Urinary potassium excretion and its association with acute kidney injury in the intensive care unit

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Urinary potassium excretion and its association with acute kidney injury in the Intensive care unit.

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Abstract

Purpose: Using urinary indices as a quick bedside test to assist management of oliguria and acute kidney injury (AKI) has long been sought. This study assessed whether urinary potassium excretion is related to simultaneously calculated creatinine clearance (CrCl) and can predict AKI in the critically ill.

Materials and methods: In this prospective cohort study, the correlation between 2-hour urinary potassium excretion and simultaneously calculated CrCl of 61 critically ill patients was assessed by Pearson's correlation coefficient, and their ability to predict AKI (≥ stage 1 KDIGO) in the subsequent 7 days was assessed by area under the receiver-operating-characteristic (AUROC) curve.

Results: Urinary potassium excretion (median 6.2mmol, range 0.8-24.3) correlated linearly with CrCl (correlation coefficient: 0.58, 95% confidence interval [CI] 0.38-0.72; p=0.001), and had a moderate ability to predict subsequent AKI (n=19 [31%]; AUROC 0.747, 95%CI 0.620-0.850; p=0.001), especially in patients without prior exposure to furosemide within 24-hours (correlation coefficient 0.61, 95%CI 0.41-0.76; AUROC 0.789, 95%CI 0.654-0.890; p=0.001, respectively).

Conclusions: Urinary potassium excretion correlates with CrCl and predicts AKI in the critically ill without recent furosemide exposure. Given 2-hour urinary potassium excretion can be measured easily, its potential as a marker of renal function deserves further study.

Introduction

AKI (Acute Kidney Injury) affects 22-67% of the critically ill [1,2] and is associated with significant morbidity and mortality [3]. In patients admitted to intensive care units (ICU) between 4 to 13.5% require renal replacement therapy (RRT)[4,5] and, within this highest risk group of patients, there is a 50-60% mortality with a further 5-20% remain dependent on dialysis on hospital discharge [4]. Early identification of patients at high-risk of AKI facilitates risk stratification, avoidance of nephrotoxins, and proactive investigation of any possible untreated pathologies. Although many renal biomarkers (e.g. NGAL) have been shown to be sensitive in identifying patients before they develop AKI based on clinical criteria, their use as a bedside risk-stratifying tool remains limited, due to their availability, high cost, and also a slow turn-around time.

Human body is a net potassium producer. In addition to adjusting the reabsorption from the glomerular filtrate, the kidney also has an ability to actively secrete potassium – primarily by H-K-ATPase in the intercalated cells and Na-K-ATPase in principal cells (controlled by aldosterone) located in the distal convoluted tubule and collecting duct – to maintain potassium homeostasis. Due to a combination of reduction in glomerular filtration rate (GFR) and tubular function, a high plasma potassium concentration is not rare in patients with severe chronic renal failure. Evidence suggests that in chronic renal failure, those who maintain a reasonable amount of daily urinary potassium excretion have a better long-term prognosis than patients with a lower urinary potassium excretion [6]. In the general population without chronic renal failure, a high urinary potassium excretion has also been reported to be associated with a lower risk of cardiovascular events, hypertension [7], and death [8] compared to those who have a lower renal capacity to excrete potassium.

A high fractional urinary excretion of potassium [$FE_K = (U_K \times P_{Cr}) \times 100/(P_K \times U_{Cr})$], measured prior to the occurrence AKI, has been reported to be associated with an increased risk of persistent AKI for those who develop AKI [9]. Unfortunately, FE_K is mathematically inversely related to the calculated

creatinine clearance (CrCl), making it difficult to exclude a high FE_K being a confounder in the relationship between reduced CrCl and poor renal outcomes. Currently, it is uncertain whether urinary potassium excretion is increased or decreased in AKI. As the kidney function fails, the renin-angiotensin-aldosterone pathway is activated as a part of the AKI [10], which would enhance distal tubular potassium excretion [9]. On the other hand, a reduction in GFR and tubular potassium secretory function may dominate in determining how much potassium can be excreted in the urine, resulting in hyperkalemia commonly seen in severe AKI [9].

Numerous studies have looked at blood and urinary indices to try to establish their roles in identifying early AKI [11,12] prior to the rise in plasma urea and creatinine concentration. Historically, fractional sodium excretion (FE_{Na}), as a discriminator between prerenal and non-prerenal AKI, has been the main focus of investigation [13]. More recently FE_{Na} has also, unfortunately, been shown to be a poor discriminator between persistent and transient AKI in the critically ill care [14]. It is uncertain whether urinary potassium excretion would be more useful than urinary sodium excretion in identifying patients at risk of AKI in the acute care setting. We hypothesized that urinary potassium excretion is reduced in AKI, and may have a direct relationship to the calculated CrCl. In this pilot prospective cohort study, we aimed to assess whether urinary potassium excretion was linearly related to the calculated CrCl, and whether it could discriminate between patients who subsequently developed AKI and those who did not.

