The Egyptian Journal of Critical Care Medicine (2013) 1, 69–77

The Egyptian College of Critical Care Physicians

The Egyptian Journal of Critical Care Medicine

http://ees.elsevier.com/ejccm
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ORIGINAL ARTICLE

Comparison between fractional excretion of sodium and fractional excretion of urea in differentiating prerenal from renal azotemia in circulatory shock

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Received 25 February 2013; revised 24 April 2013; accepted 7 May 2013
Available online 10 June 2013

KEYWORDS

Fractional excretion;
Sodium;
Urea;
Acute kidney injury;
Circulatory shock

Abstract *Background:* Fractional excretion of sodium (FENa) is used to differentiate renal from prerenal azotemia. However, many drugs and medical conditions affect the sodium (Na^+) handling in the kidney. But the fractional excretion of urea (FEurea) is dependent on passive forces and is less influenced by the diuretic therapy.

Objective: Comparison between FENa and FEurea in differentiating renal from prerenal azotemia in circulatory shock, and the effect of diuretics on their handling.

Methods: Both FENa and FEurea were measured in 40 patients (pts) with AKI complicating circulatory shock. The pts were divided into 26 pts with prerenal (group-1) and 14 pts with renal azotemia (group-2). Group-1 was subdivided into 12 pts who did not receive diuretics 24 h before the sampling process (group-1a) and 14 pts who received diuretics (group-1b).

Results: Compared to patients with renal azotemia (group-2), those with prerenal azotemia (group-1) showed significantly lower FENa (0.99 ± 0.66 and 2.57 ± 1.73, $P < 0.05$) respectively, and significantly lower FEurea (29.7 ± 7.6 and 43.7 ± 15.4, $P < 0.001$) respectively. For differentiating renal from prerenal azotemia, compared to FENa, FEurea showed better sensitivity (78.1% vs. 71.4%) and specificity (88.5% vs. 69.4%) respectively. Moreover, FEurea was not significantly affected by the use of diuretics; sensitivity (78% vs. 78%) and specificity (92% vs. 88%) respectively, compared to pts who did not receive diuretics. On the other hand, compared to pts who did not receive diuretics, the use of diuretics significantly affected FENa; sensitivity (64% vs. 71%) and specificity (58% vs. 70%) respectively.

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Peer review under responsibility of The Egyptian College of Critical Care Physicians.

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 http://dx.doi.org/10.1016/j.ejccm.2013.05.001

Conclusions: FEurea is more sensitive, specific and less affected by the use of diuretics than FENa in differentiating renal from prerenal azotemia in patients with AKI complicating circulatory shock.

Introduction

The incidence of acute renal injury (AKI) in ICU is ~ 5%. The mortality rate increases up to 50% if AKI is a part of multiple organ dysfunction syndrome [1,2]. Prerenal azotemia is responsible for 30–40% of cases of oliguria in ICU; a situation which can be usually reverted by correcting the underlying disorder, but prolonged or severe prerenal azotemia may proceed to oliguric renal azotemia [2].

The outcome of patients with acute renal azotemia differs significantly from those with prerenal azotemia; the latter reflects the physiological hemodynamic regulatory mechanisms of conservation of water and electrolytes in response to hypovolemia and decreased glomerular filtration rate (GFR) [1–3].

Early detection of renal and excluding prerenal azotemia improve outcome [4]. Relying solely on the patient's response to fluid challenge in order to differentiate prerenal azotemia from renal azotemia, may lead to massive fluid overload in the latter [4,5].

Many laboratory tools have been used for differentiating renal from prerenal azotemia including blood urea/serum creatinine ratio, urine analysis (granular casts in patients with renal azotemia), urine osmolarity and specific gravity, urinary sodium level, urinary to plasma creatinine ratio (U/P Cr). However; all of them lack reasonable sensitivity and specificity [4–6].

One of these laboratory tools is the fractional excretion of sodium (FENa) which showed a reasonable sensitivity and specificity. FENa is the percentage of Na^+ filtered by the kidney which is not reabsorbed and excreted in the urine. It depends upon the fact that in the state of decreased renal blood flow, as in hypovolemia, there is enhanced reabsorption of Na^+ from renal tubules, making the fraction of the filtered Na^+ that is excreted in urine less than normal which means lower FENa in prerenal azotemia [4,5,7].

But unfortunately FENa (despite a simple lab tool) is affected by many drugs and medical conditions. Drugs that increase Na^+ excretion in urine as diuretics and dopamine [8] lead to false increase in FENa, and drugs that decrease Na^+ excretion as norepinephrine lead to false decrease in FENa [8,9]. Because 70% of ICU pts with AKI receive diuretic therapy, and many patients with circulatory shock receive dopamine and norepinephrine, the results of FENa are not justified in such conditions [7]. Moreover, lactic acidosis that occurs with circulatory shock may result in natriuresis and may falsely increase FENa as well [10].

