Singapore Med J 2012; 53(2) : e42

Rhabdomyolysis in a recreational swimmer

Stella JJ[1], MBBS, *Shariff AH*[1], MBBS, MMed

ABSTRACT Rhabdomyolysis is a clinical and biochemical syndrome resulting from skeletal muscle injury, which may ultimately lead to acute renal failure (ARF) and death. Exertional rhabdomyolysis refers to skeletal muscle injury that is usually induced by strenuous eccentric exercises in a hot and humid environment. It is usually seen in marathoners and military personnel. We present the case of a 32-year-old Malaysian man who had rhabdomyolysis and myoglobinuria without ARF after two episodes of unaccustomed swimming. He was treated conservatively, and recovered uneventfully. A brief discussion on the pathophysiology of rhabdomyolysis, the principles of management and recuperation is included.

Keywords: athletic injuries, case management, myoglobinuria, pathophysiology, rhabdomyolysis
Singapore Med J 2012; 53(2): e42–e44

INTRODUCTION

Rhabdomyolysis is defined as a clinical and biochemical syndrome resulting from skeletal muscle injury that sufficiently disturbs the integrity of sarcolemma to allow the release of muscle cell content into the plasma. Epidemiologically, it is often underreported; as many as 26,000 cases may occur per year.[1]

Exertional rhabdomyolysis refers to skeletal muscle damage induced by exercise, especially strenuous and eccentric exercises. A search in the iMalaysiana and the Singapore Medical Journal did not yield any result on exertional rhabdomyolysis. This report describes the case of a recreational swimmer who presented with rhabdomyolysis.

CASE REPORT

A 32-year-old Malaysian Indian man presented to the Emergency Department with complaints of sudden onset of shortness of breath, headache, dizziness and generalised body aches that occurred while he was swimming several hours earlier. This was the second such episode; the first having occurred about a month ago, when he began to swim after not doing so for several years. In both incidents, the patient had been asymptomatic and well prior to swimming; his difficulties became apparent only after he had swum several laps in the standard swimming pool of the hotel where he worked. He did not seek treatment for the first incident.

The patient had no significant medical history except for an undiagnosed headache of ten years duration, which was unrelated to physical activity. He had been working as a hotel receptionist for six years, and had recently doubled up as a lifeguard at the same hotel. He worked out at the hotel gym thrice weekly, focusing mainly on aerobic endurance exercises with varying intensities, with occasional strength training. He was able to exercise without much distress or body ache.

He neither smoked nor consumed alcohol or other substances. He was also not on any alternative or traditional remedy. He had no family history of neuromuscular disease; however, both his parents had diabetes and hypertension.

Physical examination revealed the patient to be a medium-built man who was alert and able to walk into the Consultation Room unaided. His vital signs were stable, but he was slightly tachycardic. He had a blood pressure of 124/82 mmHg, heart rate 116 beats per minute, respiratory rate 20 per minute, pulse saturation 98% at room air, and was afebrile. Musculoskeletal examination revealed generalised muscle tenderness, which was more prominent on the proximal upper body region (bilateral pectoralis, deltoid, biceps, triceps). However, muscle power was preserved and he had no neurological deficit. The patient had normal joint movements but experienced some difficulty in squatting due to the pain in his lower limbs. The rest of the physical examination was normal.

Laboratory examination revealed elevated serum creatine kinase (CK) level of 112,400 U/L (normal range [NR]: 35–230 U/L), with CK-MB of 980 U/L (NR: 0–12 U/L). Renal function and electrolytes were normal. The patient's liver enzymes were elevated: lactate dehydrogenase (LDH): 3566 U/L (NR: 100–190 U/L), alanine aminotransferase 142 U/L (NR: 30–65 U/L), aspartate aminotransferase 535 U/L (NR: 15–37 U/L); alkaline phosphotase, and full blood count were all normal. His urine appeared dark-coloured and laboratory examination showed haemoglobin 5+ but negative for erythrocytes, proteinuria of 5.00 g/L and a pH of 5.0. There were also amorphous urate crystals 1+, bacteriuria 2+ and leucocyte esterase 1+. Electrocardiogram showed mild sinus tachycardia with no other significant findings.

The patient was treated conservatively with total rest at home. On follow-up three days later, he reported a decrease in body aches. His serum CK level had decreased to 49,437 U/L with normal CK-MB value. His LDH also decreased to 1178 U/L. At the subsequent follow-up one week later, the patient was

[1]Unit of Sports Medicine, University of Malaya, Kuala Lumpur, Malaysia
Correspondence: Dr Stella Jane Joseph, Postgraduate Trainee, Unit of Sports Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur 50603, Malaysia. stella6873@gmail.com

asymptomatic with almost normal laboratory examination of his urine, and was discharged with advice to return if symptoms resurfaced.