Methods

After obtaining approval from the Clinical Safety and Quality Unit approval (No 14537), we aimed to recruit 50 critically ill patients (>16 years-old) admitted to Royal Perth Hospital Intensive Care Unit (ICU), between April and June 2017, into this pilot prospective cohort study. Patients who were admitted to the ICU within 24-hours with an indwelling urinary catheter and due to have early morning (6am) plasma electrolytes and renal function test were eligible for enrolment. Patients who were anuric (<100ml/day), already undergoing dialysis or RRT, with known pre-existing renal failure, or had previously been included in this study in their prior ICU admission were excluded. A total of 61 patients was recruited by the end of the three-month study period.

Urinary volume between 4am and 6am was recorded, and samples for urinary biochemical analyses were collected at 6am (from the urine collected between 4-6am) by the nursing staff in the first morning of a patient's admission to ICU (within the first 24-hours of ICU admission), immediately prior to the morning blood collection for plasma electrolytes and renal function test. The urinary sodium, potassium and creatinine concentrations were measured by the hospital biochemistry laboratory, using the Abbott Diagnostics® (ARCHITECT c16000) chemistry analyzer. All reagents and consumables used for the assays were provided by Abbott Diagnostics®.

In addition to demographic data, Sequential Organ Failure Assessment (SOFA) score (on the day of enrolment), Acute Physiology and Chronic Health Evaluation (APACHE) II score, whether the patient had received furosemide or other diuretics within the 24-hours (including the type and dose) prior to enrolment, invasive/non-invasive respiratory support and vasopressors were also recorded. All patients were followed up to hospital discharge including data on the peak plasma creatinine concentration within 7 days of enrolment and during the whole hospital stay, KDIGO AKI grading after enrolment, RRT, and mortality.

The total amount of urinary potassium excreted in 2-hours was calculated by multiplying the 2-hour urine sample potassium concentration (mmol/l) by the urine volume (litres in the 2 hours collection period) to give mmol of potassium excreted in 2 hours. We chose a 2-hour urine collection period for this study because of previous studies, including one on ICU patients, have shown that it correlates well with the calculated CrCl over a 24-hour period [15, 16].

CrCl was calculated using the below formula:

$$\text{Cr Cl (ml/min)} = \frac{\text{Urine Creatinine (mol/l) x Urine Volume (ml)}}{\text{Plasma cratinine (mmol/l) x Time (mins)}}$$

Statistical analyses

Categorical and continuous variables with skewed distributions were analysed by Chi Square and Mann Whitney tests, respectively. Pearson's correlation coefficient was used to assess the degree of linear relationship between urinary potassium excretion and the calculated 2-hour CrCl, stratified by whether diuretic was given within 24 hours prior to study enrolment. Area under the receiver-operating-characteristic (AUROC) curve was used to assess the ability of urinary potassium and sodium excretion (in 2 hours), SOFA score and calculated 2-hour CrCl to predict the risk of KDIGO AKI (stage 1 or higher) within 7 days of study enrolment. All statistical analyses were performed by SPSS for Windows (version 24.0, IBM, USA) and MedCalc® Statistical Software (version 17.9.6, Ostend, Belgium); and a two-tailed α-error of <5% was taken as significant.

Results

Patient characteristics

The patient group was representative, for the most part, of our ICU population (**Table 1**). There was a slightly larger proportion of male patients (62%), primarily because the study center is Western Australia's State Trauma Center and trauma patients constituted 25% of our study patients. The median APACHE II score was 15, 25% required mechanical ventilation and 36% were treated with vasopressors at the time of enrolment. The acuity of the study cohort was skewed towards the lower end, in part, due to excluding patients who had already started dialysis. Exclusion of these dialysed patients, in addition to the exclusion of those with pre-existing chronic renal impairment, may also explain why only a small proportion of our study patients eventually required RRT (5%). In the patient group, 9 received frusemide in the 24 hours prior. No other diuretics were administered.

Correlation between 2-hours urinary potassium excretion, urinary sodium excretion, SOFA scores and calculated CrCl

The absolute quantity of urinary potassium excreted correlated with the calculated CrCl in a relatively linear fashion (Pearson's correlation coefficient: 0.58, 95% confidence interval [CI] 0.38-0.72; p=0.001), especially when only patients without an exposure to diuretics within 24-hours prior to enrolment were considered (Pearson's correlation coefficient 0.61, 95%CI 0.41-0.76) (**Fig 1**). Prior recent exposure to furosemide occurred in nine patients (15%) and, as expected from the actions of furosemide, the urinary potassium excretion was not significantly correlated with the calculated CrCl in these patients. Both plasma potassium concentrations (Pearson's correlation coefficient -0.24, 95%CI -0.47 to 0.01) and urine output alone (Pearson's correlation coefficient 0.16, 95%CI -0.10 to 0.39; p=0.233) had no significant correlation with the calculated CrCl (**Fig 2 and Fig 3,** respectively).

