

**Case Patient.** 50 yo male, h/o cocaine use, found down after suffering unwitnessed seizure. The patient is oliguric, what urine is seen is very dark. CK=50,000. What is the likely diagnosis and how will you treat this patient?

## Rhabdomyolysis

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**Definition:** an acute or subacute event causing necrosis of striated muscle, causing elevations of muscle enzymes, and in its severe form, causing electrolyte imbalances and acute renal failure.

**Clinical Presentation.** Patients may note dark urine and myalgias/swollen muscles, signs of dehydration, signs of trauma such as ecchymoses, as well as fever, altered mental status, seen in malignant hyperthermia, infection.

**Causes:** Trauma/CRUSH injury  
Surgery  
DRUGS (cocaine, heroin, colchicines, some HIV meds, EtOH, CSA, coumadin, digoxin, macrolides, halothane)  
\*\*statins can cause myositis, rhabdo less common unless in conjunction with other lipid-lowering agents (especially gemfibrozil and other fibrates)  
Immobilization, coma  
Overuse/overheat (e.g. marathon runners)  
Seizures, neuroleptic malignant syndrome, myopathies  
Infection, snake bite  
Endocrine disorders (hypo/hyperthyroidism, DKA, pheo)  
Hypothyroidism thought to be associated with reversible defect glycogenolysis, leading to insufficient muscle energy production during exercise, leading to muscle breakdown, correctible by treating the hypothyroidism  
Electrical injury  
Compartment syndrome  
Malignant hyperthermia  
Myositis/myopathies  
Ischemia and resultant reperfusion

-Hypokalemia, hypophosphatemia, hypo/hypernatremia and hyperosmolar state can be predisposing factors to muscle necrosis.

-Rhabdo can also lead to DIC, hepatic inflammation.

### Lab Presentation - Diagnosis usually made by labs

*CK and myoglobin are the key labs to diagnosing rhabdomyolysis*

-myoglobin (seen in serum and urine), with + heme on urine dipstick; pigmented casts in the urine

-elevated CK-MM fraction, usually > 10,000 IU/L but no defined cutoff value

-patients often times present with acute renal failure the exact cause of which uncertain, probably multifactorial from renal tubular obstruction by precipitated myoglobin, direct myoglobin toxicity, hypotension, dehydration, decreased glomerular filtration rate. Can be oliguric.

-elevated PO<sub>4</sub>, uric acid, metabolic acidosis from renal failure and/or myocyte necrosis.

-K can initially be low as a contributing factor, but can become high with increased K release from damaged muscle. When muscle is damaged in rhabdo, Ca can leave the serum and deposit in the

damaged muscle, resembling hypocalcemia, but w/ treatment of rhabdo with IVF the Ca can remobilize back into the serum.

-Labs c/w DIC, *e.g.* elevated D-Dimer, PT, PTT and INR, low platelets, low fibrinogen.

-Elevated AST and ALT levels, hypoalbuminemia can be seen.

**Management. THE EARLIER THE BETTER - MAKE THEM PEE!** Renal failure can occur in up to 30% of patients with rhabdo.

--Aggressive IVF hydration with isotonic saline, recommended bolus 1.0- 1.5 L/hour, goal urine output = 300 cc/hour until myoglobinuria ceases, cont. IVF at rate appropriate to keep good urine output until CK<1000 minimally.

--Follow serial values of K,PO4, CK, uric acid, Ca, Cr.

--The hyperkalemia of rhabdo, which is worst at 10-40 hours after injury, responds poorly to insulin/glucose, and IV Ca can cause metastatic calcifications and resultant hypercalcemia in recovery. Kayexelate or HCO<sub>3</sub> good first-line options, consider HD in severe hyperkalemia.

--Beware of potential hypercalcemia with treatment of rhabdo - the Ca remobilizes from the muscle. Ca replacement in an asymptomatic, hypocalcemic patient not recommended, for it can further muscle injury and cause hypercalcemia later on

--Treat specific causes. *e.g.* cooling blankets for heat stroke, dantrolene for NMS, stop offending meds, fasciotomy for compartment syndrome.

--Forced diuresis with mannitol (10 g/L), recommended only once BP and urine output are stable - avoid other diuretics. Efficacy over just IVF still being debated, but many experts still recommend it based on small studies.

--To give bicarb or not to give bicarb? There is no great data on alkalinizing the urine. The thought is that doing so prevents conversion of myoglobin to toxic methemoglobin and increases the solubility of the pigmented casts, but it can also increase Ca deposition. Recommended is 44 mEq/L, goal urine pH > 6.5

**A 2004 review of cases of rhabdo from trauma showed no difference in incidence of acute renal failure w/ or w/o mannitol and bicarb, recommending further review**

--D/C criteria: CK values have steadily decreased over 48 hours, no significant metabolic abnormalities exist, pt. feels clinically well and is willing to f/u in 1 week as outpatient.

**Case follow-up.** Pt. was diagnosed with rhabdomyolysis, CK's trended down with 0.9 NS 1.5 L/hour with 2 amps NaHCO<sub>3</sub> per liter. Pt. eventually transitioned to aggressive po fluid hydration when he was awake and CK was < 1000.

### Clinical Pearls

- Aggressive, early IV fluid hydration is the key! Keep the kidneys flowing!
- Watch your electrolytes carefully as you treat, avoid IV calcium infusions if at all possible

### References.

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