Severe Rhabdomyolysis Secondary to Adenovirus Infection

Daniel Tseytlin DO
Lehigh Valley Health Network, Daniel.Tseytlin@lvhn.org

Sharon E. Maynard MD
Lehigh Valley Health Network, Sharon_E.Maynard@lvhn.org

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Severe Rhabdomyolysis Secondary to Adenovirus Infection

INTRODUCTION

• Rhabdomyolysis presents with weakness, myalgia, and dark urine. There is severe muscle injury with extrusion of myocyte contents into the blood.
• There have been several reports of viral myositis resulting in rhabdomyolysis. The most common cause of rhabdomyolysis is influenza, accounting for 33-42% of adult cases.1 Viral myositis causing rhabdomyolysis, CPK elevations can exceed 800,000 U/L.2,3
• Adenovirus is a double-stranded DNA virus of the Adenoviridae family most commonly implicated in pediatric upper respiratory illness. Uncommonly, adenovirus can cause encephalitis, meningitis, hemorrhagic cysts, myocarditis, and rhabdomyolysis.2,4
• Only 2 cases of myositis from adenovirus have been reported in the literature.2,4 Adenovirus most commonly manifests as a febrile illness with pharyngitis and cervical adenitis.
• The diagnosis of viral myositis precipitating rhabdomyolysis is based on characteristic clinical features, results of viral culture and PCR, and exclusion of other causes of rhabdomyolysis.

The pathogenesis of viral myositis is not well understood. Direct myocyte infection by the virus is one proposed mechanism, though visualization of viral inclusions on muscle biopsy is inconsistent.5

In most cases, identification of the specific viral pathogen will not impact management or inform prognosis; thus, the benefit of serology testing is questionable.5,6 Osmotic myolysis, or release of intracellular phosphate and subsequent movement of extracellular calcium into damaged muscle cells, is one proposed mechanism of myocyte injury in rhabdomyolysis.7-9

The incidence of acute kidney injury among hospitalized patients with rhabdomyolysis is estimated between 13% and 50%.10,11 Nearly 4% of patients suffering acute kidney injury from rhabdomyolysis require hemodialysis,11,12 for a 10-fold increase compared to the general population.13

• Predicting incidence and severity of renal impairment, need for renal replacement therapy, and mortality based on CPK level has been studied. In a cohort of critically ill patients with rhabdomyolysis, de Meijer and colegues14 found a positive correlation between the degree of CPK elevation and the incidence of acute kidney injury. However, other studies have shown a lack of correlation of the CPK level on presentation or the peak CPK level on presentation with need for renal replacement therapy.15,16

• Hypocalcemia in rhabdomyolysis is due to movement of extracellular calcium into damaged muscle cells, and subsequent complexing with intracellular phosphate and soft tissue calcification.17

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Calcium and Phosphorus vs. Time

<table>
<thead>
<tr>
<th>Serum Parameter</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 64</th>
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<tr>
<td>SERUM POTASSIUM (mEq/L)</td>
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<tr>
<td>PHOSPHORUS (mg/dL)</td>
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<td>4.7</td>
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<td>SERUM CALCIUM (mg/dL)</td>
<td>11.6</td>
<td>11.6</td>
<td>&lt;5</td>
<td>&lt;5</td>
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</table>

HEMODIALYSIS, CALCIUM AND PHOSPHATE MANAGEMENT

- Despite aggressive volume resuscitation, the patient remained oliguric and hypertensive, and was initiated on hemodialysis (HD) on hospital day 2. His initial dialysis treatment consisted of 2 hours with a blood flow rate of 200 mL/min and a dialysate flow rate of 300 cce/hr, against a 2 mEq/L potassium bath and 2.5 mEq/L calcium bath. His treatment was continued on daily HD for an additional 7 days.2,3 On the day of HD, he was maintained on CVHD until the potassium normalized, and he was transitioned back to intermittent HD on hospital day 7. On admission the patient weighed 111 kg. His peak body weight was 118.5 kg on hospital day 9, reflecting accumulated volume from fluid resuscitation. On day of discharge the patient weighed 110 kg.

- Our patient developed severe hypercalcemia by hospital day 2, without signs of neuromuscular irritability. Initially, severe hyperparathyroidism predicated calcification in the heart, lungs, and brain. However, case reports suggest oseltamivir is not effective in preventing or treating influenza-associated rhabdomyolysis.14

- Hypocalcemia is rhabdomyolysis is due to movement of extracellular calcium into damaged muscle cells, and subsequent complexing with intracellular phosphate and soft tissue calcification.17

- Low levels of 25(OH)D, Vitamin D and skeletal resistance to parathyroid hormone may also contribute to hypocalcemia in this setting.18 Calcium supplementation is usually avoided unless hypocalcemia is symptomatic to minimize the development of hypercalcemia during the recovery phase. Hypocalcemia should be treated with intravenous calcium if carpopedal spasm, tetany, or seizures develop.

CONCLUSION

Adenoviral infection can lead to rhabdomyolysis with severe acute kidney injury. Respiratory viral panel should be included in workup of rhabdomyolysis when no other cause is evident. Treatment includes supportive care, intravenous fluid resuscitation, and renal replacement therapy. Continuous renal replacement therapy should be considered in severe cases for management of severe electrolyte abnormalities and volume status.

References:
7. Lehigh Valley Health Network, Allentown, Pennsylvania