Analyzing all patients as a whole, urinary sodium excretion had little correlation with the calculated CrCl (Pearson's correlation coefficient -0.04, 95%CI -0.21 to 0.29; p=0.741) (**Fig 4)**. However, for

those who had recently received furosemide, a higher urinary sodium excretion – likely representing a good urinary response to furosemide – was significantly correlated with the calculated CrCl (Pearson's correlation coefficient 0.79, 95%CI 0.27-0.96) [17].

Although urinary potassium excretion obtained within the first 24-hours of ICU admission was not as good as the simultaneously calculated CrCl in predicting AKI within the subsequent 7 days after the urinary analyses, it still had a moderate ability to discriminate between patients with and without subsequent AKI (n=19 [31%]; AUROC 0.747, 95%CI 0.620-0.850; p=0.001) (**Fig 5**). This ability was further improved after restricting the analyses to patients who did not receive any furosemide within 24-hours prior to enrolment (AUROC 0.789, 95%CI 0.654-0.890; p=0.001) (**Table 2**), and was better than both SOFA score (AUROC 0.744, 95%CI 0.605-0.839; p=0.001) and urinary sodium excretion (AUROC 0.635 95%CI 0.530-0.779 p=0.024).

Discussion

This pilot prospective cohort study showed that urinary potassium excretion had a reasonably linear correlation with the simultaneously calculated CrCl in the critically ill if they did not have recent exposure to diuretics. In addition, urinary potassium excretion also had a modest ability, and was better than both SOFA score and urinary sodium excretion, to predict patients who subsequently developed AKI. These findings have some clinical relevance and require further consideration.

First, our results showed that the absolute amount of urinary potassium excretion has a reasonably strong positive correlation with the simultaneously calculated CrCl, but only in patients without recent exposure to diuretics. This finding is not surprising because plasma potassium is freely filtered through the glomeruli; the absolute amount of potassium excreted in the urine will be, at least in part, dependent on the GFR. Based on the mechanism of actions of furosemide, both the reabsorption of potassium in the ascending loop of Henle and secretion of potassium in the distal tubule and collecting duct would be affected by an exposure to furosemide, increasing urinary potassium excretion independent of the GFR. As such, any interpretation of urinary potassium excretion, either as a surrogate marker of CrCl or prognostic marker for subsequent AKI, is only valid for patients without recent (<24 hours) diuretics exposure.

Second, given urinary potassium concentration can be easily measured by any biochemical laboratories and also many modern point-of-care blood gas analyzers, it may represent a simple test to alert clinicians about the renal status of their patients, better than using the SOFA score and urine output alone (**Fig 5**). Based on the AUROC curve of urinary potassium excretion, using a cut-point of urinary potassium excretion $\leqslant$3.8mmol in 2 hours would have a specificity of 85% and sensitivity of 77% (Youden index J=0.62) in predicting subsequent AKI (KDIGO stage $\geqslant$1) within 7 days of testing. This is different from the relationship between urinary sodium excretion and the calculated

CrCl or risk of subsequent AKI. In the first instance, for those without recent furosemide exposure, urinary sodium excretion had hardly any correlation with the simultaneously calculated CrCl. Even though our results showed that there was a reasonably high correlation between urinary sodium excretion and the calculated CrCl for those with recent exposure to furosemide, the range of the quantity of urinary sodium excreted between patients was narrow (<15mmol over 2 hours) – which can be potentially confounded by variations in dose, route of administration, and timing of urinary analyses – making it clinically not useful.

Third, we need to acknowledge the limitations of our study. This was a pilot study aiming at testing the hypothesis of that there was a correlation between the calculated CrCl and urinary potassium excretion. In retrospect, the interpretation of our findings would be more comprehensive if we had included an equal number of patients with and without recent furosemide exposure. The population that was studied may also be atypical for other ICUs, as the state trauma center we have a proportionately larger number of trauma patients, this results in male being overrepresented and a younger population demographic. Trauma patients without prior renal impairment (as per exclusion criterion in this study) have a lower risk of developing AKI and, when it occurs, the pathophysiological mechanisms leading to AKI are likely different from sepsis – limiting the generalizability of our results to septic patients. It should be noted that the correlation between urinary potassium excretion and the calculated CrCl or risk of subsequent AKI would theoretically be weakened by any variations in dietary intake of potassium and extra-renal loss of potassium. These two extra-renal factors would only have affected urinary potassium excretion, but not the calculated CrCl or risk of AKI, resulting in a weaker ability for urinary potassium to correlate or predict these two outcomes.

We also noted that some of our study patients had augmented CrCl related to over-representation of trauma patients in this study [18], and this could have affected or amplified the degree of correlation

between CrCl and potassium excretion [19]. Additionally, as we enrolled patients at their first routine blood collection (4-6am approx.) we lost the opportunity to study patients who required RRT between ICU admission and the time of morning blood test the day after. Coupled with the exclusion criteria, this could, in part, explain the lower acuity of our study patients and the low rate of progressing to requiring RRT. We could not confirm whether urinary potassium excretion would be useful in predicting risk of subsequent RRT in the critically ill, due to low numbers. A large multicentre prospective study will be important to assess whether urinary potassium excretion can be used to predict risk of severe AKI requiring RRT in the critically ill.