DISCUSSION

The American College of Cardiology/American Heart Association's Muscle Toxicity and Rhabdomyolysis defines rhabdomyolysis as "muscle symptoms associated with marked CK elevation, typically substantiated more than ten times the normal upper limit." Exertional rhabdomyolysis typically occurs secondary to strenuous exercise in otherwise healthy individuals, as reportedly seen in marathoners and military personnel who exercise in hot and humid environments.[2] Much knowledge about exertional rhabdomyolysis comes from laboratory studies using high-force eccentric contractions to induce damage.[3] This damage is attributed to mechanical tearing of the fibres and may result in CK levels of > 5000 U/L (NR < 250 U/L), peaking at four days post exercise. In endurance sports, muscle damage is most likely a result of metabolic crisis (compromised energy production) induced by prolonged exercise.[4] It typically produces CK levels < 5000 U/L, peaking at about 24 hours post exercise.

The American College of Sports Medicine classifies swimming in Group II of aerobic exercise modes. This classification is based on skill demands and can be considered as a moderate-to-high intensity activity.[5] Furthermore, swimming is performed almost exclusively with concentric contractions, regardless of the swimming style.[6] Although our patient was unaccustomed to swimming, the type and degree of activity that brought about his rhabdomyolysis in the absence of drug or alcohol usage raises the question of whether he had underlying genetic aetiologies that rendered him susceptible to rhabdomyolysis with minimal insult. In addition, considering that he had been regularly working out in the gym thrice weekly with a variety of aerobic and strength training exercises, the only assumed differences would be the higher humidity level of the pool and the chlorinated water. However, these and other risk factors such as ambient temperature and hydration status at the time of the event could not be ascertained.

Diagnoses of post-exercise muscular pain are often based on the time-frame of the onset and severity of symptoms, level of conditioning and environmental factors.[7] Acute muscle soreness (onset 0–24 hours) and delayed-onset muscle soreness (onset 24–72 hours) are the result of muscle overuse and have similar clinical features. Exertional rhabdomyolysis, however, tends to present with more severe muscle soreness, occurring as a triad together with weakness and dark urine. Early complications include hyperkalaemia, hypocalcaemia, hepatic inflammation, cardiac arrhythmia and cardiac arrest. Late complications include acute renal failure (ARF) and disseminated intravascular coagulation. Compartment syndrome may present early or late.[1]

Whatever the initial insult may be, at cellular level, it leads to a cascade of pathologic cellular responses – a decrease in intracellular adenosine triphosphate leading to extravasation of extracellular calcium ions from the sarcoplasmic reticulum into the intracellular space.[1] Intracellular calcium stimulates the activation of catalytic enzymes and proteases that degrade muscle cell membranes, thus releasing copious levels of degradation products such as CK, LDH, alanine transaminase, calcium, myoglobin, potassium, uric acid and phosphorus into the systemic circulation. Under physiologic conditions, the plasma concentration of myoglobin is very low (0–1.713 nmol/L). However, if more than 100 g of skeletal muscle is damaged, serum haptoglobin binding becomes saturated; therefore, the circulating myoglobin becomes 'free' and is filtered by the kidneys, giving rise to dark urine.[8]

CK is the most sensitive laboratory indicator of rhabdomyolysis. It is used as a surrogate measure of myoglobin as it is released from muscle proportionately with myoglobin and can be quickly assessed less expensively.[3] Recent studies of laboratory-controlled exertional rhabdomyolysis performed after eccentric exercise have found that CK and myoglobin levels can be profoundly elevated (with CK levels up to 80,000 U/L) without any consequence of renal failure,[9] as in our case. It was suggested that other factors such as dehydration and aciduria that decrease the solubility of myoglobin precipitates increase the risk of renal injury.[10]

ARF occurs in approximately 15% of patients with rhabdomyolysis.[1] There are three main pathways through which ARF develops: tubular obstruction by myoglobin and urate precipitates, tubular damage by oxidant injury and vasoconstriction.[11] Thus, the management strategy of rhabdomyolysis in the acute stage is two-fold: first, it is to treat the underlying cause when present, and secondly, is to prevent if not minimise ARF development. Ideally, restoration of muscle blood flow by volume expansion should be done to prevent more extensive muscle necrosis and ARF, but paradoxically, it can also transiently increase muscle damage by causing reperfusion injury.[12] Thus, for such patients like ours with no past or current indications of impending ARF (normal urine output and pH, serum electrolytes and renal function) in spite of elevated CK levels, the management dilemma of whether to institute aggressive intravenous fluids does arise. There is no doubt, however, that our patient would need close and serial monitoring of his urine output, muscle enzymes, renal function and acid-base status. We highly recommend generous fluid consumption, rest and self-monitoring of his urine output.

Three major issues need to be addressed after an athlete, be it elite or recreational, recovers from rhabdomyolysis.[13] First, who is at risk for recurrence and requires further evaluation? Second, for athletes not requiring further evaluation, when can they safely resume their sport? Finally, should any restrictions be placed on the athlete, and for how long?

