

Case Letter

A case of exercise induced rhabdomyolysis from calf raises

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Dear editor,

A 27-year-old male presented to the emergency department with acute exercise induced rhabdomyolysis (EIR) following low intensity, high repetition physical activity. It is paramount for the clinician to consider this diagnosis in the differential of the patient presenting with a complaint of musculoskeletal pain. This case highlights the necessity of staying vigilant for a condition that can develop with seemingly minor, repetitive training of a single muscle group, such as in the exercise of calf raises.

CASE

A 27-year-old male medical student presented to the emergency department with report of bilateral lower leg pain. The patient described the pain as crampy in nature, localized to the posterior aspect of both legs in the distribution of the gastrocnemius-soleus complex. Three days prior to arrival the patient engaged in an intensive exercise routine consisting of over 200 calf raises. The patient reported this activity as the only exercise he had preformed and that he did not engage in any routine exercise prior to the episode. Over the course of the preceding two days, the patient developed intense calf pain which impaired his ability to ambulate. The patient also reported the development of brown-colored urine and a generalized sense of weakness which caused him to seek medical attention. The patient had a past medical history significant for an unspecified mood disorder, gastroesophageal reflux disease, and obesity with a body mass index of 36. Surgical history was significant for wisdom teeth extraction. Outpatient medications included omeprazole, ritalin, and wellbutrin. He denied any drug allergies. Social history was negative for alcohol, tobacco, or illicit drug use.

His initial presenting vital signs were significant for relative hypertension with a blood pressure of 151/77

mmHg, heart rate of 96 beats per minute, respiratory rate of 16 breaths per minute, and he was afebrile with an oral temperature of 98.4 degrees Fahrenheit. Physical exam revealed a well appearing male in no acute distress. His pulmonary, cardiovascular, and abdominal exams were all unremarkable. On musculoskeletal exam, the patient was noted to have tenderness upon palpation over the posterior aspects of his bilateral lower legs. Of note, the patient was found to have full range of motion, full and symmetric strength testing of the lower extremity muscle groups, and no evidence of edema or calf asymmetry was identified.

The emergency physician ordered a complete blood count, basic metabolic panel, total creatine kinase, and urine analysis with associated microscopic evaluation. Results included blood urea nitrogen of 19 mg/dL, creatinine of 1.12 mg/dL, without an available baseline value in the record for comparison, white blood cell count of $9.2 \times 10^3/\mu\text{L}$, hemoglobin of 16.0 g/dL, platelets of $184 \times 10^3/\mu\text{L}$, and a total creatine kinase of 31 166 U/L. Urine analysis revealed a clear yellow appearing urine with a specific gravity of >1.030 , qualitatively large blood, and no evidence of nitrates or leukocyte esterase. Microscopic analysis of the urine sample revealed 3–5 red blood cells per high power field.

The clinical presentation of myalgia in a specific muscle group two days following a strenuous training regimen, a significantly elevated total creatine kinase level, and large blood in a urine sample containing only a small quantity of microscopic hematuria pointed towards the presumptive diagnosis of acute rhabdomyolysis. The patient was administered two liters of isotonic crystalloid and admitted to the inpatient general medical floor for appropriate surveillance and intravenous hydration. The patient's hospital course lasted a total of six days after which time he was discharged home in stable condition. Of note, the patient's total creatine kinase level was

trended closely and displayed a marked elevation from the initial value obtained in the emergency department to a peak of 51 592 U/L. This marker was followed and declined over the course of admission to a value of 2 310 U/L on the day of discharge. An initial target for discharge was a total creatine level of 1 000 U/L. However, at day 6 of hospitalization the inpatient team felt he was stable for release after appropriate downward trend was monitored. He was maintained on a fluid regimen of normal saline at 250 mL/hour over the bulk of his hospital stay. His intake and output was closely monitored as well as his renal function which remained intact throughout the course of his illness. His symptoms of myalgia improved rapidly with fluid administration and were resolved by the second day of hospitalization. At discharge he received strict instruction to avoid all strenuous physical activity until evaluated by his primary care physician in one week.