Solutes other than Na^+, including urea, have been suggested to improve the diagnostic ability in the above mentioned clinical situations instead of FENa [7]. The fractional excretion of urea (FEurea) is mainly dependent on passive forces in the kidney and so is less influenced by the diuretic therapy [7].

The purpose of this study was to compare FENa and FEurea in differentiating renal from prerenal azotemia, and to detect the effect of diuretics on their handling. Few studies have specifically addressed this issue, but never in patients with AKI complicating circulatory shock.

Methods

This prospective study was conducted on 40 Egyptian patients with AKI complicating circulatory shock admitted to the general ICU of Al Fayoum University Hospital, from July 2011 to July 2012. Consents were taken from all of them according to the local ethics committee approval.

The patients were divided into two groups:

(1) *Group-1:* Included 26 patients; mean age 60.0 ± 15.15 (21–88) years (10 males) that had acute prerenal azotemia, and was subdivided into:
   - *Group-1a:* Included 12 patients who did not receive diuretics in the last 24 h before taking the urine and blood samples.
   - *Group-1b:* Included 14 patients who received diuretics, in the last 24 h before taking the urine and blood samples.

(2) *Group 2:* Included 14 patients; mean age 56.29 ± 19.5 (23–92) years (18 males) that had acute renal azotemia.

Regarding age and sex, groups-1 and 2 were comparable.

Inclusion criteria

All patients presented with AKI complicating circulatory shock during their stay in the ICU:

(1) Circulatory shock was diagnosed according to the Empiric Criteria [11], which requires 4 out of the following:
   - Ill appearing or altered mental status.
   - Heart rate > 100 beat/min.
   - Respiratory rate > 22/min, or $paCO_2$ < 32 mmHg.
   - Arterial hypotension (systolic blood pressure ⩽ 90 mmHg, decrease in systolic blood pressure by 40 mmHg from baseline or mean arterial blood pressure ⩽ 70 mmHg), for > 20 min duration.
   - Urine output < 0.5 mL/kg/h.
   - Arterial base deficit < −5 mEq/L, or lactic acidosis (> 2 mmol/L)

(2) Acute kidney injury was diagnosed according to the RIFLE-criteria (Risk, Injury, Failure, Loss and End-stage Kidney) in oliguric patients [12]: (Table 1)

(3) Prerenal azotemia diagnosis was settled when ⩾6 of the following criteria was found [10]:
   - *History:* Volume depletion, congestive heart failure, severe liver disease or other edematous states and anaphylaxis favor prerenal azotemia, while exposure to exogenous toxins such as medications and recent

Table 1 RIFLE-criteria for diagnosing acute kidney injury.

Class	Glomerular filtration rate criteria	Urine output criteria
Risk	Serum creatinine × 1.5	< 0.5 mL/kg/h for 6 h
Injury	Serum creatinine × 2	< 0.5 mL/kg/h for 12 h
Failure	Serum creatinine × 3, or ⩾4 mg/mL with an acute rise > 0.5 mg/mL	< 0.3 mL/kg/h for 24 h, or anuria ⩾ 12 h
Loss	Persistent acute renal failure = complete loss of kidney function > 4 weeks	
End-stage kidney disease	End-stage kidney disease > 3 months	

The serum creatinine increase is measured from its baseline value.

radiographic procedures with contrast, or endogenous toxins as in the case of myoglobinuria would favor acute tubular necrosis
- *Physical examination:* Loss of skin elasticity and skin turger in dehydrated patients, orthostatic changes in heart rate, presence of ascites or pedal edema suggests prerenal state.
- *Response to fluid challenge:* Favors prerenal state [7].
- *Urine analysis:* No abnormalities or hyaline and fine granular casts in prerenal states, while presence of muddy brown granular casts and renal tubular epithelial cells or casts indicates acute renal failure [13].
- *Urine specific gravity* ⩽ 1010 favors acute renal failure, while > 1018 favors prerenal state.
- *Urinary to plasma creatinine ratio (U/P Cr):* If > 20 is consistent with prerenal while levels < 15 suggest acute tubular necrosis.
- *Blood urea/serum creatinine ratio:* Would favor prerenal if > 40 and acute renal failure if < 40.
- *Urinary sodium (UNa):* If < 15 mEq/L favors prerenal azotemia, while > 20 mEq/L is consistent with acute tubular necrosis.

(4) Acute renal failure diagnosis was settled when < 6 of the previously mentioned criteria were present [10].

Exclusion criteria

- Chronic kidney disease.
- End stage kidney disease receiving renal replacement therapy.
- Obstructive nephropathy.
- Renal transplantation.
- Osmotic diuresis as in usage of mannitol or acetazolamide [7].

All patients were subjected to the following:

(1) Full history and clinical evaluation including the routine ICU monitoring; fluid balance sheet and hourly urine output measurement.
(2) Routine renal function tests:
- Blood urea, serum creatinine, and Na^+ and K^+ levels.
- Blood urea/serum creatinine ratio.
- Urinary/serum creatinine ratio.
- Urinary Na^+ level.
- Urine analysis.
- Urine specific gravity.
- Albumin/creatinine ratio or 24 h proteins in urine were used to exclude diabetic nephropathy [6].

