Acute Renal Failure in Toxic Shock Syndrome Owing to Rhabdomyolysis

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ABSTRACT

Toxic shock syndrome is frequently complicated by renal dysfunction. The mechanism of this functional disturbance is unclear. The present report concerns a 33-year-old woman with reversible renal failure in the course of toxic shock syndrome owing to rhabdomyolysis. It is suspected that the renal consequences of muscle injury play a pathophysiological role in the acute renal failure of toxic shock syndrome.

Report of a Case

D.W. is a 33 year old white female who presented to another hospital with a one day history of nausea, vomiting, diarrhea, arthralgias, and sore throat. Her sister had noted the onset of tachypnea and cyanosis one hour earlier. The patient had been menstruating for three days using Playtex® tampons. In the emergency room, vital signs were: temperature 104.4, respiratory rate 44, blood pressure 60 to 80 palpable, and pulse 120. Skin was cyanotic but warm. She was admitted to the intensive care unit for presumed septic shock and large amounts of intravenous fluids were administered. Appropriate antibiotics, methyl prednisolone, mannitol, and

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dopamine were given. A vaginal lesion was positive for *Staphylococcus aureus* on gram stain and culture. After seven hours, the patient was transferred by airplane to the University of Oregon Health Sciences Center. She required three liters of intravenous fluid, a norepinephrine drip and a MAST suit to maintain her blood pressure en route. Her creatinine on presentation to the referring hospital had been 3.6 mg per dl.

Upon arrival at the University Hospital, the patient was agitated, cyanotic, and hypotensive. A diffuse erythroderma was noted which later exfoliated. The distal aspects of several digits were black. Her physical examination was otherwise unchanged. BUN was 34 mg per dl and creatinine was 3.5 mg per dl. Blood gases showed pH 7.18, pCO₂ 27 mmHg and PO₂ 257 mmHg. Measured bicarbonate was 6 mEq per L and lactic acid was 11.3 mmol per L. CPK was 26,000 I.U. on administration and peaked at 50,000 I.U. (Normal female, 25 to 135 I.U.). Calcium was 7.9 mg per dl, phosphate 7.9 mg per dl, and uric acid 14.3 mg per dl. Urinalysis showed a cloudy yellow urine which became grossly bloody within a few hours of admission. Specific gravity was 1.010, pH 5, protein 1+, hemoglobin 3+ with negative glucose, ketones and bilirubin. Microscopic analysis showed >100 red blood cells and 2 to 3 white blood cells per high power field. Pigmented granular casts were noted several days later. Fractional excretion of sodium was 11; however, mannitol and furosemide had been given within 12 hours. Urine osmolality was 321. Urine myoglobin was present.

Urine output was 55 ml per hr for the first 12 hours, but then diminished to 5 ml over the next 24 hours in spite of mannitol and furosemide therapy and vigorous volume repletion with Swan-Ganz catheter monitoring. Vigorous supportive therapy including early hemodialysis was instituted. The patient was oliguric and markedly azotemic for 21 days but then gradually regained renal function. Presently, her BUN is 15 mg per dl and creatinine 0.7 mg per dl.

**Discussion**

Criteria for diagnosis of TSS have been published elsewhere.1,3 This patient met all of the standard criteria. She had renal dysfunction at the time of presentation and followed a clinical course compatible with myoglobinuric acute renal failure. A role for hypotension, high doses of pressors and/or the putative staphylococcal exotoxin cannot be ruled out. This patient survived but has serious neurological sequelae. She has difficulty in swallowing and has severe dysarthria. In addition, she has intellectual deficiencies which were not present prior to her acute illness.

Saul et al4 reported a severely ill 13 year old girl with nonoliguric renal failure and rhabdomyolysis. Her menstrual history, tampon use, pelvic examination, and vaginal culture results are not stated. Her four week history of intermittent abdominal pain and limited fluid requirement (1490 ml on the first day) are not typical of TSS. If she did have TSS, then her cause further supports an etiologic association between rhabdomyolysis and the renal failure of TSS.

It is urged that in seriously ill patients with TSS, rhabdomyolysis and myoglobinuria be investigated. Mannitol appeared to delay the onset of oliguria for 36 hours in this patient. Further use of mannitol could conceivably have averted oliguria entirely. Aggressive early hemodialysis, as in rhabdomyolysis from other causes, is probably warranted if oliguria ensues.

**References**