In summary, this pilot prospective study indicates that urinary potassium excretion correlates relatively linearly with CrCl, and can predict AKI in the critically ill in patients without recent furosemide exposure. Given 2-hour urinary potassium excretion can be measured easily, its potential as a marker of renal function deserves further study.

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Table 1. Characteristics of the cohort (N=61).

Variables	**Median (range) unless stated otherwise**
Age, yrs	51 (18-90)
Male, no. (%)	38 (62)
Diagnosis, no. (%)	
- Sepsis	8 (13)
- Cardiac arrest	4 (6.5)
- Trauma	15 (24.5)
- Respiratory failure	4 (6.5)
- Overdose / poisoning	3 (5)
- Postoperative	14 (23)
- Other	13 (21)
Requiring vasopressor, no. (%)	15 (24.6)
Requiring mechanical ventilation, no. (%)	22 (36.1)
SOFA score on the study day	5 (0-17)
Plasma creatinine, umol/l*	71 (36-451)
2-hr urinary output, ml	110 (20-650)
Measured 2-hr creatinine clearance, ml/min	114 (4.0-305)
Plasma sodium, mmol/l	139 (131-153)
Plasma potassium, mmol/l	4.0 (2.3-5.5)
2-hr urinary sodium concentration, mmol/l	48 (7.5-214)
2-hr urinary potassium concentration, mmol/l	59 (4-152)
2-hr urinary sodium excretion, mmol	4.6 (0.03-64.3)
2-hr urinary potassium excretion, mmol	6.2 (0.8-24.3)
KDIGO AKI (stage ≥1) within 7 days, no. (%)	19 (31)
Subsequent dialysis or CRRT, no. (%)	3 (5)
Hospital mortality, no. (%)	8 (13)
Furosemide within 24 hrs prior, no. (%)	9 (15)
Length of hospital stay, days	12 (2–120)
Length of ICU stay, days	3 (1-18)
APACHE II score	15 (3–37)

APACHE, Acute Physiology and Chronic Health Evaluation. CRRT, Continuous renal replacement therapy. KDIGO, Kidney Disease: Improving Global Outcomes. AKI, Acute Kidney Injury. SOFA, Sequential Organ Failure Assessment. *median plasma creatinine = 0.80mg/dl (range: 0.41-5.10).

Table 2. Ability of each predictor to discriminate between patients with and without developing acute kidney injury (KDIGO AKI stages 1-3) within 7 days of testing.

			AUROC (95%CI)	
Predictor	**Whole cohort* (N=61)**	**P value**	**Without furosemide within 24hrs prior (n=52)**	**P value**
1. Urinary 2-hr K^+ excretion	0.747 (0.620-0.850)	0.001	0.789 (0.654-0.890)#	0.001
2. Urinary 2-hr Na^+ excretion	0.663 (0.530-0.779)	0.024	0.635 (0.490-0.764)	0.118
3. SOFA score	0.734 (0.605-0.839)	0.001	0.744 (0.604-0.855)	0.004
4. Measured 2-hr CrCl	0.900 (0.796-0.962)	0.001	0.885 (0.766-0.956)	0.001

*19 patients (31%) developed stages 1-3 KDIGO AKI. #A cut-point of urinary potassium excretion ≤3.8mmol in 2 hrs had a specificity of 85% and sensitivity of 77% (Youden index J=0.62) in predicting AKI (KDIGO stage ≥1) within 7 days of testing.
AUROC, area under the receiver-operating-characteristic curve. CI, confidence interval. CrCl, creatinine clearance. KDIGO, Kidney Disease: Improving Global Outcomes. SOFA, Sequential Organ Failure Assessment.

Highlights

- Absolute urinary potassium is positively associated with creatinine clearance.
- Urinary potassium excretion has a modest ability to predict renal failure.
- Urinary potassium outperforms SOFA and urinary sodium at predicting renal failure.