(3) All patients had subjected to abdominal ultrasound to exclude any chronic kidney disease or obstructive nephropathy.
(4) Fractional excretion of sodium and fractional excretion of urea
- FENa was calculated as [7]:

$$\text{FENa} = [(\text{urinary sodium}/\text{plasma sodium})/(\text{urinary creatinine}/\text{plasma creatinine})] \times 100$$

- FEurea nitrogen was calculated as [7]:

$$\text{FEurea nitrogen} = [(\text{urinary urea nitrogen}/\text{blood urea nitrogen})/(\text{urine creatinine}/\text{plasma creatinine})] \times 100$$

Laboratory preparation of urea estimation to calculate FEurea nitrogen

- Samples:
  Specimen:
  – Serum or heparinized plasma (except ammonium heparin).
  – Urine was diluted at 1/20 to 1/50 with distilled water before analysis.

  Storage:
  – Serum and plasma are stable up to 24 h at room temperature, for one week at 4 °C. Frozen between −15 and −20 °C, these samples are stable for at least 2–3 months.
  – Urine samples are stable up to 4 days if stored at 4–8 °C. Urines could be preserved with thymol to avoid bacterial action or by maintaining the pH below 4.

- Blood and urine chemistries and electrolytes were performed on a Beckman automated analyzer (Beckman Instruments, Fullerton, CA, USA). (SEPPIM ELITech Group Company, European manufacturer of clinical chemistry diagnostic kits.)

(5) According to the medical situation and the cause of the circulatory shock, all patients continued their medications including the vasopressor drugs during their enrollment in the study.

Statistical methods

The collected data were organized, tabulated and statistically analyzed using Statistical Package for Social Sciences (SPSS Inc., USA) version 15.0. For quantitative data, the means ± standard deviations (SD) were calculated. The independent *t*-test and one-way ANOVA were used to test the statistical differences between groups. For qualitative data, the number and percent distribution were calculated, chi square (χ^2) was used as a test of significance. Pearson's and Spearman's correlations were used to determine the relation between FENa and FEurea with variables of the study when applicable. ROC-curve was used to determine the cut-off point at which highest sensitivity and specificity of FENa, FEurea and other variables were differentiated between prerenal and renal failures. *P*-values $\leqslant 0.05$ were considered statistically significant while $\leqslant 0.001$ were considered highly statistically significant.

Results

The patients who suffered from renal azotemia (group-2) significantly needed the mechanical ventilation and the renal replacement therapy than the patients with prerenal azotemia (group-1). Furthermore, the mortality rate was significantly higher in group-2 (Table 2). But, the number of patients who needed vasopressor drugs in their medication in both groups was comparable (Table 2).

The results of the routine lab parameters were comparable in both groups (Table 3).

Compared to group-2, the values of blood urea/serum creatinine ratio, the urinary creatinine/plasma creatinine ratio and the total urine output in the 1st 24 h were significantly higher, and the serum creatinine was significantly lower in group-1. On the other hand, the values of blood urea, serum Na^+ and urine Na^+ in both groups were comparable (Fig. 1).

Compared to group-2 (renal patients), both FENa and FEurea values of group-1 were significantly lower (Fig. 2).

Using one-way ANOVA test, both FENa and EFurea showed significantly incremental values with higher RIFLE-criteria from mild AKI (RIFLE-R), moderate AKI (RIFLE-I) to severe AKI (RIFLE-F) class (Table 4).

Weak correlations could be detected between the serum creatinine and both FENa and FEurea values ($r = 0.36$, $P < 0.05$ and $r = 0.37$, $P < 0.05$) respectively, but good

Table 2 The demographic and clinical data.

	Group-1 (prerenal)	Group-2 (renal)	*P* value
MAP (mmHg)	38.4 ± 9.1 (25–58)	39 ± 8.2 (30–58)	NS
Heart rate (beat/min)	112 ± 22 (52–168)	122 ± 27 (46–176)	NS
Temperature (°C)	36.9 ± 0.8 (35.5–40.2)	37.0 ± 0.6 (35.6–40.4)	NS
Respiratory rate (breath/min)	22 ± 11 (10–44)	19 ± 8 (10–48)	NS
Hypertension (n)	13 (50%)	6 (42.8%)	NS
Diabetes mellitus (n)	10 (38.5%)	5 (35.7%)	NS
Diuretics (n)	12 (46.2%)	8 (57.1%)	NS
Mechanical ventilation (n)	7 (27%)	7 (50%)	<0.05
Renal replacement therapy (n)	3 (11.5%)	9 (64.3%)	<0.001
Dopamine (n)	18 (69.2%)	11 (78.5%)	NS
Dobutamine (n)	14 (53.8%)	7 (50%)	NS
Noradrenaline (n)	13 (50%)	8 (57.1%)	NS
ICU stay (days)	8.0 ± 4.6 (2–15)	7.4 ± 3.6 (2–16)	NS
Mortality (n)	5 (19.2%)	7 (50%)	<0.05

NS: Non-significant; MAP: Mean arterial blood pressure.

