Rhabdomyolysis after spin class?

Two case reports involving the increasingly popular activity of “spinning” underscore the need for proper conditioning and adequate hydration before exercising strenuously.

Primary care physicians frequently encourage patients to lead a more active, healthy lifestyle. The rise in popularity of endurance events, yoga, and organized gym-based fitness classes has, no doubt, improved the health of those who participate. But what happens when an individual moves too quickly from a sedentary existence to a more physically active one?

In this article we describe 2 clinical cases of rhabdomyolysis that occurred after healthy individuals participated for the first time in a class involving high-intensity stationary cycling, known as “spinning.” This exercise activity originated in California around 1989 when a competitive cyclist introduced variable resistance and speed training to stationary cycle workouts. Over the last 10 years, spinning has gained a worldwide following as a means of building cardiovascular endurance while achieving a significant calorie burn.

CASE 1 ★
Lack of conditioning, improper hydration spell trouble
A previously healthy 38-year-old white man presented with left lower extremity pain and dark urine. Three days earlier, he had participated in a spin class for the first time. Despite a sedentary lifestyle, he had no difficulty completing the session and felt fine during the class. He did feel mildly fatigued afterward. The next day, he began noticing discomfort and swelling in his left knee, which progressed to his anterior thigh. That evening, he became concerned because of a dark red tint to his urine. He was not taking any medications.

The physical exam was unremarkable except for a moderately swollen, tender knee with reduced range of motion. An x-ray of the knee showed a moderate suprapatellar effusion, but no fracture or dislocation. Urinalysis was remarkable for blood and myoglobin. The CPK value was 149,985 U/L (normal, 24-170 U/L), AST was 2234 U/L (normal, 9-25 U/L), ALT was 570 U/L (normal, 7-30 U/L), and BMI was 26.5 kg/m². Renal function was normal, as evidenced by a BUN of 17 mg/dL and a creatinine level of 1.0 mg/dL. He was afebrile and his WBC count was 9.6 x 10⁹/mm³.

We hospitalized the patient with a diagnosis of rhabdomyolysis and started him on aggressive intravenous (IV) hydration. The patient’s CPK and transaminase levels started trending down the next day, urine output (UOP) remained at goal, and renal function remained stable. Pain and swelling diminished over the next 3 days. He was discharged home on Day 4. At discharge, his CPK level was 26,180 U/L, BUN 10 mg/dL, and creatinine 0.8 mg/dL. At 1 month follow-up, his CPK was within normal limits.

CASE 2 ★
Even those who exercise regularly can overdo it
A previously healthy 26-year-old white wom-
an sought care at our clinic complaining of bilatera1 leg pain and dark urine. Despite being overweight, she regularly engaged in moderate exercise, and 2 days prior had participated in her first spin class. She felt some discomfort 30 minutes into the class, and the next day noted discomfort in her anterior thighs, which progressively worsened. Two days after the workout, her pain was worse and her urine became reddish-brown. She was not taking any medications.

The physical exam was unremarkable except for antalgic gait and tenderness of the anterior thighs, which were also moderately firm and warm to the touch. Urinalysis showed a large blood concentration and was positive for myoglobin. ALT was 366 U/L, AST was 1383 U/L, CPK was 86,592 U/L, and BMI was 33.36 kg/m². A BUN level of 11 mg/dL and creatinine level of 0.8 mg/dL suggested normal renal function. Her WBC count was 12.2 x 10⁹/mm³.

We hospitalized the patient for a presumptive diagnosis of rhabdomyolysis, and initiated aggressive IV hydration to achieve a UOP of at least 200 mL/h. CPK levels and renal and liver function were closely monitored. On hospital Day 2, the patient's thighs were tender and tight, so we consulted orthopedics about possible compartment syndrome. The consultant believed that intervention was unwarranted.

By Day 3, the swelling and pain began to resolve. UOP remained at target, and CPK and transaminase levels continued to trend down. Renal function remained stable. The patient was discharged home on Day 4 with a CPK of 11,388 U/L, BUN of 8 mg/dL, and creatinine of 0.7 mg/dL. At her 2-week follow-up, CPK was down to 772 U/L, and transaminases were within normal limits.