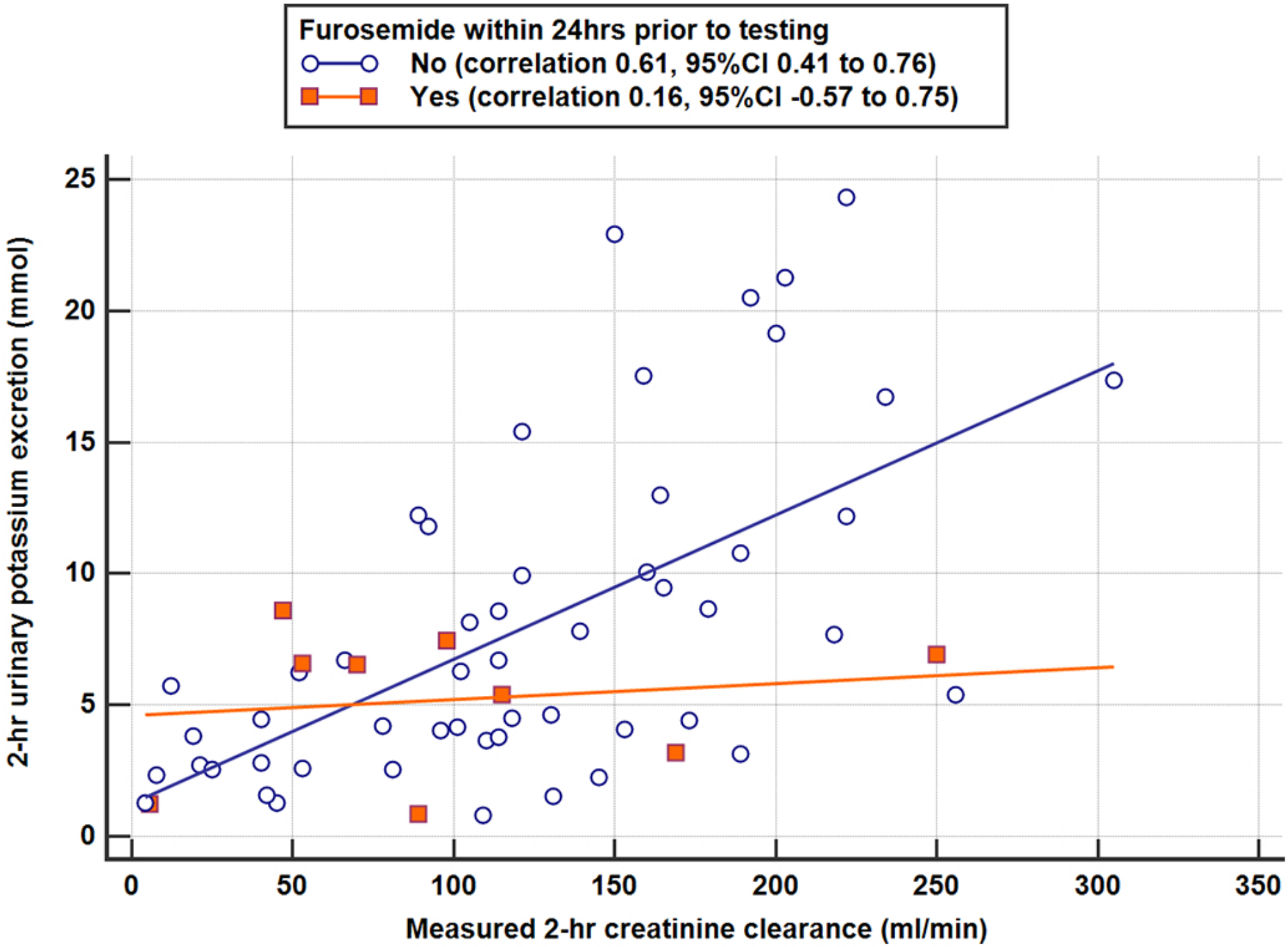
Furosemide within 24hrs prior to testing
No (correlation 0.61, 95%CI 0.41 to 0.76)
Yes (correlation 0.16, 95%CI -0.57 to 0.75)
2-hr urinary potassium excretion (mmol)
0
5
10
15
20
25
0
50
100
150
200
250
300
350
Measured 2-hr creatinine clearance (ml/min)