Table 3 The routine lab results.

	Group-1 (prerenal)	Group-2 (renal)	*P* value
Hemoglobin (mg/mL)	9.9 ± 2.1 (6.1–15.6)	11.2 ± 2.8 (5.6–17.3)	NS
WBC × 1000 (cell/cm^3)	17,2 ± 4,5 (2,6–27,1)	15,5 ± 3,2 (3,2–32,1)	NS
Platelets × 1000 (n/cm^3)	275 ± 70 (90–412)	264 ± 75 (65–392)	NS
AST (mg/mL)	89 ± 48 (27–353)	109 ± 77 (31–342)	NS
ALT (mg/mL)	82 ± 33 (22–312)	86 ± 16 (26–317)	NS
PT (seconds)	17.2 ± 4.2 (12.5–35)	18.4 ± 3.1(12.6–41)	NS
PC (%)	72.2 ± 17 (34–97)	69.2 ± 22 (28–96)	NS
INR (ratio)	1.6 ± 1.1 (1.1–4.2)	2.1 ± 0.8 (1.1–4.8)	NS
Albumin (mg/mL)	2.9 ± .6 (1.7–5.2)	2.5 ± 0.4 (1.8–4.9)	NS
Total bilirubin (mg/mL)	1.4 ± 0.5 (0.6–6.1)	1.4 ± 0.6 (0.5–7.1)	NS
pH	7.25 ± 0.15 (6.92–7.31)	7.27 ± 0.22 (6.88–7.28)	NS
paO_2 (on room air) (mmHg)	64 ± 14 (38–115)	59 ± 11 (39–122)	NS
paO_2 (on MV) (mmHg)	109 ± 33 (45–350)	97 ± 32 (55–275)	NS
$paCO_2$ (mmHg)	34.2 ± 12.5 (12.4–66.3)	29.1 ± 11.7 (14.6–80.1)	NS
HCO_3 (mmol/L)	12 ± 6.1 (3.6–16.1)	11 ± 5.5 (5.1–15.2)	NS

NS: Non-significant; WBC: White blood cell; AST: Aspartate transaminase; ALT: Alanine transaminase; PT: Prothrombin time; PC: Prothrombin concentration; INR: International normalized ratio.

