

## Steep elevation of creatinine in a patient with acute flank pain

Sunday, 01 July 2007

A 46-year-old, Caucasian, male patient presented to the emergency department suffering, for 12 hours, from acute, severe, left flank pain radiating to the left lower quadrant, with associated emesis. The patient reported minimal urine output after symptoms had started. Past medical history was unremarkable, and previous routine laboratory examinations had shown only chronic mild hyperuricemia.

The physical examination revealed mild tachypnea and left costovertebral angle tenderness, but was otherwise normal. On admission, abnormal laboratory findings included leukocytosis (17.220 white blood cells per mm<sup>3</sup>), a urinalysis showing hematuria (>100 red blood cells/high power field) and pyuria (20-22 white blood cells/high power field), and elevated values of serum creatinine (sCr) and blood urea nitrogen (BUN) (2,4 and 34 mg/dl, respectively).

What is the cause of the renal dysfunction?

### Steep elevation of creatinine in a patient with acute flank pain

A 46-year-old, Caucasian, male patient presented to the emergency department suffering, for 12 hours, from acute, severe, left flank pain radiating to the left lower quadrant, with associated emesis. The patient reported minimal urine output after symptoms had started. Past medical history was unremarkable, and previous routine laboratory examinations had shown only chronic mild hyperuricemia.

The physical examination revealed mild tachypnea and left costovertebral angle tenderness, but was otherwise normal. On admission, abnormal laboratory findings included leukocytosis (17.220 white blood cells per mm<sup>3</sup>), a urinalysis showing hematuria (>100 red blood cells/high power field) and pyuria (20-22 white blood cells/high power field), and elevated values of serum creatinine (sCr) and blood urea nitrogen (BUN) (2,4 and 34 mg/dl, respectively).

What is the cause of the renal dysfunction?

### Diagnosis

Renal ultrasonography documented a hypertrophic left kidney (major diameter of 16,8 cm), with mild dilatation of the renal pelvis; the right kidney could not be visualized and the urinary bladder was almost empty of urine.

Based on the above findings, a diagnosis of acute urinary tract obstruction was suspected, being one of the most frequent causes of anuria (1). Although in our case there were only minimal signs of hydronephrosis, it has been shown that in acute urinary tract obstruction the collecting system may be relatively incompressible (2).

Treatment had commenced with hydration by intravenously administered fluids at a rate of 3 l/day, analgesics in the form of diclofenac which was discontinued to prevent any further renal damage and was substituted by pethidine, and antimicrobial therapy with ciprofloxacin plus amoxicillin/clavulanate for the possibility of concomitant complicated infection. However, the patient remained anuric and the pain was not alleviated. Thirty-one hours after admission sCr and BUN rose to 7,0 and 63 mg/dl, respectively.

The diagnosis of urinary tract obstruction was confirmed by an abdominal CT scan, which revealed a 6 mm calculus located in the left ureterovesical junction along with minimal dilatation of the left pelvicalyceal system, and a small, atrophic, right kidney (Figure 1). To relieve the obstruction ureteroscopy was performed, during which the stone was removed and a ureteral stent was placed to allow for drainage of residual stone fragments.

After the intervention, massive diuresis was noted and 15 hours later sCr and BUN fell to 3,1 and 48 mg/dl, respectively. At discharge, 3 days after admission, sCr and BUN values had nearly normalized (1,4 and 26 mg/dl, respectively) (Figure 2).

### Teaching points

- In the case we report, excessive fluctuations in serum creatinine and BUN values were noted in a male, middle-aged patient with acute anuric renal failure, caused by unilateral urinary tract obstruction of a solitary functioning kidney. The measured rate of rise of the patient's serum creatinine was 4,6 mg/dl in a 31 hours interval, which corresponds to a rise of 3,6 mg/dl per day. Although our measurements were not repeated, so as to control for possible technical inaccuracies, the fact that BUN values exhibited analogous variations to those of serum creatinine, strengthens the validity of our measurements (Figure 2).

- In contrast to our observation, it has generally been regarded, that in acute renal failure serum creatinine typically rises with a rate of up to 1-1,5 mg/dl per day (1, 3). However, there are various factors that may influence the concentration of serum creatinine, generally by affecting the rate of generation of creatinine, its volume of distribution and the rate of its

renal excretion, as analyzed below (4). First, the rate of generation of creatinine reflects total muscle mass and the metabolic state. So, in cases of acute renal failure due to rhabdomyolysis, in which generation of creatinine is greatly increased, the rate of rise of creatinine can reach values of 2-2,5 mg/dl per day (5). Also, intake of exogenous creatinine by meat consumption can cause transiently an appreciable rise in serum creatinine concentration. Regarding the volume of distribution of creatinine, which is practically the volume of total body water, when it is decreased, as in cases of dehydration, values of serum creatinine concentration may appear higher. As for the rate of tubular secretion of creatinine, it can be reduced by some drugs, mainly cimetidine, trimethoprim and salicylates (6). Moreover, in cases of greatly reduced urine flow, tubular reabsorption of creatinine might ensue from back diffusion into the blood compartment. Finally, it should be mentioned, that depending on the assay used, various drugs, such as cefoxitine or flucytosine, or organic substances, such as acetoacetic acid, can interact in the measurement of creatinine.

- The case of acute renal failure caused by urinary tract obstruction that we report, shows that a greater than typically expected rate of elevation of serum creatinine concentration may be observed, possibly due to an interplay of the various known factors or even of other not still documented ones, that can influence serum creatinine values.

#### Acknowledgments

The case of steep elevation of creatinine in a patient with acute flank pain in acute urinary obstruction was prepared by Drs. G. Peppas, KG. Kiriakidou, and DE. Karageorgopoulos; it was submitted for consideration for publication.

#### References

1. Singri N, Ahya SN, Levin ML. Acute renal failure. *JAMA*. 2003;289:747-51.
2. Platt JF, Rubin JM, Ellis JH. Acute renal obstruction: evaluation with intrarenal duplex Doppler and conventional US. *Radiology*. 1993;186:685-8.
3. Moran SM, Myers BD. Course of acute renal failure studied by a model of creatinine kinetics. *Kidney Int*. 1985;27:928-37.
4. Perrone RD, Madias NE, Levey AS. Serum creatinine as an index of renal function: new insights into old concepts. *Clin Chem*. 1992;38:1933-53.
5. Grossman RA, Hamilton RW, Morse BM, Penn AS, Goldberg M. Nontraumatic rhabdomyolysis and acute renal failure. *N Engl J Med*. 1974;291:807-11.
6. Andreev E, Koopman M, Arisz L. A rise in plasma creatinine that is not a sign of renal failure: which drugs can be responsible? *J Intern Med*. 1999;246:247-52.