Discussion
Rhabdomyolysis occurs as a result of damage to the striated muscle cell membranes. Such injury releases into the systemic circulation calcium, potassium, phosphate, urate myoglobin, CPK, aldolase, lactate dehydrogenase, AST, and ALT. In the presence of excess calcium, further muscle fiber necrosis occurs and can lead to acute renal failure. Serum haptoglobin binding capacity becomes overly saturated. This results in free myoglobin, causing renal tubular obstruction. Myoglobin then dissociates into ferrihemate and globulin. Ferrihemate further exacerbates failure of the renal tubular transport system, eventually resulting in cell death and renal failure.¹⁰

Military trainees and casual athletes comprise many of the cases of exercise-induced rhabdomyolysis.⁴⁻⁶ People who exercise regularly are less likely to develop the condition than their more sedentary counterparts. As with our cases, a sudden increase in the intensity and duration of vigorous exercise, without proper training, may increase the likelihood of rhabdomyolysis.⁶

Other potential underlying causes. In addition to exercise and dehydration as depicted in our cases, rhabdomyolysis can result from burns, shock, acidosis, infections, crush trauma, immobility, malignancy, medications, toxins, abuse of drugs, or pre-existing illness such as sickle cell trait or other metabolic conditions.⁷⁻⁸

Clinical presentation varies. Regardless of the cause, patients typically present with muscle pain, weakness and cramping, and discolored urine.⁹ However, many patients will have dark urine associated with other symptoms, such as general malaise, visceral pain, swelling, muscle stiffness and tightness, fever, tachycardia, nausea, and vomiting. A careful history may help elucidate the cause.

Laboratory clues. Diagnostic guidelines commonly specify a serum CPK level 5 times the upper limit of normal as an indication of rhabdomyolysis, specifically in the exertional variety.⁹ Typically the level of this is around 1000 U/L.² However, there is no agreement on what CPK level is diagnostic of rhabdomyolysis. Suggestions range from 1000 to 20,000 U/L.³⁻⁴ A CPK level in excess of 5000 U/L increases the risk for acute renal failure and renal cell death.³⁻⁰ In athletes, an elevated CPK after working out is not uncommon and may be much higher than in other individuals.⁶⁻⁸ Endurance exercises such as marathon running or cycling have been noted to elevate CPK for up to 2 hours postexercise.⁸

Myoglobin becomes detectable in urine...
Hydrate patients aggressively with IV normal saline or crystalloids to maintain a urinary output of 200 to 300 mL/h. When it exceeds 1.5 mg/dL, urine becomes tea-colored or reddish-brown when myoglobin is >100 mg/dL.

**Complications** from rhabdomyolysis include compartment syndrome, hyperkalemia, disseminated intravascular coagulation, coagulopathies, and acute renal failure.

**Treatment** for rhabdomyolysis consists of aggressive IV hydration with normal saline (with variable rate) or crystalloids to maintain a UOP of 200 to 300 mL/h. Avoid fluid overload in the elderly and those with renal or cardiac disease. As CPK and myoglobin continue to trend down, it’s important to adjust IV fluids and electrolyte replacement. Using bicarbonate to alkalize the urine is controversial, with no studies showing any benefit. In severe situations, consider a nephrology consult for hemodialysis to bring CPK, which may be secondary to renal failure and hyperkalemia. However, renal failure is less likely to occur in physically active, healthy athletes.

**Advice after recovery.** After an episode of acute rhabdomyolysis, conditioned athletes can return to physical training with resolution of their symptoms or a CPK level from 1000 to 5000 U/L, usually within a week. A more judicious approach may be needed for less fit individuals. Regardless of their fitness level, advise patients to avoid diuretics and alcohol before exercise, remain hydrated during and after exercise, and avoid overhydration to decrease the likelihood of developing rhabdomyolysis. However, in patients with sickle cell trait, exertional sickness can occur with intensity of exercise without overhydration.

In the case of our male patient, poor physical conditioning and intensive, prolonged exercise followed by poor hydration and the diuretic effect of alcohol created the perfect storm for the development of rhabdomyolysis. On the other hand, our female patient routinely exercised, but still pushed herself beyond her limit and went too far too fast. Although BMI may play a role in the development of rhabdomyolysis, it does not appear to be as significant a factor as hydation status and overall physical conditioning.

Our patients’ prompt attention to the need for medical help and the recognition of the problem by their clinicians contributed to good outcomes in both cases.

**References**
