Acute Renal Failure Due to Rhabdomyolysis in a Spastic Man

C.	Cem	Sungur
T.	Tekin	Akpolat
O.	Oktay	Oymak
M.	Murat	Colakoglu
U.	Unal	Yasavul
C.	Cetin	Turgan

Nephrology Unit, Hacettepe School of Medicine, Ankara, Turkey

Cem Sungur, MD, PK (PO Box) 272, TR-06693 Kavaklıdere-Ankara (Turkey)

Dear Sir,

Rhabdomyolysis is a well-known cause of acute renal failure. Renal vasoconstriction, toxic effects of myoglobin on renal tubular cells and tubular obstruction are suggested as the probable mechanisms leading to acute renal failure [1, 2]. The causes of acute rhabdomyolysis include a wide variety of etiologies ranging from excessive muscular activity to genetic disorders [3]. Here, we report a man with spastic cerebral palsy who developed acute renal failure after an accidental fall and vigorous attempts to attain his proper position. A 22-year-old spastic man was admitted to the emergency service after an accidental fall while alone at home. His intellectual capabilities were well-developed but there was no motor activity in his legs and he could not coordinate the motor activities of his arms. He had a neurogenic bladder but he could control his urination. He was alone and watching television when he fell from his wheelchair. He showed vigorous efforts to straighten up for 3 h but was unsuccessful. His initial complaints were pain in the right thoracoabdominal region but he developed nausea, vomiting and decline in urine output the next day. His physical examination in the emergency unit disclosed a thin man with superficial lesions and bruising involving the upper right quadrant of the abdomen. The results of the laboratory work-up were as follows: Hb 16.5 g/dl, Htc 47%, WBC $17.2 \times 10^9/\text{mm}^3$, Na 135 mEq/l, K 5.8 mEq/l, BUN 63 mg/dl, creatinine 4.3 mg/dl, uric acid 11.0 mg/dl, calcium 8.6 mg/dl, phosphorus 5.2 mg/dl, total CPK 170.410 U/l, AST 1,963 U/l, ALT 656 U/l, alkaline phosphatase 58 U/l, GGT 8 U/l, LDH 4301 U/l, total bilirubin 0.8 mg/dl, albumin 4.2 g/dl, PT 12 s (with a control of 12.3 s), blood pH 7.36. There was slight discoloration of the urine, and urinary pH was 5. There was 60 mg/l protein excretion, urine density was 1,014, and microscopic examination revealed 5 RBC and 6 WBC per high-power field with numerous urate crystals. Urinary sodium excretion was measured as 79.8 mEq/l. Ultrasonographic examination showed no evidence of stones, pelvicalyceal and/or ureteral dilatation, but 2 normally sized kidneys with enhanced echogenicity and diminished visualization of the corticomedullary junction.

The patient was admitted to the intensive care unit and was hydrated intravenously, and alkalization of the urine was undertaken. He responded promptly to this supportive treatment and an urine output of 3 liters day was achieved. His CPK, LDH and AST levels returned to normal values within 5 days. This recovery phase was not complicated by hyperkalemia or hypercalcemia. After full recovery of his renal functions, he was discharged from the hospital.

Our observations suggest that this patient developed acute renal failure due to nontraumatic rhabdomyolysis. The reason of muscular breakdown was his struggle to straighten up and extensive muscular activity during these attempts.

References

Honda N: Acute renal failure and rhabdomyolysis. *Kidney Int* 1983;23:888-898. Ward MM: Factors predictive of acute renal failure in rhabdomyolysis. *Arch Intern Med* 1988;148:1553-1557.

Gabow PA, Kaehny WD, Kelleher SD: The spectrum of rhabdomyolysis. *Medicine* 1982; 61:141-152.