Currently, there is no standard guideline to determine return to play after an episode of rhabdomyolysis.[13] Although several authors have recommended resuming play once symptoms resolve, no consensus exists regarding the acceptable CK level

Table I. Risk-stratification for recurrence of rhabdomyolysis.[13]

High-risk	Low-risk
• Delayed recovery (> 1 week) when activities have been restricted • Persistent elevation of CK (> 5 times the upper limit of the normal lab range) despite rest for at least two weeks • Rhabdomyolysis complicated by acute renal injury of any degree • Personal or family history of rhabdomyolysis • Personal or family history of recurrent muscle cramps or severe muscle pain that interferes with activities of daily living or sports performance • Personal or family history of malignant hyperthermia, or family history of unexplained complications or death following general anaesthesia • Personal or family history of sickle cell disease or trait • Muscle injury after low to moderate work or activity • Personal history of significant heat injury (heat stroke) • Serum CK peak ≥ 100,000 U/L	• Rapid clinical recovery and CK normalisation after exercise restrictions • Sufficiently fit or well-trained athlete with a history of very intense training/ exercise bout • No personal or family history of rhabdomyolysis or previous reporting of debilitating exercise-induced muscle pain, cramps or heat injury • Existence of other group or team-related cases of rhabdomyolysis during the same exercise sessions • Suspected or documented concomitant viral illness or infectious disease • Taking a drug or dietary supplement that could contribute to the development of rhabdomyolysis

or rate of progression. O'Connor et al suggested, from a military perspective, that individuals who experience exertional rhabdomyolysis be risk-stratified as either at a low or high risk for a recurrence (Table I).[13] The low-risk athlete would follow a three-phase guideline on return-to-play, which consists of rest, gradual introduction of activities while monitoring serum CK and serum uric acid levels, and gradual return to sporting activities and physical training. The high risk athlete would need to undergo a complete history and physical examination, and consult an expert to consider further myopathic disorders before returning to play in any moderate-to-high intensity sport. During the extensive evaluation, an athlete's activity level can be individualised to allow physical exertion within a 'reasonably safe range'.

Based on the criteria in Table I, our patient is considered 'high-risk for a recurrence' and would require extensive evaluation before swimming or any moderate-to-high intensity sports can be resumed. Care should be taken to avoid precipitating factors and 'safe' activity level should be determined to prevent further episodes of rhabdomyolysis.

In conclusion, rhabdomyolysis could have serious and fatal consequences; therefore, awareness among medical personnel and those involved in emergency care and care of the athlete is crucial for preventing complications and recurrence. Once out of the dangers of its complications, the management of this condition needs to be individualised based on risk stratification, with gradual return to activities and close monitoring to ensure safe return to sport.

REFERENCES

1. Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. Am Fam Physician 2002; 65:907-12.
2. Baxter RE, Moore JH. Diagnosis and treatment of acute exertional rhabdomyolysis. J Orth Sports Phys Ther 2003; 33:104-8.
3. Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. Am J Phys Med Rehabil 2002; 81(11 Suppl):S52-69.
4. Clarkson PM. Exertional rhabdomyolysis and acute renal failure in marathon runners. Sports Med 2007; 37:361-3.
5. Kravitz L, Vella CA. Energy expenditure in different modes of exercise. In: ACSM Current Comments [online]. Available at: www.acsm.org/docs/current-comments/energyexpenindifferentexmodes.pdf. Accessed May 31, 2010.
6. Costill DL, Maglischo EW, Richardson AB. Handbook of Sports Medicine and Science: Swimming. 1st ed. UK: Blackwell Science Ltd, 1992.
7. Brudvig TJ, Fitzgerald PI. Identification of signs and symptoms of acute exertional rhabdomyolysis in athletes: a guide for the practitioner. Strength and Conditioning Journal 2007; 29:10-14.
8. Knochel JP. Mechanisms of rhabdomyolysis. Curr Opin Rheumatol 1993; 5:725-31.
9. Clarkson PM, Kearns AK, Rouzier P, Rubin R, Thompson PD. Serum creatine kinase levels and renal function measures in exertional muscle damage. Med Sci Sports Exerc 2006; 38:623-7.
10. Sinert R, Kohl L, Rainone T, Scalea T. Exercise-induced rhabdomyolysis. Ann Emerg Med 1994; 23:1301-6.
11. Holt SG, Moore KP. Pathogenesis and treatment of renal dysfunction in rhabdomyolysis. Intensive Care Med 2001; 27:803-11.
12. Odeh M. The role of reperfusion-induced injury in the pathogenesis of the crush syndrome. N Engl J Med 1991; 324:1417-22.
13. O'Connor FG, Brennan FH Jr, Campbell W, Heled Y, Deuster P. Return to physical activity after exertional rhabdomyolysis. Curr Sports Med Rep 2008; 7:328-31.