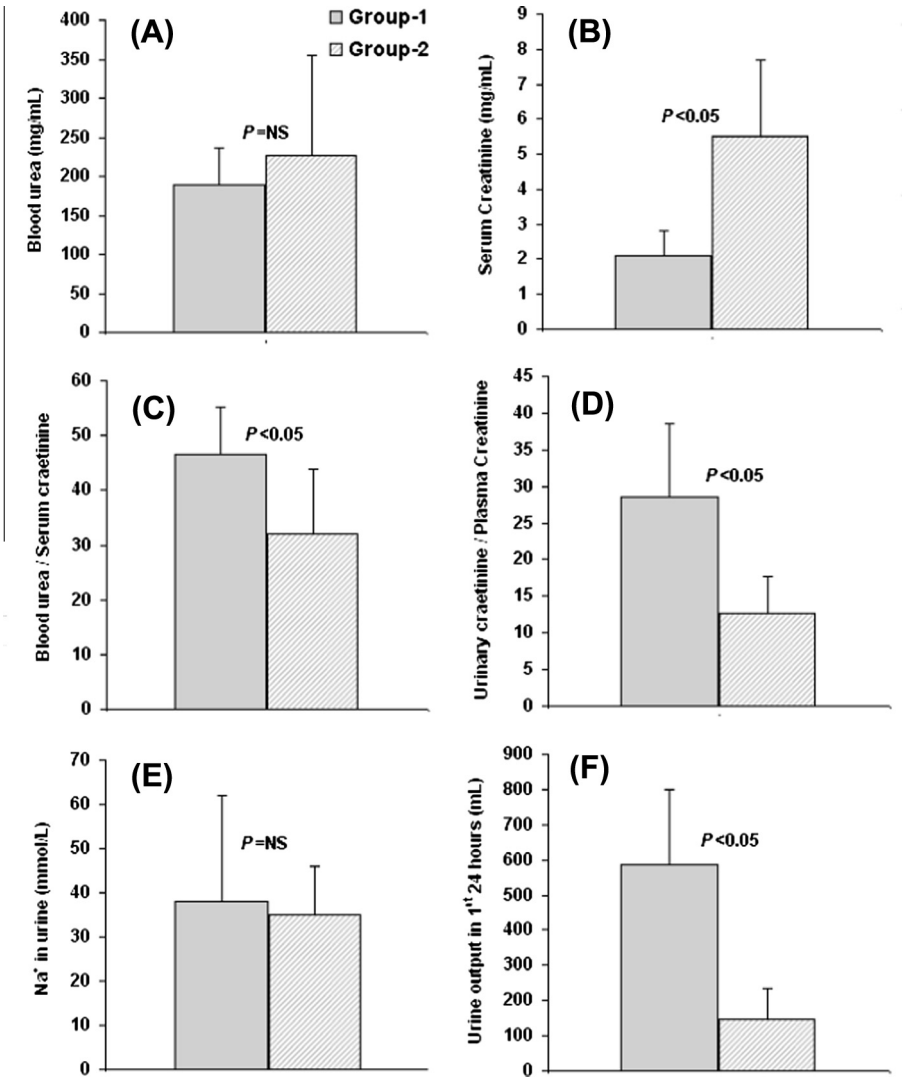

Figure 1 The renal function tests: Panel (A) Blood urea, (B) Serum creatinine, (C) Blood urea/serum creatinine ratio, (D) Urinary creatinine/plasma creatinine, (E) Na^+ in urine and (F) Urine output in 1st 24 h.

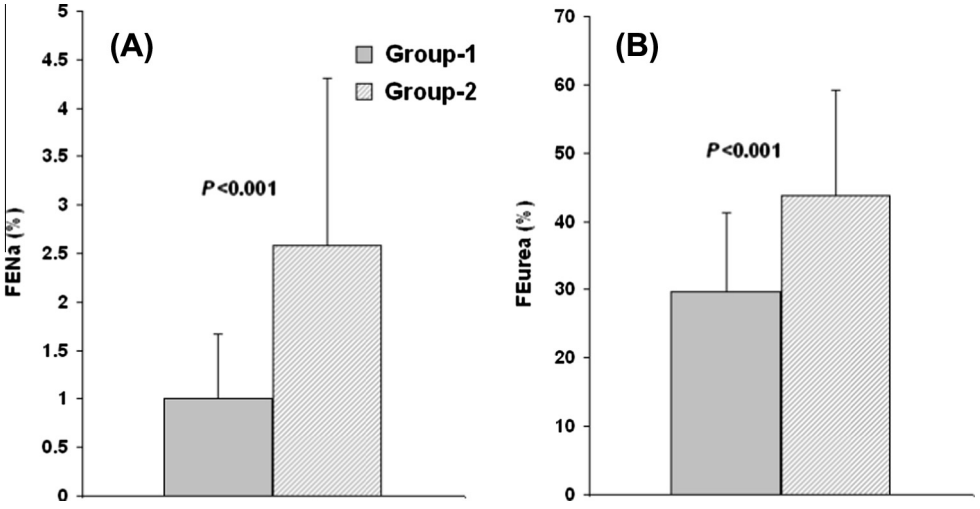

Figure 2 Comparison between prerenal (group-1) and renal (group-2) azotemia using FENa (panel A) and FEurea (panel B).

Table 4 Effect of increased severity of AKI on FENa and FEurea values using one-way ANOVA.

	FENa (%)	FEurea (%)
RIFLE-R	1.1 ± 0.3	31.4 ± 2.3
RIFLE- I	1.3 ± 0.5	35.4 ± 3.6
RIFLE-F	2.3 ± 0.4	41.4 ± 5.6
P value	<0.05	<0.05

AKI: Acute kidney injury; RIFLE: Risk, Injury, Failure, Loss and End-stage Kidney; R: Risk; I: Injury; F: Failure.

correlation could be detected between FENa and FEurea values ($r = 57$, $P < 0.0010$ (Fig. 3).

Compared to FENa, on using FEurea as a predictor in differentiating renal from prerenal azotemia in all patients, higher sensitivity (78.6%), specificity (88.5%) and overall accuracy (85%) could be detected (Fig. 4A), than FENa; sensitivity (71.4%), specificity (69.4%) and overall accuracy (67.5%) (Fig. 4B).

For patients who received diuretics, FEurea as a predictor also could differentiate renal from prerenal azotemia (group-1b) with higher sensitivity (78.4%), specificity (92.1%) and overall accuracy (85.7%) (Fig. 4C), than FENa; sensitivity (64.3%), specificity (58.4%) and overall accuracy (59.4%) (Fig. 4D).

As a predictor of differentiation, the cutoff value of FEurea was 34.12% ($P < 0.001$) and the cutoff value of FENa was 1.1% ($P < 0.05$).

