Chris Chase, MD

Recognizing Rhabdomyolysis in the ED

Rhabdomyolysis involves the **injury and necrosis of skeletal muscle causing a release of myoglobin and an increase in creatinine kinase (CK)**. This condition can be precipitated by **alcohol/drug abuse, trauma, strenuous physical activity, heat-related illness, toxin ingestion and infection**. It is important to detect the presence of rhabdomyolysis in order to prevent complications such as **acute renal failure, DIC, electrolyte imbalances, and compartment syndrome**.

Clinical history that may raise suspicion for this illness includes **recent immobilization with prolonged muscle compression, increased muscle activity (seizures, drug intoxication, strenuous exercise), electrical/lightning injury, traumatic crush injury, heat stroke and co-morbid diseases such as sickle cell, dermatomyositis, and polymyositis**. Obtunded patients secondary to alcohol or heroin intoxication are at risk due to immobilization and muscle compression, whereas drugs such as methamphetamine and cocaine can cause agitation, hyperthermia and excessive skeletal muscle energy demand. Rhabdomyolysis syndromes are acute in onset and most patients present with **myalgias (most commonly involving postural muscles of calf, thigh and back), weakness, low-grade fever, and dark urine** due to myoglobinuria. A high index of suspicion is needed when evaluating patients with a supporting clinical history for rhabdomyolysis because a patient may not exhibit all the classic symptoms early on in their presentation.

Rhabdomyolysis is diagnosed with a **5-fold or greater increase in the serum CK** above the upper limit of normal. CK levels **rise 2-12 hours** after muscle injury and **peak in 24-72 hours**. Urine will contain myoglobin when plasma concentrations exceed 1.5mg/dL and will appear reddish-brown once myoglobinuria reaches 100mg/dL. Urine dipstick tests will be positive but there will be an **absence of microhematuria with myoglobinuria**.

Work-up includes **serum electrolytes, BUN/Creatinine, calcium, phosphorus, uric acid, urinalysis, CBC, CK, CK-MB, EKG, and DIC profile** (order labs as indicated clinically). The most serious complication is **ARF** due to breakdown of myoglobin into ferrihemate, which is nephrotoxic (exact mechanism is controversial). **CK elevation correlates to extent of muscle injury, but is not directly related to the probability of developing ARF**!! It has been estimated that 17% to 35% of adults and 42% to 50% of children with rhabdomyolysis develop ARF. The combination of muscle breakdown and ARF can lead to **hyperkalemia**, so EKG and cardiac monitoring should be obtained promptly. **Hypocalcemia** is common in rhabdomyolysis due to early precipitation with anions, but is subsequently followed by hypercalcemia so treatment of the hypocalcemia is not recommended early on. The mainstay of therapy is **IV rehydration** with NS or 5% dextrose in NS with a goal urine output of 2.5ml/kg/hr. Foley catheters should be placed to monitor urine output. Serial examinations are warranted to monitor for renal failure, electrolyte disturbances, and compartment syndrome. **Hemodialysis** may be necessary when significant renal failure occurs. **In ED patients with initial CK > 1,000 U/L the incidence of death or hemodialysis has been found to be as high as 8% within 30 days**, therefore early recognition and adequate rehydration are crucial to prevent complications from rhabdomyolysis.

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