CRITICAL CARE

LETTER

Urine biochemistry in acute kidney injury: are we moving in the right direction?

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See related research by Pons *et al.*, http://ccforum.com/content/17/2/R56

We read with interest the article by Pons and colleagues [1] in a recent issue of *Critical Care* and we would like to ask them a few questions. They excluded patients 'for whom urine could not be collected according to the study protocol' [1]. What were the reasons for this - anuria, renal replacement therapy (RRT), ICU discharge or death? The authors said that there was no patient receiving RRT at the time of the study, but then they mentioned 14 patients requiring RRT in the first 24 hours. How many patients were excluded due to less than 72 hours of follow-up? Since acute kidney injury (AKI) diagnosis was mandatorily done on admission in order to define AKI reversal in the first 3 ICU days, what about patients who were 'no-AKI' on admission but developed AKI in these 3 days?

The authors evaluated urinary indices which are actually calculated variables, dependent on multiple measured parameters. In a pilot study [2], we demonstrated that urinary sodium (NaU) was lower on admission in patients who developed AKI in the first 4 ICU days. We believe that sequential NaU measurement is useful, especially in the absence of diuretics, and that early AKI development is characterized by decreases in NaU which may precede increases in creatinine, in both transient and persistent AKI (unpublished data). These findings suggest that transient and persistent AKI are different magnitudes of the same pathophysiological process and not synonyms of functional/structural AKI [3]. This may partially explain the absence of discernment ability of the urinary indices. What was the time course of NaU in your three groups in the absence of diuretics? We believe that there is a role for urine biochemistry in AKI assessment and to exclude it from daily practice is the wrong direction.

Authors' response

Bertrand Pons and Michael Darmon

We thank Maciel and Vitorio for their comments and appreciate their interest in our work. We enrolled 202 patients, and 58 were excluded as consequences of missing urine samples or an early ICU discharge precluding patient classification.

Patients without AKI at the time of urine collection were considered 'no AKI' even if they developed AKI after day 1. We fully agree that the rise in creatinine is delayed following renal injury and that some patients therefore might have been misclassified [4]. However, AKI was defined according to the Acute Kidney Injury Network criteria of both urine output and serum creatinine, and this may have limited the risk of patient misclassification [5,6].

Overall, 20 patients (21%) without AKI at inclusion developed AKI between days 1 and 3. These patients had similar NaU concentration at inclusion to patients without AKI from inclusion to day 3: 77 mmol/L (40 to 120) versus 79 mmol/L (34 to 110) ($P = 0.79$). Similarly, fractional excretion of urea and fractional excretion of sodium or other urinary indices in this subgroup of patients were similar to those of patients without AKI from inclusion to day 3.

Lastly, NaU concentration at inclusion was similar across patient groups - 72 mmol/L (38 to 113) in patients without AKI, 55 mmol/L (24 to 84) in patients with transient AKI, and 64 mmol/L (35 to 99) in patients

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with persistent AKI at inclusion ($P = 0.07$) - or at later time points.

We agree with Maciel and Vitorio that further research in this field is welcome. However, the evidence suggests that urinary biochemistry indices should no longer be recommended routinely [1,7].

Abbreviations

AKI: Acute kidney injury; NaU: Urinary sodium; RRT: Renal replacement therapy.

Competing interests

The authors declare that they have no competing interests.

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