Severe Dehydration with Cramping Resulting in Exertional Rhabdomyolysis in a High School Quarterback

Daniel J. Ruiz, Israel D. Mitchell*, Lindsey E. Eberman, Michelle A. Cleary
Florida International University Miami, FL and *Belen Jesuit Preparatory School Miami, FL

Objective: We present a case of a unique pathophysiological injury involving severe dehydration, muscle cramping, and resultant rhabdomyolysis in a high school football player**. Background:** A 16 y old male football player (body mass = 69.1 kg, height =175.3 cm) reported to the ATC after the morning session on the second day of two-a-days complaining of severe muscle pain and cramping. Upon arrival to the emergency department, intravenous (IV) fluid was administered and blood analysis revealed creatine kinase (CK) that peaked at 3363 $IU \cdot L^{-1}$ (normal range=26-174 $IU \cdot L^{-1}$). Following initial testing, the athlete was transported to a children's hospital for differential diagnosis. **Differential Diagnosis:** The physician suggested severe dehydration, exertional rhabdomyolysis, or myositis. CK testing revealed elevated levels indicating mild rhabdomyolysis. **Treatment:** Eight liters of IV fluid was administered within the 48 hr hospitalization period. Early fluid replacement is the key to managing acute exertional rhabdomyolysis. **Uniqueness:** To our knowledge, no reports of exertional rhabdomyolysis in an adolescent athlete have ever been reported. In addition, increased CK levels have been reported in contact football during preseason practices but players were participating in contact drills. Our athlete developed rhabdomyolysis even after being well conditioned and acclimated for exercising in pre-season non-contact conditioning practice. **Conclusions:** The athlete was released from the children's hospital in stable condition on Day 5 post-incident with a CK level of 1550 IU·L. The athlete was cleared and returned to practice on Day 7, participated in all activities and was monitored for any return of symptoms. **Key Words:** heat illness, creatine kinase, acclimatization

Acute exertional rhabdomyolysis is a problem encountered by athletes as a result of extreme or novel physical demands placed on the musculoskeletal system. Exertional rhabdomyolysis results from the degeneration of skeletal muscle caused by excessive or unaccustomed eccentric exercise.[1] Athletes participating in hot and humid environments are even at greater risk due to the effects of dehydration and hyperthermia on skeletal muscle. Muscle damage and necrosis mostly occur in dehydrated, untrained individuals during downhill walking, running, or resistance-training exercises.[1,2] Rhabdomyolysis has also been reported in long distance runners, weight lifters, football players[3] and recently in a correctional facility.[4] Rhabdomyolysis has many underlining causes, some of which are genetically inherited while others are acquired. In the United States, 26,000 cases are reported annually, but most of the cases involve military personnel, law enforcement, and fire department trainees.[5] We found few reports of exertional rhabdomyolysis in athletes and none in adolescent American football players. We present a case that occurred on the second day of high school pre-season football training in a 16 year old male (mass = 69.1 kg, height = 175.3 cm) quarterback who initially presented to the Certified Athletic Trainer with severe muscle cramps.

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Case Report

Case History

The athlete reported to pre-season practices well acclimatized with indoor (primarily)/outdoor conditioning for the previous eight weeks and reported no previous history of hospitalization for exertional heat illness, medical conditions recent illnesses, medications, or sport supplementation. Participation in pre-season practice consisted of team conditioning and non-contact, position specific drills with approximately 7.5 min water breaks every 45 - 60 min. On the first day of pre-season conditioning, the team was educated on the importance of rehydration. The athlete reported to the ATC after the morning session on the second day of two-a-days complaining of severe muscle pain and cramping in the lower legs and hamstrings that progressed into the lower back and abdominal muscles. The athlete reported that throughout the 3.5 hr practice, he consumed water during the three breaks and estimated to have drunken approximately 6-8 oz at each break. The treatment consisted of copious water and carbohydrate-electrolyte beverage consumption and mild stretching of the affected muscles. During treatment, painful and spasmodic involuntary contractions began to affect the larger muscles groups of the lower back and abdominal muscles, along with the previously mentioned lower legs. Ice bags were then immediately placed on the cramping muscle groups to desensitize the involuntary muscle contractions. Vital signs were closely monitored every 15 min for approximately 1 hr and were considered within normal limits. The emergency medical response unit was contacted bringing the total elapsed time from initial evaluation to hospitalization to approximately 3 hr.