Figure 1

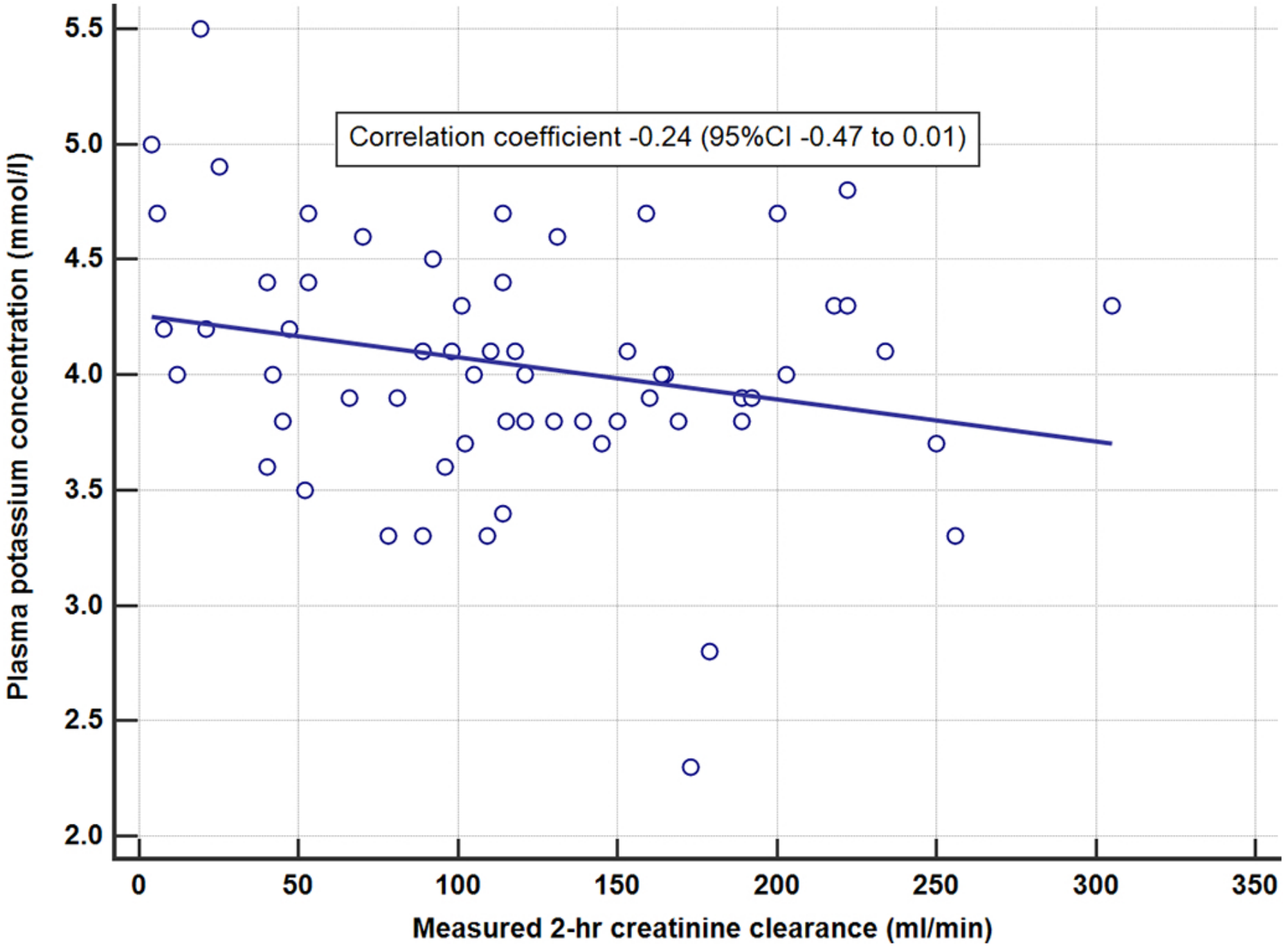
Correlation coefficient -0.24 (95%CI -0.47 to 0.01)
Plasma potassium concentration (mmol/l)
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
0
50
100
150
200
250
300
350
Measured 2-hr creatinine clearance (ml/min)

Figure 2

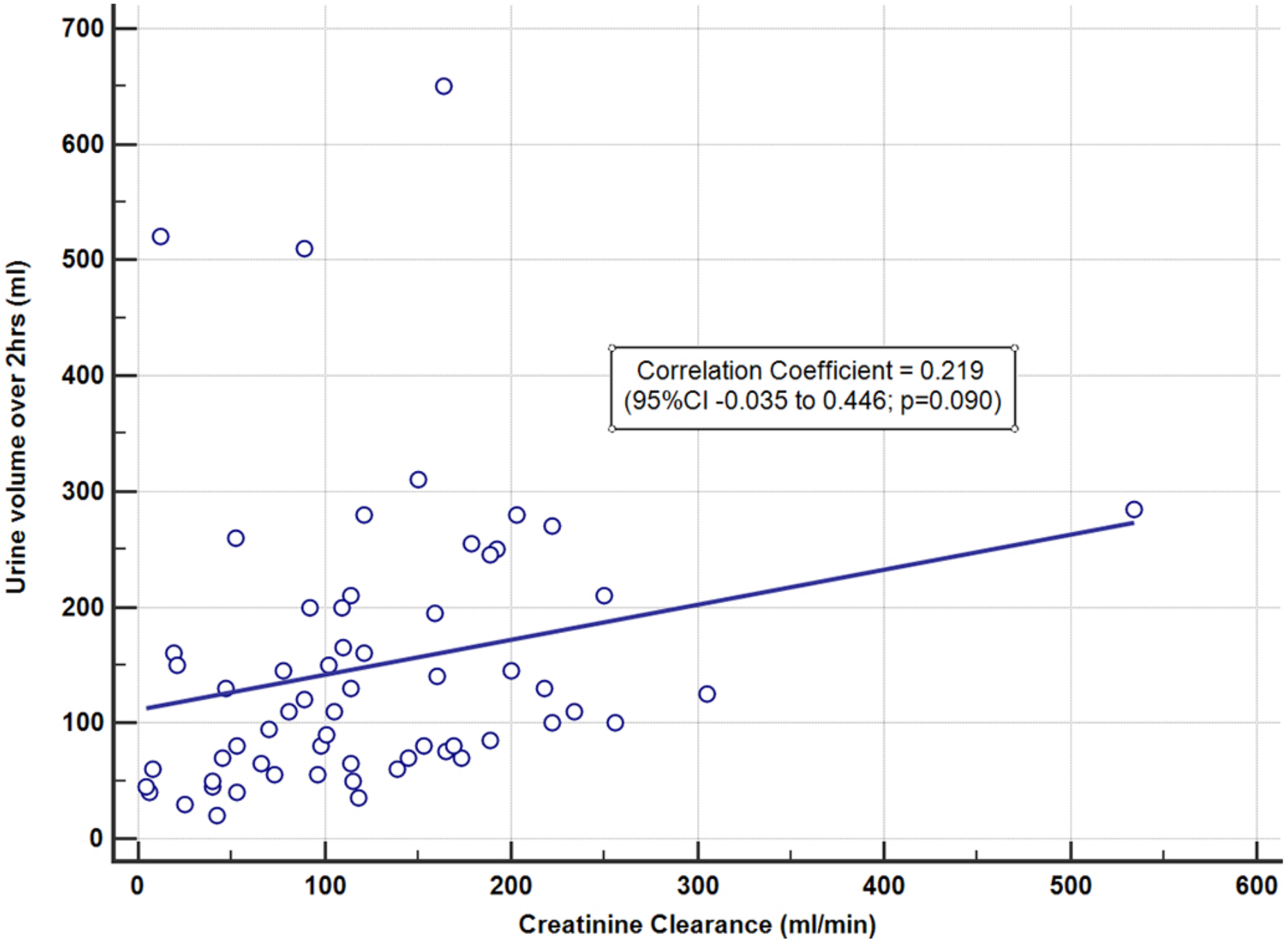
Urine volume over 2hrs (ml)
700
600
500
400
300
200
100
0
Correlation Coefficient = 0.219
(95%CI -0.035 to 0.446; p=0.090)
0
100
200
300
400
500
600
Creatinine Clearance (ml/min)

