LETTER TO THE EDITOR

Urine sodium profile in the course of septic acute kidney injury: insights relevant for kidney function monitoring

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Dear Editor,

Current literature has excluded urine sodium (NaU) from acute kidney injury (AKI) management, especially in sepsis.[1] We have recently questioned this paradigm.[2] There is no recent clinical study which has evaluated this parameter sequentially in the course of septic AKI. We therefore would like to present an example of the NaU profile (Figure 1A) measured once daily (spot urine sample) in a 30-day period which has included AKI development, renal replacement therapy (RRT), until complete renal recovery. The patient was a 68-year-old man admitted in our ICU in the immediate postoperative period (D0) after an urgent right hemicolectomy due to intestinal obstruction. 5 liters of crystalloids were infused in the operating room due to hemodynamic instability and the patient was admitted with oliguria, severe hypoxemia with extensive lung infiltrates and an already increased serum creatinine (Figure 1B). Furosemide was used early in his management (including intra-operatively) in order to improve ventilation and maintain adequate urine output. In the first 3 days NaU had apparently random values (Figure 1A) but actually NaU values were low, increasing only soon after furosemide administration. Serum creatinine remained increasing daily between D3 and D5, coinciding with daily and consistent decreases in NaU value and urine output, even with furosemide administration. Significant hemodynamic instability was present and blood pressure was maintained with norepinephrine infusion. At D5, continuous RRT (CRRT) was then initiated. We decided to keep collecting daily NaU even during CRRT since there was no complete anuria. It is noteworthy that NaU values continued to decrease during all the RRT period and remained decreasing even after renal function has started to recover around D17 (Figure 1A, B). Progressive increases in NaU only occurred when serum creatinine was close to normal values and systemic inflammation was solved (near zero value of c-reactive protein in blood, Figure 1A). Furosemide was administered at D9, D10 and D15 with no significant repercussion in NaU profile.

In this case, NaU profile highlights some relevant points to our understanding and monitoring of AKI: first, early AKI development may be characterized by decreased NaU values which can be "artificially" increased with the addition of diuretics, as occurs with urine output; second, low NaU values in the immediate postoperative period could be a sign of low effective circulating volume but severe hypoxemia and extensive lung infiltrates after massive fluid resuscitation intra-operatively have precluded additional fluid challenge - we believe that low NaU in this context was mainly due to bacterial translocation and sepsis; third, NaU and fractional excretions of sodium and urea (Figure 1C) seem to behave as in a progressively more severe "pre-renal", avid-sodium retaining state, at least in sepsis. This condition may be due to microcirculatory impairment in the kidneys even in the presence of an increased total renal blood flow.[3] Interestingly, a preserved capacity of the tubules to retain sodium is possible until advanced stages of AKI. This is against the paradigm of persistent AKI as a predominantly structural AKI.[2, 4] In addition, RRT does not seem to interfere in the NaU profile which seems to have its own behavior. Importantly, NaU recovery has not occurred in parallel with serum creatinine or urine output. The early phase of renal recovery was characterized by a low-sodium urine, and re-incorporation of sodium into urine only occurred when systemic inflammatory process was solved and renal function was again close to normal. Of course, spot NaU interpretation is not always simple since, besides diuretic use, NaU depends on the amount of sodium received, regulation of effective circulating volume, etc. Moreover, the information obtained from the analysis of NaU in a 24h collected urine as compared to the analysis from a random urine sample may be greatly different. We must emphasize, however, that the aspect of the spot NaU curve during AKI development and recovery was so that it seems to give us a good notion of overall NaU profile. If there was a great fluctuation of NaU in the 24h period, we would expect a more oscillating, "up and down" val-