Upon arrival to the emergency department, intravenous (IV) fluid was administered and complete urinalysis, hematology, and chemistry reports were ordered. The urinalysis report was negative for hemoglobinuria and the urine (volume=700 mL; specific gravity$\leq$1.005 μG) was clear and had a straw colored appearance. Blood analysis revealed creatine kinase-MB levels at 17.2 $ng \cdot mL^{-1}$ (normal = 0.6–6.3 $ng \cdot mL^{-1}$) and creatine kinase (CK) peaked at 3363 $IU \cdot L^{-1}$ (normal range = 26-174 $IU \cdot L^{-1}$). Blood urea nitrogen (24 $mg \cdot dL^{-1}$) and creatinine (1.6 $mg \cdot dL^{-1}$) were also elevated above normal ranges. Calcium was slightly elevated at 10.8 $mg \cdot dL^{-1}$ (normal = 8.6-10.3 $mg \cdot dL^{-1}$) and potassium was within its normal limits. The athlete was maintained on a fluid replacement therapy and two additional blood reports were ordered throughout the course of the evening. The athlete received 8000 mL of intravenous saline in addition to 900 mL by mouth, bringing the total fluid intake to 8900 mL with 700 mL excreted by the urinary system. The athlete's additional blood analysis continued to demonstrate elevated CK-MB and CK levels, at which time the attending physician opted to transport the athlete to the children's hospital for further treatment of dehydration and to rule out rhabdomyolysis as a differential diagnosis.

Differential Diagnosis

Admission to the children's hospital occurred approximately 15 hr after emergency department admission. Upon physician evaluation, the athlete maintained strict volume intake of 120 $mL \cdot hr^{-1}$ of IV fluid. A new series of laboratory exams followed to rule out rhabdomyolysis. The results of the urinalysis for our athlete produced no traces of myoglobinuria, hematuria, or hemoglobinuria, and a 1.015 μG specific gravity with a clear and yellow color. Although urine specific gravity appeared to be normal, patients suffering from renal dysfunction tend to have urine specific gravity equal to that of blood plasma (1.008 - 1.010 μG) regardless of changes in the patient's sodium and water intake.[6]

Physicians suggested a differential diagnosis of severe dehydration, exertional rhabdomyolysis, or myositis. The CK test, also known as the total CK, is a laboratory exam

ordered if a patient complains of muscle pain or general body weakness or if a myocardial infarction is suspected. Testing for CK is the most reliable diagnostic indicator for rhabdomyolyis.[7] In our case, CK analysis revealed CK levels peaking approximately 12 hr after the end of exercise and then declining around 24 hr. The CK levels at 36 hr had further reduced. The athlete's electrolytes were within normal limits and the athlete was discharged after consultation with a nephrologist on the second day of hospitalization.

Case Evolution and Denouement

The athlete was cleared and returned to practice on Day 7 and participated in all activities although he had a 2.75% body mass loss at the time of return to activity and sustained a maximum loss of 4.6% body mass throughout the course of the condition and treatment. The ATC was advised to monitor the athlete's return of symptoms. The ATC encouraged the athlete to take frequent water breaks throughout practices and consume supplemental fluids while at home. For approximately two weeks after his return to full activity, the athlete consumed 16 oz of carbohydrate-electrolyte beverage prior to and following each practice to supplement water intake. The athlete's return to participation has not stimulated any additional bouts of muscle cramping or dehydration.