Figure 3

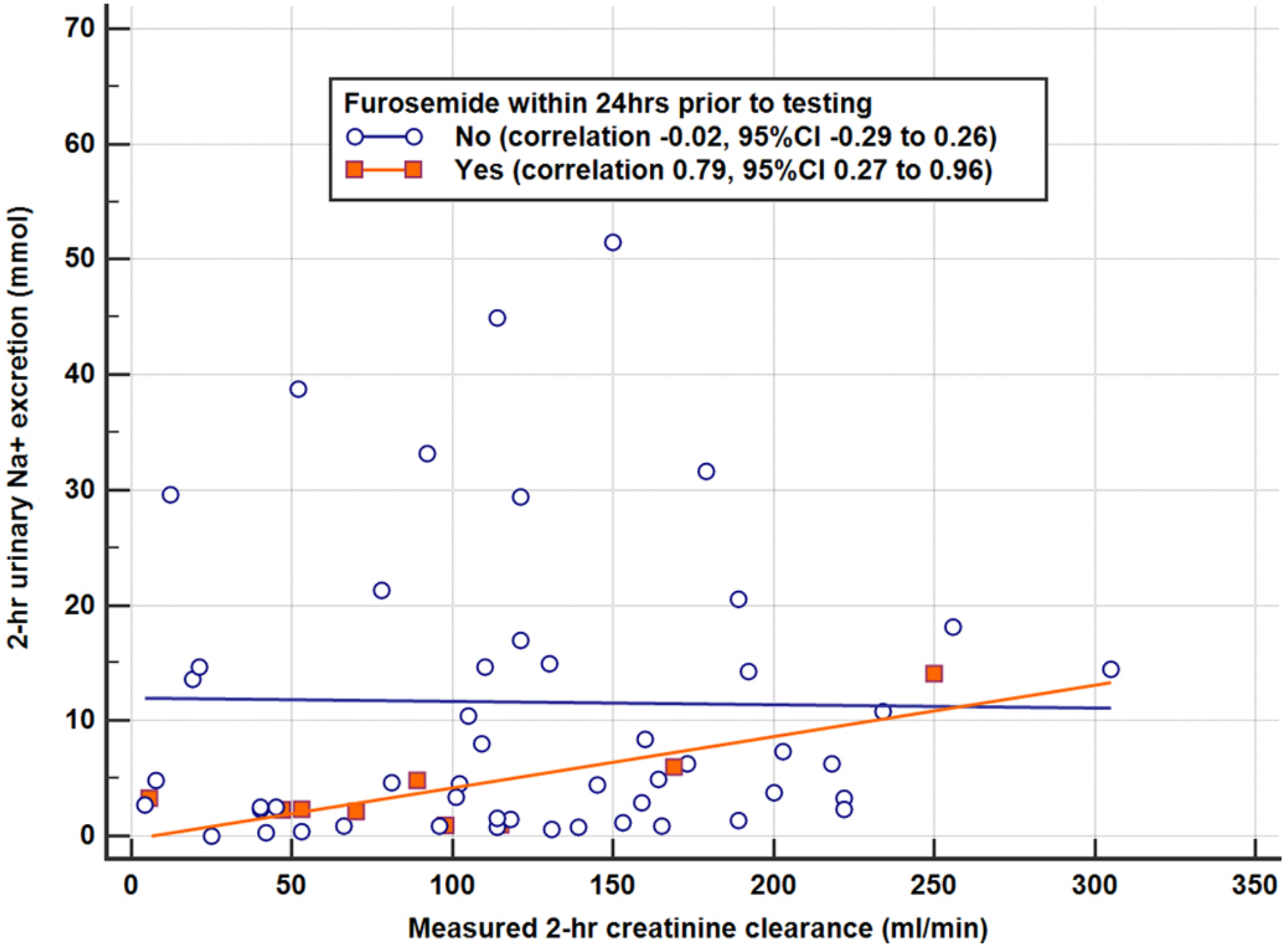
Furosemide within 24hrs prior to testing
No (correlation -0.02, 95%CI -0.29 to 0.26)
Yes (correlation 0.79, 95%CI 0.27 to 0.96)
2-hr urinary Na+ excretion (mmol)
Measured 2-hr creatinine clearance (ml/min)
0
10
20
30
40
50
60
70
0
50
100
150
200
250
300
350