DISCUSSION

Rhabdomyolysis is a medical condition characterized by the excessive destruction of muscle cells.^[1] Clinically, this is seen as a patient presenting with complaints of myalgia, muscle weakness, and myoglobinuria commonly manifesting as brown colored urine.^[2] A number of different agents have been recognized as triggers in the development of this process which include notably mechanical injury to muscle tissue, ischemia, and drug or toxin exposure in susceptible individuals. Regardless of the precipitant, at the cellular level rhabdomyolysis is characterized by a depletion in the intracellular stores of adenosine triphosphate (ATP) and an elevation in ionized calcium within the mycoplasma. This process results in the activation of specific calcium responsive proteases which contribute to myofibril destruction.^[3] The activity of these proteases leads to the structural changes of the myofibril and to the appearance of the sarcomere. This can be seen as changes of sarcomere organization referred to as Z-line streaming or with continued destruction to Z-line breakdown.^[4] The destruction of myofibrils leads to the liberation of intracellular contents into the blood stream. This can be followed clinically by monitoring blood levels of myoglobin, creatine kinase, aspartate aminotransferase, alanine aminotransferase, and potassium.^[5] The diagnosis of rhabdomyolysis is dependent on the presence of the clinical symptoms and an elevated creatine kinase level at least five times the upper limit of normal.^[6]

In this case, the patient presented with a specific form of the condition known as exertional or exercise induced

rhabdomyolysis (EIR). This is a relatively rare condition with an incidence of approximately 29.9 cases per 100 000 patient years.^[6] The condition is often considered in weightlifters, marathon runners, military recruits and the like who are subject to exceptionally strenuous exercise regimens. However, recent case reports in the literature demonstrate the prevalence of EIR in otherwise atypical patient populations including low intensity weight lifters, the pediatric population, and participants in cycling classes.^[1-3,7]

Our case presents a patient who was sedentary and became involved in a high repetition exercise using only his body weight for resistance and training of a single muscle group. The development of acute exertional rhabdomyolysis from calf raises has to this point not been reported in the literature. The clinician needs to have a high index of suspicion for the diagnosis of EIR when seeing a patient who reports symptoms of myalgias or signs of myoglobinuria, even though they might report an atypical exercise history. Of note, it is important to consider the interplay of additional factors which could have made this patient susceptible to the development of rhabdomyolysis. This includes the possibility of medication use increasing his susceptibility to the condition. There is an association between the use of the stimulant medication phentermine and the development of EIR.^[3] It is possible the patient's use of the stimulant Methylphenidate could have posed a similar mechanism in precipitating rhabdomyolysis. Cases of bupropion induced rhabdomyolysis also exist in the literature, and the patient's use of the drug may have contributed to his diagnosis.^[8] This is also true for the use of omeprazole, which has been associated with development of drug induced acute rhabdomyolysis in intensive care unit patients.^[9]

CONCLUSION

Clinicians need to consider the diagnosis of acute exertional rhabdomyolysis in patients who present with persistent myalgia or weakness regardless of the degree of precipitating physical activity. Acute exertional rhabdomyolysis can develop in patients who engage in low intensity, high repetition training of a single muscle group. It is also important to consider the interplay certain medications can have in precipitating rhabdomyolysis in the non-athlete.

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REFERENCES

- 1 Tran M, Hayden N, Garcia B, Tucci V. Low-intensity repetitive exercise induced rhabdomyolysis. *Case Rep Emerg Med.* 2015;2015:281540.
- 2 Hummel K, Gregory A, Desai N, Diamond A. Rhabdomyolysis in adolescent athletes: review of cases. *Phys Sportsmed.* 2016; 44(2):195–9.
- 3 Hohenegger M. Drug induced rhabdomyolysis. *Curr Opin Pharmacol.* 2012;12(3):335–9.
- 4 Hikida RS, Staron RS, Hagerman FC, Sherman WM, Costill DL. Muscle fiber necrosis associated with human marathon runners. *J Neurol Sci.* 1983;59(2):185–203.
- 5 Gagliano M, Corona D, Giuffrida G, Giaquinta A, Tallarita T, Zerbo D, et al. Low-intensity body building exercise induced rhabdomyolysis: a case report. *Cases J.* 2009;2(1):7.
- 6 Tietza DC, Borchers J. Exertional rhabdomyolysis in the athlete: a clinical review. *Sports Health.* 2014;6(4):336–9.
- 7 Kim D, Ko EJ, Cho H, Park SH, Lee SH, Cho NG, et al. Spinning-induced rhabdomyolysis: eleven case reports and review of the literature. *Electrolyte Blood Press.* 2015;13(2):58–61.
- 8 Miladli A. Rhabdomyolysis associated with bupropion use as a smoking cessation adjunct: review of the literature. *Mil Med.* 2008;173(10):1042–3.
- 9 Tanaka K, Nakada TA, Abe R, Itoga S, Nomura F, Oda S. Omeprazole-associated rhabdomyolysis. *Crit Care.* 2014;18(4): 462.

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