

Rhabdomyolysis: Recognizing Risks

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PATIENTS PRESENT WITH different histories, yet perianesthesia nurses anticipate a myriad of possible conditions that need to be assessed and consider possible interventions. What do the following cases have in common?

- A 32-year-old male who suffered a severe crushing injury to his lower leg in a mountain bike accident.
- A 21-year-old female training for an ultramarathon complaining of significant muscle pain, weakness, fatigue, and tea-colored urine.
- A 47-year-old electrician admitted for an electrical arch injury affecting his face, right hand, arm, and upper chest.
- A 54-year-old female with a familial history of malignant hyperthermia admitted for colon resection.
- An 82-year-old female found down for more than 6 hours at home with a broken hip.

The cases all involve skeletal muscle injury which places the individual at risk of developing rhabdomyolysis. This clinical syndrome can be caused by traumatic and crush injuries, severe infections, extreme physical exertion, temperature extremes, electrical injuries, vascular ischemia, and adverse drug reactions (eg, statin agents).^{1,2} Clinical symptoms of rhabdomyolysis can be nonspecific: fever, malaise, nausea and vomiting, and tachycardia. More overt symptoms include muscle pain and aches (eg, myalgia), weakness and swelling of the involved muscle, dark “tea-colored” urine (eg, myoglobinuria), electrolyte abnormalities (eg, hyperkalemia, hypophosphatemia, hypocalcemia), metabolic acidosis, elevated serum

creatinine kinase (CK), and myoglobinuria. Possible complications seen in patients with rhabdomyolysis include development of compartment syndrome, cardiac arrhythmias connected with hyperkalemia, and disseminated intravascular coagulation syndrome. However, the largest clinical concern associated with rhabdomyolysis is progression to acute kidney injury (AKI) which is estimated to occur in up to 40% of patients.³

Assessment and Management Considerations

Rhabdomyolysis is the result of direct or indirect muscle injury that leads to muscle fiber necrosis specifically to the myocyte, the muscle cell. Cellular injury disrupts the availability of adenosine triphosphate that is needed for cellular ion transport. The depletion of adenosine triphosphate causes cellular pump dysfunction resulting in an influx of sodium followed by water intracellularly and an outflow of potassium, phosphates, CK, and myoglobin extracellularly. The alterations in the muscle cell ability to function leads to a cyclic inflammatory destruction of muscle fibers and the release of muscle cell contents into the extracellular space and bloodstream.^{1,3} The large release of myoglobin overwhelms the renal tubule, creating a nephrotoxic state to include renal vasoconstriction and renal tubular casts. As the skeletal muscle injury persists, renal toxicity from myoglobin places the patient at risk of AKI.

Assessment

Assessment of the patient includes an understanding of the history and potential severity of skeletal muscle injury and, in addition, evaluation of the patient’s subjective symptoms (ie, fatigue, myalgia, weakness) and objective symptoms (ie, extremity color, pulses, serum CK and potassium, and urine myoglobin). While there is no gold standard for laboratory diagnosis, the CK upper limit of 1,000 U/L is commonly used; a CK value > 5,000 U/L raises concerns for the risk of AKI.^{3,4} Metabolic acidosis will ensue with the destruction of muscle cells and associated AKI.

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The significant release of potassium extracellularly places the patient at risk for cardiac arrhythmias. Assessment of the patient's electrocardiogram for peaked T waves, bradycardia, and dysrhythmias is indicated by the perianesthesia nurse.

Compartment syndrome may develop especially in cases in which the skeletal muscle necrosis was caused by a traumatic crush injury. Evaluation of the extremity for changes in pain, paresthesia, paresis, pallor, pulselessness, and poikilothermia is necessary to alert the interprofessional health care team of possible compartment syndrome that can also exacerbate the rhabdomyolysis.³ The coagulation cascade can be overactive with injury and consumption of clotting factors, leading to an imbalance; disseminated intravascular coagulation may result.

Management

Management of the patient with rhabdomyolysis focuses first and foremost on restoring tissue perfusion and addressing hypovolemia associated with fluid sequestration in the muscles and interstitial spaces. Fluid resuscitation of up to 30 ml/kg with isotonic saline is typically prescribed and titrated until the serum CK reaches normal levels.¹⁻³ Fluid resuscitation goals strive to achieve a urine output of 2 ml/kg/hour. Urine myoglobin levels typically clear before serum CK levels reach a clinical range that is no longer concerning; thus, monitoring urine for changes in color is a less reliable marker of clinical progress. Diuretics are currently considered somewhat controversial but may be prescribed to assist with renal perfusion.

Treating hyperkalemia will depend on the patient's symptoms. Treatment may include administration

of sodium polystyrene sulfonate (ie, Kayexalate), intravenous calcium gluconate, intravenous glucose and insulin, or possibly hemodialysis.^{2,4} Similarly, metabolic acidosis may require intervention. Alkalinization of the urine with intravenous bicarbonate administration along with hemodialysis may be prescribed; however, the efficacy of these interventions is unclear as a standard intervention but may be necessary based on individual patient clinical presentations.^{2,3}

Close hemodynamic monitoring, management of the patient's pain, and frequent re-evaluation of serum CK will be necessary in the care of the patient.

Implications for Practice

Rhabdomyolysis is a complex syndrome that can have significant morbidity and mortality risks for the patient. Patients with malignant hyperthermia should be carefully evaluated to allow for proper and rapid treatment of this life-threatening complication associated with anesthetic agents. Patients postoperatively presenting with tachycardia, masseter spasm, rigidity, extreme hyperthermia, hyperkalemia, acidosis, decreasing oxygen saturation, myoglobinuria, and family history of malignant hyperthermia require similar but different treatment priorities, namely, administration of dantrolene sodium.⁴ The patient with rhabdomyolysis requires aggressive fluid resuscitation, correction of electrolyte abnormalities, and kidney-supportive therapies. Perianesthesia nurses' assessment of the patient's past medical history, current injury, and suspicion of possible complications associated with skeletal muscle injury is essential for rapid and effective management of a patient with significant skeletal muscle injury and rhabdomyolysis.

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