Figure 4

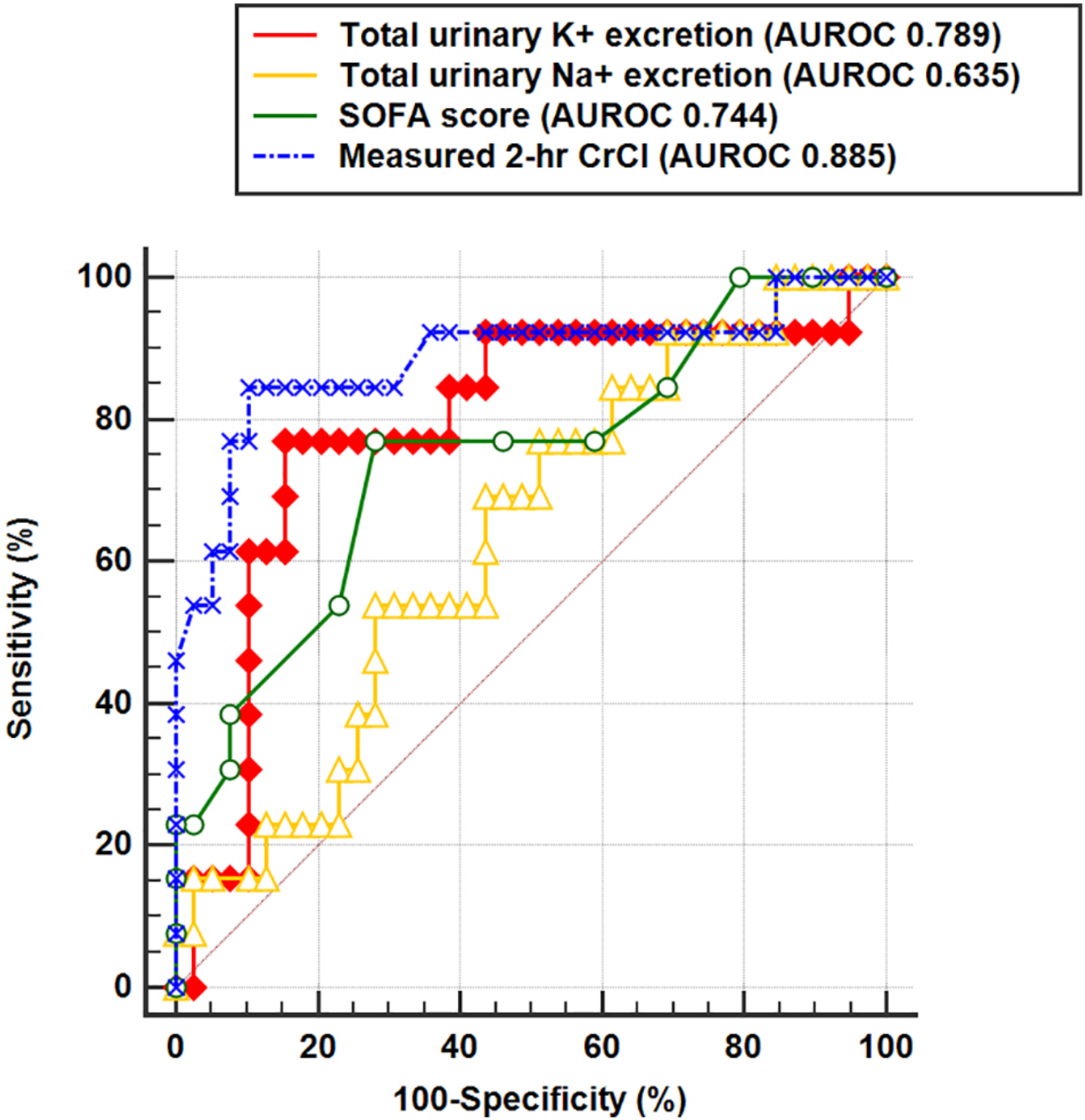
Total urinary K+ excretion (AUROC 0.789)
Total urinary Na+ excretion (AUROC 0.635)
SOFA score (AUROC 0.744)
Measured 2-hr CrCl (AUROC 0.885)
Sensitivity (%)
100
80
60
40
20
0
0
20
40
60
80
100
100-Specificity (%)

Figure 5