Discussion

Fractional excretion of sodium (FENa) has been routinely used for differentiating renal from prerenal azotemia [14]. However, the values of FENa are usually affected by the commonly used medications as diuretics, dopamine and norepinephrine, and also by some clinical conditions as

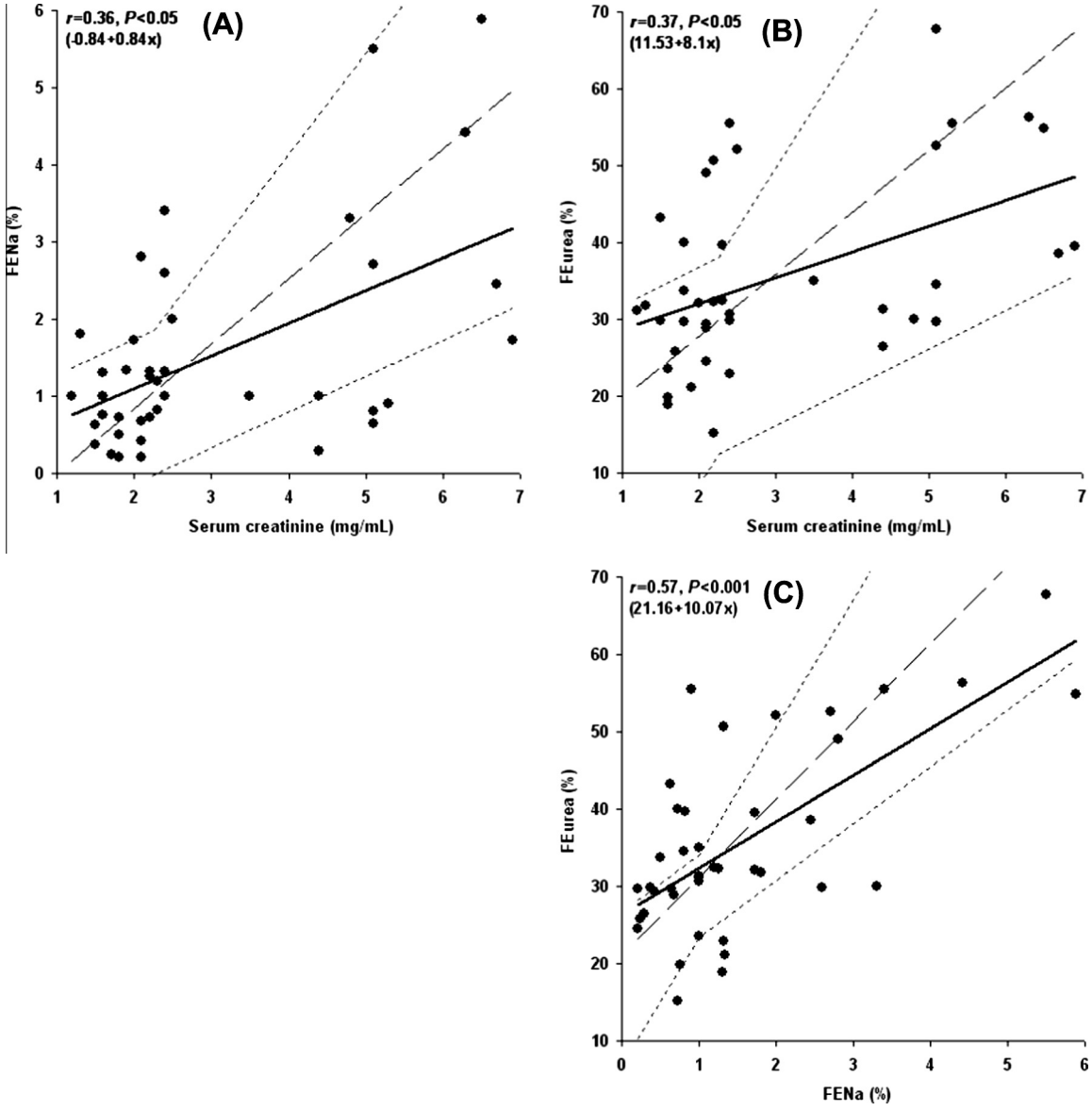

Figure 3 Regression plot with Passing and Bablok fit for: Panel (A) Serum creatinine vs. FENa, (B) Serum creatinine vs. FEurea and (C) FENa vs. FEurea. The correlations were good. (—) Trendline, (—) Identity line and (. . .) Passing and Bablok fit lines.