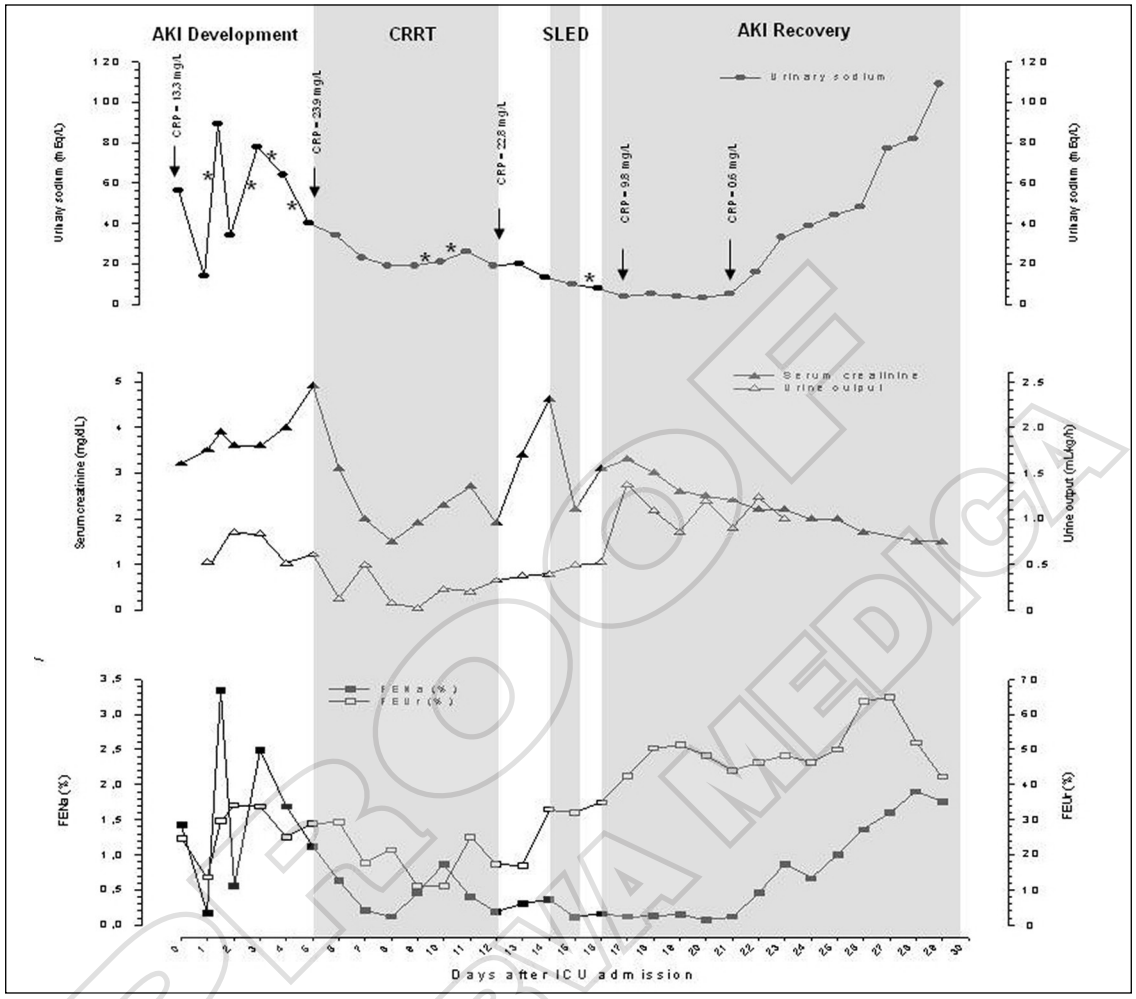

Figure 1.—Thirty-day time course of daily spot urine sodium (A), urine output and serum creatinine (B), and fractional excretions of sodium (FENa) and urea (FEUr) (C) in all phases of septic acute kidney injury (AKI) development and recovery. CRRT: continuous renal replacement therapy SLED: sustained low efficiency dialysis CRP: c-reactive protein.
*intervals in which furosemide was administered. The respective dosages of furosemide were 80mg, 20 mg, 60 mg, 40 mg, 40 mg, 40 mg and 20 mg in each interval.

ues of spot NaU, which has only occurred in the first 2 days and clearly related to diuretic use. We then suggest that NaU, even in spot samples, may be useful in AKI monitoring and its particular physiology in septic AKI should be more extensively studied.

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Conflicts of interest.—The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Received on July 4, 2013. - Accepted for publication on October 10, 2013.

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