CASE REPORT

Early diagnosis of acute kidney injury in a critically ill patient using a combination of blood and urinary physicochemical parameters

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INTRODUCTION

Acute kidney injury (AKI) is common among critically ill patients. Because it is associated with high rates of morbidity and mortality, efforts are being made to identify AKI early to increase the likelihood of successful intervention. Serum creatinine, the primary parameter used to diagnose AKI, is universally considered a late marker.

One of the major problems associated with AKI is acid-base disturbance. The physicochemical approach proposed by Stewart (1) is increasingly recognized as useful in managing complex acid-base disturbances. In this case, we demonstrate that, during the development of AKI, evolutive alterations in physicochemical serum and urinary variables are easily measured and may reliably precede increases in creatinine. These parameters may thus aid in the early diagnosis of AKI.

CASE DESCRIPTION

A 45-year-old male was admitted to our intensive care unit (ICU) from the ward, where he was concluding antibiotic treatment for a urinary tract infection and being treated with oral anticoagulants for antiphospholipid syndrome. On the day of ICU admission (day 0), the patient was found unconscious on the floor of the ward. A cranial CT scan revealed an acute subdural hematoma. The patient was immediately transferred to the ICU and intubated. The anticoagulant was reversed with vitamin K and plasma. Soon after, the patient was transferred to the operating room, where the hematoma was drained and an intracranial pressure (ICP) monitoring device was installed. After the surgery, the patient returned to the ICU. Because of his elevated ICP, the patient was kept sedated with thiopental. Norepinephrine was infused to maintain an adequate cerebral-perfusion pressure. Initial exams after surgery (day 1) revealed normal blood urea nitrogen (BUN) and creatinine levels and increased levels of serum Na⁺ and Cl⁻ (Table 1).

Blood gas analysis revealed a mild non-anion gap metabolic acidosis. A simultaneous spot sample of urine also revealed high levels of Na⁺ (NaU) and Cl⁻ (ClU) as well as a high urinary strong ion difference (SIDu = [Na⁺] + [K⁺] - [Cl⁻]). By day 2, BUN and creatinine levels had decreased slightly. Urine output over the previous 24 h had been normal. Serum and urine levels of Na⁺ and Cl⁻ remained elevated, and the SIDu remained high, but the blood gas analysis was normal. The ICP was controlled; thiopental was replaced by dexmedetomidine, and the dose of norepinephrine was reduced. By day 3, BUN and creatinine levels had increased only minimally. Urine output had decreased over the previous 24 h but did not fall below the normal range (Table 1). Serum Na⁺ and Cl⁻ levels were evolutive similar, while NaU and ClU levels remained high. A significant increase in SIDu was observed, which resulted from increases in NaU and K⁺ (KU) levels and decreases in ClU levels (Figure 1). Sedation was stopped, but norepinephrine was still necessary to maintain the patient’s mean blood pressure above 65 mmHg. C-reactive protein levels and the degree of leukocytosis increased daily, and cultures were collected. On day 4, the patient developed tachycardia, and urinary flow continued to decrease, while norepinephrine levels increased to maintain the mean blood pressure. There was no fever. Treatment with broad-spectrum antibiotics was initiated empirically. During this time, creatinine levels had not increased, but the BUN level increased slowly each day to levels that did not exceed the normal range. Serum phosphate and unmeasured anions (Table 1) also increased daily. Serum Na⁺ and Cl⁻ were still high, but NaU and ClU levels decreased; KU levels increased daily from day 2 onward. On day 5, the patient developed clear oliguria, although the creatinine measurements performed at the beginning of the day were normal. The BUN level continued to increase. An abrupt decrease in NaU and ClU levels occurred in parallel with the increases in KU levels. The SIDu decreased but remained very high. On day 6, the patient developed fever, severe metabolic acidosis, and refractory shock with overt multiple-organ failure. NaU and ClU levels fell further, but the KU and SIDu levels remained high. The patient’s creatinine levels increased abruptly, and the nephrology team indicated the need for continuous renal-replacement therapy; however, the patient died before therapy could be initiated. None of the cultures collected revealed the presence of an infectious agent.

DISCUSSION

The case described above is a common example of AKI (possibly septic in origin), and it demonstrates that creatinine
Table 1 - Traditional renal and physicochemical parameters in the evolution of AKI.

|                  | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 |
|------------------|-------|-------|-------|-------|-------|-------|
| BUN (mg/dL)      | 15    | 10    | 13    | 17    | 27    | 47    |
| Creatinine (mg/dL) | 0.89  | 0.76  | 0.90  | 0.87  | 0.95  | 2.14  |
| SIDa (mEq/L)     | 34.5  | 37.1  | 39.5  | 39.8  | 38.4  | 40.0  |
| SIG (mEq/L) *    | 1.7   | 2.6   | 4.5   | 7.0   | 9.1   | 14.2  |
| Lactate (mEq/L)  | 0.9   | 2.1   | 3.1   | 2.6   | 3.2   | 6.4   |
| Phosphate (mg/dL) | 3.0   | 1.8   | 2.1   | 3.1   | 3.8   | 5.5   |
| FeNa (%)         | 1.5   | 4.7   | 1.8   | 0.9   | 0.1   | 0.1   |
| FeCl (%)         | 1.9   | 5.2   | 1.6   | 0.7   | 0.3   | 0.2   |
| FeK (%)          | 17.2  | 19.4  | 17.6  | 15.3  | 25.1  | 39.6  |
| Preceding 24-h diuresis (mL) | --- | 2440 | 1100 | 920 | 840 | 225 |

AKI: acute kidney injury; BUN: blood urea nitrogen; SIDa: apparent strong ion difference; SIG: strong ion gap (unmeasured anions); FeNa, FeCl, and FeK: fractional excretion of sodium, chloride and potassium, respectively.

* measured in blood.

Table 1 demonstrates the evolution of AKI through changes in traditional renal and physicochemical parameters. The table shows how these parameters change over time, which can be indicative of the progression of AKI.

The figure illustrates the changes in electrolyte levels in spot urine samples over time in the ICU setting. The graph is labeled as follows: Sodium, Chloride, Potassium.

Figure 1 - Daily changes in electrolyte levels in spot urine samples.

In conclusion, the daily evaluation of changes in simple parameters such as consistent increases in serum phosphate levels, unmeasured anions, and KU, with decreases in NaU and CIU, and a persistently high SIDu, could facilitate the early diagnosis of renal impairment before major decreases in urine output or increases in creatinine are observed. Even small alterations in these parameters might be a warning sign of ongoing renal and perhaps systemic hemodynamic impairment.

AUTHOR CONTRIBUTIONS

Maciel AT provided care to the patient, collected and analyzed the data, and wrote the manuscript. Park M provided care to the patient and collected the data.

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