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Rhabdomyolysis-Induced Severe Hyperkalemia

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A 22-year-old male was brought in by paramedics who found him unresponsive at home surrounded by drug paraphernalia. He had a blood pressure of 84/50 mm Hg; oxygen saturation of 88%; irregular pulse; and Glasgow Coma Scale of 3. His skin was cool, diaphoretic and ashen in color, except for a grossly erythematous right arm. Lab work demonstrated an initial creatine kinase of 5,414 U/L that increased to 165,300 U/L by the next day. His potassium was 8.6 mmol/L.

Hyperkalemia, defined as a potassium level greater than 5.5 mmol/L, is a potentially fatal disorder that occurs in as many as 10% of hospitalized patients.^{1,2} Causes include rhabdomyolysis, hemolysis, renal failure, acidosis, and medications.² Cardiac conduction can be impaired, resulting in dysrhythmias. Electrocardiography (ECG) is important because it may be diagnostic and dictate emergent treatment. The first ECG changes seen are shortening of the QT interval, peaked T waves, and ST segment depression, followed by widening of the QRS complex, increased PR interval, and decreased P wave amplitude. Lastly, the P wave disappears and the QRS widens until it resembles a sine wave. Ventricular fibrillation or asystole may follow. Emergent treatment with calcium gluconate or calcium chloride is indicated to stabilize the myocardium if ECG changes are present.^{2,3} Serial doses of calcium are given until the ECG normalizes.² Inhaled albuterol, intravenous sodium bicarbonate, and intravenous insulin with glucose should be used to temporarily shift potassium back into cells.³ Furosemide and sodium polystyrene should also be administered to increase the excretion of potassium. Hemodialysis may be required in the setting of renal failure or severe cases. The ultimate treatment goal of hyperkalemia is to identify and correct the underlying etiology.



Figure 1. Rhythm strip of lead II demonstrating severe hyperkalemia.

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