreviations: AKI, acute kidney injury; AUC, area under the curve; CI confidence interval; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic; SCr, serum creatinine; Se, sensitivity; Sp, specificity; SPh, serum phosphate.

in the study, slightly fewer than expected (264), but this affected the statistical power only marginally. Finally, the effect of the extent of SPh increase or decrease during AKI is unknown on long-term outcome.

Conclusion

SPh behaves as an interesting AKI biomarker, with the major asset of being easily available in daily practice. Nevertheless, additional studies are required to evaluate the effect of SPh kinetics, combined with SCr changes, on clinical decision-making regarding AKI management.

Conflict of Interest

None.

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