Letter and Reply

Urine output before the initiation of CRRT and mortality

To the Editor,

The incidence of acute kidney injury (AKI) is still increasing, and severe AKI requiring renal replacement therapy is associated with high mortality rates despite advances in medical treatment. In hemodynamically unstable patients, continuous renal replacement therapy (CRRT) is tolerated better than intermittent hemodialysis; however, the mortality rate has not been reduced. Therefore predicting the prognosis of AKI patients requiring CRRT is important, as are efforts to reduce mortality rates in AKI patients with multiorgan failure and septic shock. The prognostic factors associated with mortality from severe AKI have been identified as the severity of comorbid conditions, hemodynamic instability, and the time of initiation of CRRT [1–4].

Oh et al [5] retrospectively analyzed predictors of 90-day mortality in 67 hemodynamically unstable patients with AKI who required CRRT. They demonstrated that a urine output of less than 500 mL for 12 hours before the initiation of CRRT was the only independent predictor. This is consistent with a previous report that showed low urine output before the initiation of CRRT to be associated with mortality [2]. As the urine output during a certain period (6 or 12 hours) reflects the severity of the patient’s condition more accurately than blood pressure or laboratory parameters at one time point, we agree with the authors that urine output during a certain period can be a superior predictor to previously known factors. Moreover, urine output during the first several hours may also reflect whether the patient has responded to fluid therapy before the initiation of CRRT, and therefore may also reflect the recovery of the hemodynamic instability and kidney injury.

However, we have several concerns about this study. First, the authors defined hypotension as an initial mean arterial pressure less than 60 mmHg (about 80/50 mmHg), which is stricter than the definition of hypotension generally used—less than 100/60 mmHg. In clinical practice, CRRT cannot be started in patients with a blood pressure less than 80/50 mmHg or in those who do not respond to vasopressor treatment. Therefore factors that reflect the response to fluid therapy in the early period of CRRT treatment, such as urine output, may be more reliable than blood pressure at one time point. The authors did not analyze whether the initial blood pressure was different between the survivors and nonsurvivors, probably because all the patients were hemodynamically unstable. We suggest that the mean blood pressure during the initial fluid treatment or the response to vasopressor therapy before the initiation of CRRT could be also a reliable parameter, in addition to urine output.

Second, the authors defined the decrease in urine output before the initiation of CRRT as a urine output of less than 500 mL over 12 hours. In clinical practice, patients often undergo CRRT less than 12 hours after arrival in the emergency room. Therefore it would be better to define the period of a decrease in urine output by a minimally required period. In this study, it was not clearly described whether the included patients were those who started CRRT after the initial arrival in the emergency room.

Kim et al [2] demonstrated the criteria for urine output as being those during a 6-, 12-, and 24-hour period, and in another report a urine output less than 30 mL/h was associated with mortality. In this study, a urine output less than 500 mL during 12 hours (41.6 mL/h) independently predicted mortality. We wonder whether the authors have analyzed the results with a criterion of urine output less than 30 mL/h (360 mL for 12 hours), as in previous reports. In addition, we wonder why the authors have made the criterion for decreased urine output in patients with severe hemodynamic instability less strict than that in previous reports that included patients with less severe hemodynamic instability.

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In Reply:

We appreciate these valuable inputs on our study. The first comment concerned the definition of hypotension used in our study. Hypotension is usually defined by a blood pressure of 100/60 mmHg. Shock is defined by a mean arterial blood pressure of less than 60 mmHg and inadequate tissue perfusion [1]. In the systolic blood pressure range of 80–150 mmHg, fluctuations in blood pressure have a marginal effect on renal blood flow and glomerular filtration rate because of renal autoregulation [2]. Autoregulation fails with a drop in blood pressure below 80 mmHg, which could result in ischemic injury [2]. Renal hemodynamics are also influenced by other neurohormonal factors including prostaglandins and angiotensin II under conditions of low renal perfusion [2]. Our definition for hypotension was more stringent, with an initial mean arterial pressure of less than 60 mmHg (approximately 80/50 mmHg). Therefore most patients in our study experienced disturbances in renal hemodynamics and were predisposed to developing oliguria.

We agree with suggestion 3rd that the response during initial fluid treatment or to vasopressor therapy before the initiation of continuous veno-venous hemodiafiltration (CVVHDF) could be a reliable parameter. Maintaining hemodynamic stability is important in the resuscitation of critically ill patients. In our study, most patients responded to initial fluid or vasopressor therapies, and underwent CVVHDF after the mean arterial blood pressure had reached over 70 mmHg.

In the second comment, it was mentioned that the period of a decrease in urine output is better defined by a minimally required period than by a urine output of less than 500 mL during 12 hours in an emergency room setting. In our study, the enrolled patients experienced severe hemodynamic instability. Therefore the recovery of hemodynamic instability, insertion of a dialysis catheter, and start of CVVHDF took more than 12 hours in inpatient or emergency room settings.

Since both hourly urine output and duration of oliguria are included in the urine output criteria of RIFLE, we adopted a urine output of 500 mL over 12 hours [3]. We observed similar results by measuring a urine output of less than 400 mL during 12 hours, but we have not included these results in our paper. Since the injury group is defined by a urine output of less than 0.5 mL/kg/h during 12 hours (approximately 300–400 mL during 12 hours) according to the RIFLE criteria, we used a less stringent criteria of a urine output of less than 500 mL over 12 hours to include those patients with less severe acute kidney injury.

Conflicts of interest

All contributing authors declare no conflict of interest.

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