Discussion

Dehydration and Heat Cramps

Dehydration in athletics occurs because of inadequate replacement of sweat loss during and following training and competition.[8] While performing physical exercise in hot weather, it is essential that athletes replace the fluids lost through sweat by drinking equal quantities of water.[9] Sweat occurs independently of fluid intake and if sweat losses are not replaced by fluid intake, dehydration negatively impacts athletic performance. Dehydration levels as low as 2% impair the cardiovascular and thermoregulatory system causing a negative impact on the capacity to perform exercise.[9]

Heat cramps are extremely painful muscle spasms that occur most commonly in the calf and abdomen, although any muscle can be involved. Although conclusive evidence is lacking, heat cramps are likely the result of a sodium chloride deficit.[10] Heat cramps are also one of the most common clinical problems encountered by medical professionals dealing with athletes, especially marathon and triathlon athletes.[11]

Etiology of Exertional Rhabdomyolysis

Exertional rhabdomyolysis is one of the most common forms of rhabdomyolysis,[12] and is characterized by muscle necrosis and release of intracellular contents such as myoglobin and creatine kinase into the bloodstream.[13] Clinically, rhabdomyolysis is characterized by symptoms of nausea, vomiting, agitation, weakness, and muscle pain, along with tea-colored urine.[13] Acute renal failure is one of the most serious late-stage complications of rhabdomyolysis occurring in 33% of patients.[1]

Dehydration causes muscle damage and necrosis especially in untrained participants performing unaccustomed exercise in high temperature ambient environments.[1] During exercise, heat is generated and blood is drawn away from the gastrointestinal tract and kidneys and is shunted toward the skin to aid in heat dissipation.[1] This thermoregulatory response to exercise causes tissue and muscle hypoxia, depletion of adenosine triphosphate, and eventually muscle cell necrosis and cell death if the process is not reversed in time.[13]

Eccentric exercise has been associated with elevated levels of plasma CK in the circulatory system, as CK is one of the proteins that is released into the blood stream from the skeletal muscle when injury to the muscle occurs.[14] An elevated CK level provides the most

sensitive enzyme marker for muscle damage[14] and is extremely important in the diagnosis of rhabdomyolysis. In patients suffering from severe cases of rhabdomyolysis, CK levels may increase to 100,000 IU/L[13] or more, with normal levels ranging from 26 to 174 $IU \cdot L^{-1}$. In addition to elevated levels of CK, rhabdomyolysis typically includes elevated levels of blood urea nitrogen and creatinine as a result of pre-renal causes of acute renal failure from dehydration and myoglobinuria.[13]

Treatment and Prognosis of Exertional Rhabdomyolysis

Once rhabdomyolysis is diagnosed, early fluid replacement is necessary to preserve renal function and to prevent acute renal failure.[13] Initially, rhabdomyolysis is treated with high-volume IV fluid replacement, administered at a rate of 1.5 $L \cdot hr^{-1}$, which is usually about 200 $cc \cdot hr^{-1} \cdot liter^{-1}$ bag. Patients may require as much as 4 to 10 L of normal saline in the first 24 hr to maintain circulation and stabilize blood pressure.[15] Skeletal muscles can recover from episodes of rhabdomyolysis with minimal permanent damage and the overall survival rate after rhabdomyolysis is approximately 77%.[13]

Uniqueness of Our Case

In most cases of exertional rhabdomyolysis, strenuous exercise is the primary etiological factor because of lack of patient experience and fitness level, unaccustomed intensity levels, unaccustomed duration levels, or the type of muscle contraction performed during the exercise. In our case, a 16 y old male athlete was participating in a typical pre-season football camp when he developed severe dehydration and a mild case of exertional rhabdomyolysis. The athlete was acclimatized and had been participating in conditioning consisting of running sprints and non-contact football specific drills, most of which would not be classified as eccentric contractions. To our knowledge, this case is the first documented case of severe dehydration and rhabdomyolysis occurring in an adolescent athlete.

Clinical Implications

The physiologic effects of exercising in hot and humid environments have been widely studied and continue to be explored today. Exertional rhabdomyolysis is the most common form of rhabdomyolysis and, if not detected, can lead to severe complications such as renal failure and even death. Dehydration, high ambient temperatures, and high humidity levels are all risk factors for developing exertional rhabdomyolysis; all of which are fairly common in football. Athletic trainers are the life line needed for an athlete unaware of the signs, symptoms, and dangers of dehydration. Athletic trainers should be suspicious of rhabdomyolysis when symptoms of dehydration or severe acute muscle soreness are present. Furthermore, it is critical that during intensive conditioning programs, such as two-a-day practices, athletes are educated and closely monitored for signs and symptoms of dehydration and that appropriate re-hydration opportunities are provided before, during, and after training.

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