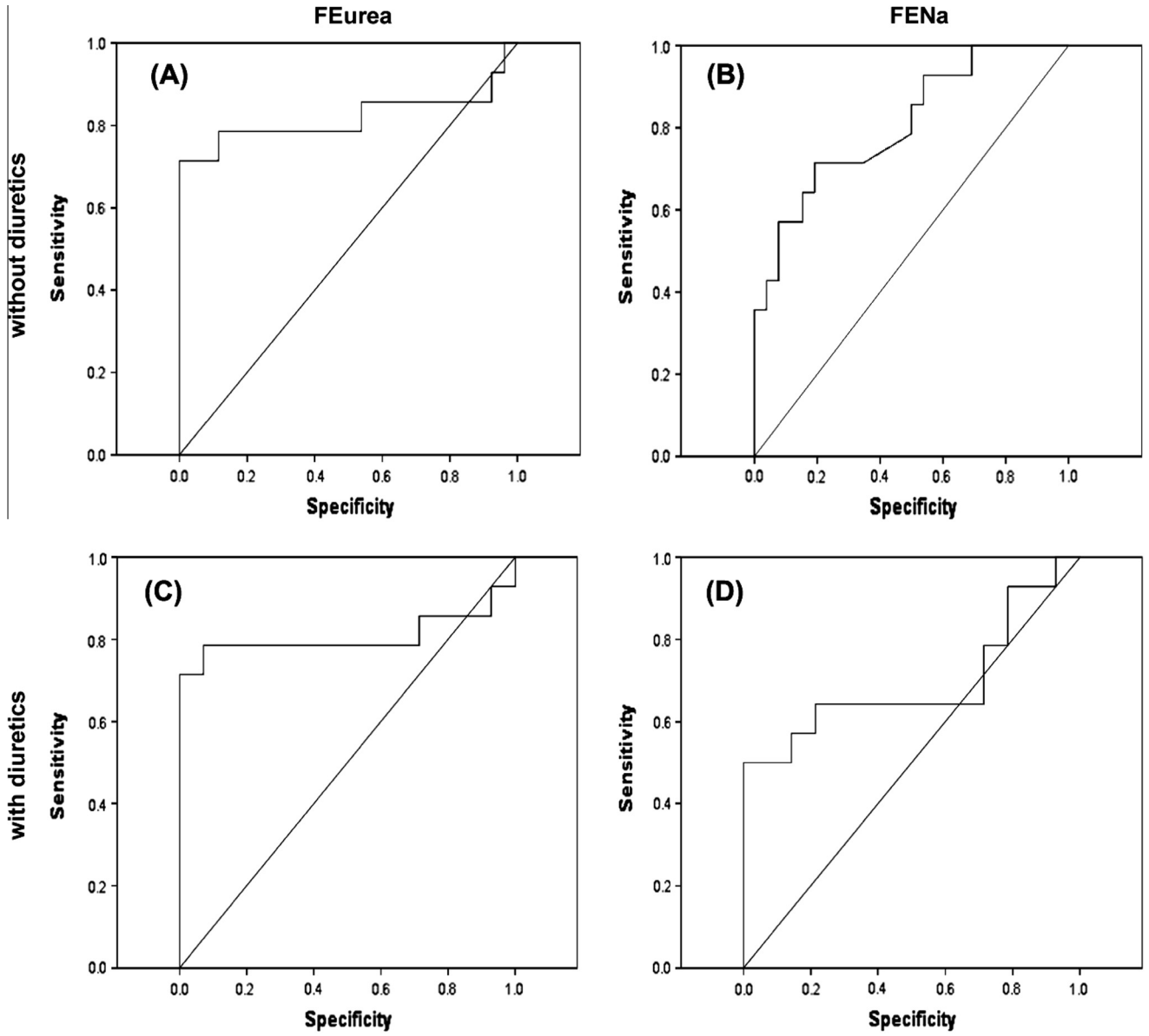

Figure 4 ROC curves of (A) FEurea to differentiate renal (group-2) from prerenal azotemia (group-1), (B) FENa to differentiate renal (group-2) from prerenal azotemia (group-1), (C) FEurea to differentiate renal (group-2) from prerenal azotemia in patients using diuretics (group-1b) and (D) FENa to differentiate renal (group-2) from prerenal azotemia in patients using diuretics (group-1b).

myoglobinuria, radiocontrast induced renal failure and different causes of metabolic acidosis [14]. On the contrary, the fractional excretion of urea (FEurea) is suggested to be used instead of FENa with better reliability to differentiate between prerenal from renal azotemia, as its values are not affected by the same drugs or medical conditions [1].

In our study we performed a prospective observation in a general ICU on patients with acute kidney injury (AKI) complicating circulatory shock, to explore the difference between using both FENa and FEurea in differentiating renal from prerenal azotemia, and up to our knowledge this is the first time to perform such a comparison in patients with AKI complicating circulatory shock in which 68% of them needed dopamine and norepinephrine together in their ICU medication, making the identification of the effect of each drug alone on FENa and FEurea statistically difficult.

In this study, we used the RIFLE-criteria to classify our patients with AKI into mild renal affection (RIFLE-R), moderate renal affection (RIFLE-I) and severe renal affection (RIFLE-F). We had detected significantly incremental values of both FENa and FEurea with the higher class of the RIFLE-criteria, and to our knowledge, it is the first time to compare FENa and FEurea versus the RIFLE-criteria.

Our study showed a fair correlation between the routine serum creatinine marker and both FENa ($r = 0.36$, $P < 0.05$), and FEurea ($r = 0.37$, $P < 0.05$), respectively. The poor correlations can be explained by the serum creatinine increase in both renal and prerenal azotemia, while FENa and FEurea increase in renal azotemia and decrease in prerenal azotemia [7]. These findings agree with the Carvounis study, in which there were fair correlations between serum creatinine and both FENa and FEurea [7].

Also, we found significantly higher values of both FENa and FEurea in patients with renal azotemia than those with prerenal azotemia ($P < 0.05$ and $P < 0.001$, respectively). These also agree with Carvounis et al., who studied 102 patients with AKI; 76% with prerenal azotemia showed significantly lower FENa and lower FEurea values ($P < 0.001$), while 24% of patients with renal azotemia showed significantly higher FENa and higher FEurea values ($P < 0.001$) [7].

It is important to mention here that good correlation could be detected between FENa and FEurea ($r = 0.67$, $P < 0.05$) in our study. This finding supports the trend of combining both FENa and FEurea in one panel; "the panel of fractional excretion of solutes" that includes FENa and FEurea which can provide the most important insight into the differentiation between renal and prerenal azotemia in various clinical states as suggested by Diskin [15]. However, our study showed that FEurea had better sensitivity (78.1% vs. 71.4%), specificity (88.5% vs. 69.4%) and overall accuracy (85% vs. 67.5%) than FENa in differentiating renal from prerenal azotemia.

Those findings agree with Dewitte et al., in which FEurea had better sensitivity (83% vs. 49%) and specificity (75% vs. 71%) than FENa in differentiating renal from prerenal azotemia [16]. But disagree with Pépin et al., in which 99 patients with AKI showed that sensitivity and specificity of FEurea were 79% and 33%, and concluded that FEurea could not be used as an alternative tool in differentiating renal from prerenal azotemia as it lacks reasonable specificity [17]. The point of difference from our study is that they mainly included patients with preexisting chronic kidney disease (40% of their population) without correction of the FEurea values as described by Nguyen et al., to compensate for the effect of the chronic kidney disease on the FEurea value [18], while in our study all patients with chronic kidney disease were excluded.

In our study FENa had a cutoff point of 1.1% for differentiating renal from prerenal azotemia with reasonable sensitivity and specificity (71.4% and 69.4%, respectively). These agree with Carvounis et al., who had a cutoff point of 1% with high sensitivity and specificity (91% and 82%, respectively) [7]. However, the relatively lower sensitivity and specificity of our FENa values can be attributed to; all our patients who had circulatory shock with lactic acidosis and both dopamine and noradrenaline infusion, which were additional factors that led to increase in the false FENa values.

In our study, FEurea had a cutoff value at 34.1% for differentiating renal from prerenal azotemia with a sensitivity and specificity of 78.1% and 88.5%, respectively. This agrees with Carvounis et al., which had a cutoff point of 35% with sensitivity and specificity of 85%, 92%, respectively [7]. But disagrees with Dewitte et al., who studied 47 ICU patients with AKI, in which the best cutoff point of FEurea was 40% [16]; a level which is higher than ours. This can be explained as the critically ill patients, as in Dewitte et al. study, commonly have hypercatabolic disease, which may increase urea production and excretion thereby increasing FEurea [16]. The point of difference from our study is that, all our patients had circulatory shock with decreased tissue perfusion which affects the catabolic state of the tissues making FEurea cutoff point in our study slightly lower.

In our study, FEurea was almost not affected by the use of frusemide diuretic therapy; sensitivity, specificity and overall accuracy were 78%, 92% and 85% and 78%, 88% and 85%, respectively without diuretics. The use of frusemide obviously affected FENa values and caused a decrease in the sensitivity (64% vs. 71%), specificity (58% vs. 70%) and overall accuracy (59% vs. 69%), respectively. Those findings agree with Carvounis et al., in which the sensitivity of FEurea was almost not affected by the use of diuretics (92% vs. 91%) while that of FENa was markedly decreased with the use of diuretics (92% vs. 52%). But disagree with Darmon et al., who concluded that FEurea may be of little help in distinguishing renal from prerenal azotemia in critically ill patients receiving diuretic therapy, however, they stated that few of their patients received diuretics and poor performance of the urinary indices was therefore related to low statistical power [19].

The mortality rate in our study was significantly higher in renal azotemia (50%) than in prerenal azotemia (19.2%), ($P < 0.05$). This is supported by Vincent et al. and by Angus et al., who found that the mortality rate was about 20% in single organ failure, 40% in two organ failure, 70% in three organ failure and reaching 90% in four organ failure [20,21].

The cutoff points of both FENa and FEurea as a predictor of mortality were not statistically justified. This is explained as all our patients had circulatory shock, and so patients with mild renal impairment may die from their severe shock state, and patients with severe renal affection may survive if their shock could be rapidly corrected. This cutoff point calculation is recommended only in patients with AKI without other organ affections or shock that may affect the mortality.

Limitations

The limited number of our patients made it difficult to perform reliable statistical tests to explore the relations between both FENa and FEurea vs. different types of shocks, which would have added a lot of information to our study.

Conclusion

Although both fractional excretion of urea (FEurea) and fractional excretion of sodium (FENa) are feasible, reproducible, and inexpensive markers used in differentiating renal from prerenal azotemia, in our study FEurea showed higher sensitivity and specificity than FENa, not only in differentiating renal from prerenal azotemia in critically ill patients complicating circulatory shock, but also its values were not affected by the use of diuretics like FENa in the same group of patients.

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